

ALMOST ICARUS

OR

RECOVERING FROM PARKINSON'S DISEASE:

UNDERSTANDING ITS CAUSE AND
MASTERING AN EFFECTIVE TREATMENT

JANICE WALTON-HADLOCK

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To my teachers, I offer this addition to the lore.

To MBG, who inspired me to begin the Little Project, I offer my love and gratitude.

People who are taking anti-parkinson's medications

We regret to announce that we cannot, at the present time, recommend the treatments suggested herein for people who have ever taken anti-parkinson's medications for more than three weeks.

For more information about our research in this regard, please read *Medications of Parkinson's Disease or Once Upon A Pill: Patient Experiences with Dopamine-enhancing Drugs and Supplements*, by Janice Walton-Hadlock. This book is available for free download at www.pdrecovery.org

According to Greek mythology, Daedalus was a brilliant inventor. To escape the Labyrinth in which he and his son, Icarus, were imprisoned, Daedalus fashioned wings of feathers and wax. They soared to safety, but Icarus, defying warnings, flew too close to the sun. The heat of the sun melted the wax. Icarus fell into the sea and was lost.

PART I

THE CAUSE AND CURE OF
PARKINSON'S DISEASE

“Although, at present, uninformed as to the precise nature of the disease, still it ought not to be considered as one against which there exists no countervailing remedy.”

— James Parkinson, 1817¹

CHAPTER ONE

PARKINSON’S DISEASE: UPROOTING THE CAUSE

Idiopathic Parkinson’s disease is not – and never has been – an incurable illness. Parkinson’s disease has a definite cause. Effective treatment for Parkinson’s disease requires removal of the cause.

This book presents evidence from case studies and details of the Asian medical theory that led to our discoveries in this area. It also teaches the techniques that the Parkinson’s Recovery Project now uses for successfully treating idiopathic Parkinson’s disease, or, as it is often abbreviated, “PD.”² This introductory chapter starts off with the unexpected observations that led to my first pilot study and then fast-forwards, ending with the distilled essence of our research.

MINDING MY OWN BUSINESS

In 1997, I was not looking for the root cause of Parkinson’s. I was not doing research at all. I was semi-retired after a pleasant career in an unrelated field and was amusing myself in a part-time practice of acupuncture when I happened to notice a similarity in the feet and legs of three patients who each had symptoms of Parkinson’s disease.

When I got my Master’s degree from Asian medicine school, the teachers had, of course, briefly covered this illness, so I knew as well as the next fellow that Parkinson’s disease was incurable, and that it stemmed from a brain glitch of unknown cause that led to a neurotransmitter deficiency.

In school, though, we had never learned anything about what the foot felt like on the side of the body that first exhibited the Parkinson’s symptoms. We certainly had not had the opportunity to feel the legs of people with Parkinson’s disease and notice that a major electrical current in the leg, a current well-studied in schools of Asian medicine, ran backwards in people with Parkinson’s.

So when I saw three patients with PD symptoms who had all received center-of-the-foot injuries in childhood, it struck me as uncanny. In each of these three, the area of the injury seemed as if it had not healed completely: the area at the center of the foot felt dead. Also, it felt as if the anatomy of the area wasn’t quite right somehow. It was hard to say for certain whether or not the bones were slightly displaced or the fascia tissue was knotted, but something felt wrong. The bones in the area did not glide correctly, as if tension was still present. The energy in

¹ Parkinson J. *An Essay on the Shaking Palsy*. Sherwood, Neely, and Jones, London, 1817. p. 56.

² “Idiopathic” means “of unknown cause.” I will continue to refer to the illness as idiopathic Parkinson’s disease or PD, in this book, even though the cause is now known, in order to differentiate this illness from drug- and toxin-induced parkinsonism.

the area seemed absent or highly distorted. I had to wonder how tension could be present if there was no energy. (Months later, as I repeatedly told colleagues that the area in the feet felt “dead,” I realized that the unusual tension in this area was more like rigor mortis – the immobility of death – than the healthy holding pattern of normal muscle tension.) At any rate, these injured feet felt and behaved as if their injuries had not fully healed.

I treated the unhealed foot injuries of all three of these patients with an extremely gentle form of Asian holding therapy. In each case, the tissues eventually loosened and energy began to flow through the old injury site. As it did so, the center-of-the-foot area lost its “unhealed” feeling.

It had struck me as uncanny that all three patients had what seemed to be an unhealed foot injury. Even more uncanny was the slow (over weeks and months) reversal of these patients’ Parkinson’s symptoms, after their feet healed.

Because they recovered from their assorted symptoms, these people evidently had *not* had Parkinson’s disease after all. I knew darned well that Parkinson’s disease was incurable. Ergo, these three had been misdiagnosed.

But it was difficult to dismiss all three as having been misdiagnosed. Maybe if it had been one patient, yes. But all three? I was puzzled. I had worked on three people who appeared to have symptoms of Parkinson’s disease. All three had an unhealed foot injury. When the foot was fully recovered from its injury, the PD symptoms went away. If they hadn’t had Parkinson’s disease, what had they actually had? The improbability of it all irked me. Like sand in an oyster, the recovery of these three “misdiagnosed” patients was a steady irritation to me. And whether they had had Parkinson’s disease or something else, I didn’t understand how an unhealed childhood foot injury could be related to my patients’ tremor, heaviness/numbness in the legs, lack of arm swing and/or absence of facial expression.

Constitutionally shy, I was alarmed to find myself standing up to make an announcement at the next meeting of the local Parkinson’s Support Group. Facing the group and nervously standing on one leg, I mumbled that I had seen a curious injury pattern in the feet of three people with symptoms resembling Parkinson’s disease. I wanted to follow up on this finding with a small pilot study: I offered to give several free acupuncture sessions to any volunteer with Parkinson’s disease who let me examine his feet.

A small pilot study

A dozen people took me up on my offer. At that time, I limited my inquiry to the injured-foot commonality. My idea was to search for evidence of either injury or energetic trauma in the feet of people with Parkinson’s disease. As with the initial three PD patients, a system of Yin-type (extremely gentle, almost imperceptible) Tui Na (Asian massage), termed FSR (forceless spontaneous release), was used to locate and assess injury. Strange though it seemed, all twelve PDers in this study seemed to have evidence of injury in the foot near the area of ST-42¹ on the side of the body that had first developed symptoms of Parkinson’s disease – just like my first three PD patients. All twelve presumably bona fide PDers had an unhealed foot injury! That concluded Part One of this pilot study.

¹ See foot diagram, Fig. 3.7, chapter 3, page 55.

Control group

Part Two of the study was examining a “control” group, a group that didn’t have Parkinson’s, by doing the same technique on them. I created a random control group by the simple method of using twelve consecutive non-PD patients from my budding acupuncture practice. These people didn’t know that I was looking for anything in particular. When they came in for their regular appointment, I included an innocent foot “massage” as a part of their treatment after I had inserted their needles for whatever ailment they had that day.

With one exception, the twelve people in the control group did not show evidence of injury in either foot. Eleven of the people had normal flexibility, relaxation response, and normal Qi¹ flow in their feet. One person did not: Tim, age 12.² Tim had the same indications of injury at ST-42 as all the Parkinson's disease patients. Tim’s left foot was stiff and did not relax in response to being supported.

I had, by now, started checking on the direction of Qi (energy) flow in the middle of PDers’ feet (at the terminus of the Stomach channel).

The Qi flow in Tim’s foot at the end of the Stomach channel was minimal. Tim was extremely intelligent, played piano and violin, competed at a high level in several sports, excelled in academics, and was his middle-school’s student body president. He was very self-controlled, with an unusual level of poise and self-restraint for a child of his age. Looking back, with what I know now, he had all the hallmarks of what some people refer to as the “Parkinson’s personality.”³

I asked Tim about the various symptoms of PD. Was he stiff, slow moving? Was his balance poor? Did he tremor? No. He had none of the classic PD symptoms. I asked him if he ever felt tremory or shaky inside. (A few PDers had told me that they felt shaky inside long, long before the external, visible tremor ever appeared – sometimes for decades prior to the visible

¹ For now, understand the word “Qi” to mean energy.

² All patient names have been changed. The genders of half the patients have been switched. In any case where a career or other factors might make it possible for someone to “guess” who a patient might have been, I have altered the non-medical factors that might lead to personal identification.

³ The Parkinson’s personality has been studied for decades. As early as the 1930s, researchers were trying to find some way to typify the extreme intelligence, vigilance, and harm avoidance that often characterize the Parkinson’s personality. This interest from the scientific community in trying to find a relationship between an illness and a personality is unusual; in recent times, searching for such a relationship borders on being politically and socially incorrect. However, people who work with PDers often notice that their PD patients do *not* represent a cross section of humanity. PDers tend to have an enormous level of self-control, high intelligence, selflessness and drive. They almost never indulge in frivolous pastimes, they tend to greatly dislike interpersonal conflicts and they usually abhor making “scenes” in public.

A neurologist in my home town has also mentioned that PDers are “different.” When pressed, he attempted to explain his generalization in this way (I paraphrase): people with Parkinson’s are different; as soon as I give them a diagnosis of Parkinson’s, they go home and research Parkinson’s more thoroughly than I can. After that, I can’t tell them anything they don’t already know. They know more about the updates on the illness than I do.

As recently as the early 2000s, an article on the subject appeared in *Proceedings of the National Academy of Sciences USA* 2001; 98:13272-7. The article, “Personality traits and brain dopaminergic function in Parkinson’s disease,” by Valteri Kaasinen, MD, PhD, presented research proving that the characteristics of the Parkinson’s personality were *not* dopamine related, inasmuch as they were not diminished by antiparkinson’s medications. Also, the Parkinson’s Personality is present in PDers in the decades prior to their diagnosis and continues even if they use antiparkinson’s medications.

appearance of tremor.) Tim said no to my repeated questions about internal tremor. I asked if he had ever hurt his foot. He said no. Over the course of several visits, during which time I worked overtly with treating his sinus condition, I practiced FSR on his left foot while his needles were in place.

A Breakthrough

I asked Tim each week if he ever felt stiff, slow, out of balance, or if he had a shaking or vibrating inside. Always, he said “No.” During the third session, as Tim and I chatted while I held his foot, he suddenly mentioned that he had once hurt that foot: when he was five years old, his mother, backing up the car in the driveway, had accidentally run over his foot. Tim told me, “It didn’t hurt. I was more worried about how bad my mother would feel if she knew than I was about the foot. I never even told her what she’d done.”

Tim suddenly became deeply relaxed. The rigidity in his foot gave way. The slightly displaced bones in his foot dropped into their correct position. The flow of energy in his foot increased to normal levels. I could feel the change as his foot and leg relaxed completely. I continued holding his foot. A short time later, Tim asked me, “You know that shaking inside that you always ask me about?” I nodded. “Well,” he said, and then paused. “I must have had it after all, because it just stopped.” Another long pause, and slowly a thoughtful look came over his face. “It feels wonderful now. It must have been there a long time. I never knew it was there until it stopped. I feel so *still* inside.”

Study conclusion

Tim was the clincher for the study. Although he didn’t even realize that he had a tremor, he *did* have a constant internal tremor that ceased when his foot injury was successfully treated. Although I had no idea what the mechanism was at that time, it seemed that, possibly, there was some relationship between his unhealed foot injury and his internal tremor.

And there was something else: not only did all twelve of the PD patients in the study have a strange lack of responsiveness in the center of the injured foot as if the center of the foot was dead, they also all had a palpable static (palpable to the trained hand) in the skin of the leg as if the electrical current of the Stomach channel was running backwards.

On the outer (lateral) section of the leg, a downward current is the normal pattern described in Asian medicine. But the PDers all had currents running from the foot *upward* along the outer leg. The non-PDer, with the one exception of Tim, had a normal electrical pattern in the legs and feet, and normal, subtle responsiveness in the center of their feet.

Tim, the only non-adult in the study, had a rigid foot, abnormal flow of energy in that foot, and a subclinical tremor. When the foot gave an internal shudder and righted itself in response to a few sessions of assessment and treatment, the internal sense of tremor and the rigidity and non-responsiveness in the foot all ceased: his body became still and peaceful inside, and the foot became relaxed.¹ The Qi in his foot and leg began flowing normally in the correct direction.

¹ The assessment of the energetic blockage and the treatment are nearly the same. The former consists of various, scarcely perceptible suggestions of movement in the hands of the practitioner as he securely holds the patient’s foot or other body part being examined. The point of these movements is to ascertain whether or not the body part can respond in the normal fashion to these extremely subtle stimuli. The latter, the treatment, consists of simply holding the injured area, supporting it. This support is what eventually attracts the recalcitrant mind to take a look at the long-ignored area and start healing it.

I was thus able to conclude that there *might* be a relationship between a mid-foot injury and the tremor, and possibly even the onset of Parkinson's.

More shocking, the changes in both Tim and some of my PD volunteers from my simple holding of their feet in the damaged area suggested that the healing of the foot trauma might actually stop or *reverse* some of the symptoms of Parkinson's. Can you imagine? And yet, that *was* one possible conclusion from this study. I published the study.¹ I mentioned in the article's conclusion that possibly the results indicated a fruitful avenue for future research. I thought that the foot injury situation might hold some answers, but I had many unanswered questions.

In retrospect, I had turned a blind eye to another important clue to the Parkinson's mystery. Tim had said, "It didn't hurt. *I was more worried about how bad my mother would feel if she knew than I was about the foot.* I never even told her what had happened." A few PDers who remembered their foot-injuring events while I treated them had said something eerily similar. However, at the time, I was only interested in the glaring similarity of unhealed injury in all the PDers' feet. I was not yet interested in the mental state that allowed the injury to stay unhealed.

Forming a hypothesis

So, my tiny research project was over, but I was more curious than ever. What did this foot idea mean for the big picture of Parkinson's? What had I proven? I had a long-shot conclusion: a hypothesis that Parkinson's disease was related to energetic confusion in the foot on the side of the body on which the PD first developed.

Slowly, over nearly two years, I constructed a hypothesis of body-wide electrical disarray, potentially initiated by an energetic blockage in the foot, which involved electrical short circuits and backwards-flowing energy. This hypothesis was completely consistent with the rules of Asian energetic schematics and the laws of basic physics (electricity and magnetism); it matched observations of the electrical presentation of PDers; *and* it could account for *all* the symptoms of Parkinson's disease, not merely the symptoms related to dopamine insufficiency.

Somehow, possibly because of my visit to the local Parkinson's Support Group, word got out that I was interested in working with people with Parkinson's disease. PDers started showing up at my office door. But although the sample size of my continuing study was increasing, it was too small to be statistically meaningful.² I also needed to know if my tiny sampling was just a fluke, if I was imagining things, or if I had actually stumbled onto something important. I needed replications of my findings by other health practitioners.

¹ Walton-Hadlock JL. Primary Parkinson's Disease: The Use of Tuina and Acupuncture in Accord With an Evolving Hypothesis of Its Cause from the Perspective of Chinese Traditional Medicine. *American Journal of Acupuncture*. Capitola, CA, 1998. Vol. 26, No. 2/3. pp. 163-177. Part 2 of the article was published in Vol. 27, No. 1/2, pp. 31-49.

² Early in my research, I consulted with a professor who had taught medical research at a medical school. He told me, "Because Parkinson's disease is considered incurable, every single patient that recovers will be declared misdiagnosed by the conventional medical community. Therefore, no one recovery will be significant by itself. You will need a thousand recoveries before your results become meaningful."

Staying busy

As my PDers continued to improve and my hypothesis tightened up, I published more articles, usually in a single-case study format.¹ A friend built a website for me. I used the website to post all of my findings. In both the website and the published articles, I included my email address with a request that acupuncturists with PD patients please examine the feet of their patients and note if there was a sort of lifeless area in the center of the foot and possibly a history of injury. I was hoping for replications of my findings. I was hoping to connect with someone, or maybe even several people, out there in cyberspace or in academia. I was very naïve about the enormous communication power of the infant Internet.

Unexpected changes in response to simple foot holding

Meanwhile, what was happening to the original group of twelve volunteers? After the search for signs of injury and/or energetic blockage was over, I continued meeting once a week with each of my volunteer patients, holding and assessing their feet. Each week I would start the free, hour-long session rolling with a quick, “How are you this week?” Then, while I held his foot, I gave that hour’s patient free rein to describe what changes, if any, he was feeling. Very often these sessions revealed much about the PDer’s childhood, and his attitude towards pain, work, and life. The talk, talk, talking helped me discover striking emotional commonalities of PDers. Several more years passed before I began to suspect the significance of these.

In these hour-long sessions, some of these patients described distinct changes that were beginning to occur in their bodies. This was happening the most powerfully in those patients whose Qi was once again running in the correct direction. Many of the people had not noticed any overt movement inside their feet during treatment. Others had felt sensations of bone rotations, muscle relaxation, static releases or strange internal foot-bone shudderings, in response to my gentle administration of Yin Tui Na techniques.

Regardless of whether or not the patients felt the corrective movement inside their feet, the patients whose Qi had resumed correct flow started having strange sensations in their feet and increased awareness of the feet, which was understandable. But they were also having changes throughout their bodies, such as a sudden appearance of bruising on the feet or other body parts that had been injured decades earlier, a blessed decrease or even cessation of insomnia, and/or, in some cases, a decrease or change in tremor.² Many, even those who had

¹ These other articles include the following: Walton-Hadlock, JL. “Parkinson’s Disease from TCM/channel Perspective: Theory and Case Study.” *California Journal of Oriental Medicine*, 2001, Vol. 12, No.1. pp. 8-13 and Walton-Hadlock, JL. “The Use of Yin Tui Na and Stomach Channel Acupuncture Points in the Treatment of Facial Immobility in Parkinson’s Disease.” *Journal of Chinese Medicine*. Sussex, England. 2002, Vol. 69, pp.43-47. The latter article included photos demonstrating the return of facial expression in a PD patient who had not been able to smile for nearly twenty years. (*Journal of Chinese Medicine* is the top-ranked peer-reviewed English-language journal of Asian medicine.)

² Several of my earliest patients, especially those who were recently diagnosed or never “officially” diagnosed, had a complete return to health over the course of a few months. This made me hopeful that everyone might recover from Parkinson’s in a few months. Since then, I have found that many people require years of treatment. I have to wonder at the providence that sent to me, at the very beginning of my research, several patients who recovered quickly. These patients kept my hopes up when the nightmarish problems of my medicated patients began to arise. Had it not been for the rapid recoveries of these unmedicated, recently diagnosed patients, I might have dropped the whole project when the drugged patients began to experience the horrors of overmedication and sudden, almost overnight, drug addiction.

been unable to smile for years, had a rapid improvement in facial expression and the return of the ability to taste and smell.

GROWTH OF A PROJECT

Results from afar

People wrote to me, primarily via email, in response to my articles and simple website. Acupuncturists, massage therapists and spouses of PDer's from around the world wrote to say that they could detect the same sort of aberrant energetics at the center of a PDer's foot. Some of their PD patients, too, responded to the foot holding treatment with shiftings in the foot bones, the foot and ankle tendons, and the micro-muscle-holding patterns, or with what felt like loosening or unwinding of twisted fascia. These foot responses were sometimes followed days or weeks later by short-term tingling, pain and/or bruising in the foot, and lasting changes in the Parkinson's symptoms.

In nearly every message, the writer said that he could detect, in a PDer, a foot situation that resembled my description. Not only that, but as the information on our website grew – eventually turning into this book – many people with Parkinson's disease wrote to say that our description of the symptoms and sensations of having Parkinson's was more complete and fit their sense of the illness better than any western (allopathic) medical description of PD.

One finding that was particularly intriguing to some of the correspondents was that some of their PD patients had no recall of any injury, but they had a foot that had clearly been injured.

And although a few PDer's wrote to say that they had a childhood injury in that part of the foot, more wrote to say that they did *not* remember any injury, but they had often wondered why they had a huge scar on their foot, “deformed-looking” feet, freezing cold feet or foot cramping, or some visible indication of foot injury. Some had no memory of a foot injury, but a sibling or friend had recalled for them a traumatic foot event when they were asked about it. Many had no recall of a foot injury but had a history of foot-related problems and/or weak ankles for “no reason.”

My findings and the Internet replies showed that the injury might or might *not* be remembered by the patient but, even so, displaced bones in the feet, a peculiar lifelessness or excess muscle tension in the foot, or scars might be indicative of an unhealed injury even if the patient had no recall of the event. In several cases, even though the PDer in question had no recall of any foot surgery, the foot had otherwise inexplicable *surgical*-type scars. These scars usually appeared as clean incision lines bordered by distinct spots which looked for all the world like the scars from neatly placed sutures. These scars, located near the center of the foot, might allow one to suspect that foot injuries and/or surgeries had in fact taken place, despite the PDer's lack of recall.

I also heard from people who did not recall any injury until they started receiving treatment. Suddenly, while their feet were being held, or shortly after, they recalled an injury event involving the foot *and* a motivation for keeping the injury hushed up. These people, frequently doubting their memories, often got confirmation from a sibling that there had been, in fact, such and such a day with such and such events transpiring. Some siblings could recall the injury and even remarked on how surprised they had been when the injury recipient had not responded in any way to what should have been a nasty hurt.

At the same time, I heard back from people who could not replicate my results and even from people who were outraged at my hypotheses. One acupuncturist wrote a strong letter to the editors of the *California Journal of Oriental Medicine*, condemning the journal for having published my work; he worried that the Asian medicine profession had enough problems without crackpots like me making us all appear laughing stocks.

Happily, I also heard from people who were getting replications of *recovery* results in some PDers, though not all. Some of those who appeared to be recovering were also experiencing many of the counterintuitive, unpleasant, painful, and even bizarre recovery symptoms that I had observed but which I had not yet written up. Almost always, these weird symptoms served to confirm our early hypotheses of foot-nerve dormancies that were coming back to life in response to treatment of long-unhealed foot injury.

I was pleased that many people were having symptoms of recovery, but I felt, at that time, that the most significant finding of my Little Project was this: people with Parkinson's were saying that my *description* of the physiological processes at work in Parkinson's matched exactly with what they *felt* inside their bodies. I cannot guess how many times I heard or read words from a PDer to this effect: "The medical descriptions of Parkinson's didn't match how I was feeling. I kept thinking that the doctor must be wrong. When I read your description, I cried. I felt as if, for the first time, someone understood how I felt: your explanation of what will ensue, eventually, if the Stomach Channel goes awry in response to a foot injury is an exact description of how I have felt inside for so many years."

Why me, Lord?

When the Internet replies started to arrive, I began to feel uneasy, even resentful, towards the fates that had so graciously satisfied my initial curiosity. I had not intended to be leading a research project. I certainly did not want to butt heads with the Big Powers of allopathic (western) medicine and the drug companies. On the other hand, if I had new information about Parkinson's disease, information that might be helpful to the millions of people with Parkinson's disease, or, for that matter, for any one individual, I had an obligation to make that information available. With uneasy gratitude for this opportunity to potentially help others, I started to formalize my little research project. It was still 1998. The Little Project had been going on for less than a year, but it was already growing: a few colleagues, both at home and abroad, had started working with me on the Little Project.

By the end of the year, several people had recovered from Parkinson's disease. Many more were manifesting physical changes that suggested that they were starting to recover from Parkinson's disease. Many of the medicated patients were experiencing symptoms of severe overmedication. And yet, as their symptoms of overmedication increased, they were finding themselves increasingly seduced by their medications. As these PDers became both less able to tolerate the medication and, suddenly, susceptible to addiction, they began having bizarre drug-based experiences. Abruptly, sometimes overnight, they no longer responded to their medications in the same manner as PDers. Instead, their ghastly behaviors, both motor and mental, were comparable with some of the cases written up by Oliver Sacks in his book, *Awakenings*, which chronicled some of the earliest research on L-dopa. His research had been performed on people who had sleeping sickness, *not* Parkinson's disease. Most of his (non-PD) patients had responded to L-dopa with the same alarming symptoms and mannerisms that my

patients were suddenly starting to manifest. This suggested that, chemically, my PDers were now behaving like non-PDers.

Though many of our cases involving medicated PDers had tragic endings, these cases also proved to us that the simple foot holding we were doing was causing people who had previously behaved like PDers, in terms of their response to medication, to no longer have PDer-like responses to their medications even if they were able to reduce their medication. This was highly significant from a research point of view. While a skeptic might wave off the unmedicated recovery cases by saying “those people probably didn’t actually have Parkinson’s; they were probably just misdiagnosed,” it would be impossible to airily dismiss the terrifying, rapid changes that the medicated PDers experienced.¹

The emotional component

Meanwhile, among the unmedicated PDers, we were noticing that the pace and style of each PDer’s recovery seemed related to his degree of emotional wariness. The level of emotional wariness that we saw ranged from minimal to extreme. It was not related to severity of Parkinson’s *symptoms*. Those PDers with the most flagrant Parkinson’s *Personality*, though, had the most difficult time responding to treatment. They also had the most difficulty, after their feet were healing, in allowing themselves to attain the non-wary emotional state necessary in *anyone*, not just PDers, for triggering dopamine release.

We spent years struggling to make sense of this. Eventually, new research in other fields allowed us to propose the chemical and emotional brain mechanisms that could explain our observations. Finally, we understood why our more stoic and/or wary patients “stalled” during recovery and why, for example, all five of our professional musician PDers recovered in a very rapid and straightforward manner.

Still, this emotional component meant that some PDers recovered easily and others did not. This meant that, in terms of western medical science, we could not say that we had a one-size-fits-all “cure.” To be able to say that we had found an effective way to treat idiopathic Parkinson’s disease, we needed to find a way to treat the emotional wariness that prevented some partially recovered PDers from learning how to consistently release dopamine.

When I say “partially recovered PDers,” I am referring to those people who manifested signs of physical recovery throughout their anatomy, but whose ability to initiate movement suddenly became highly irregular, or even radically worse than before. Dopamine release is mood- and expectation-dependent. If the expectations of partially recovered PDers were entrenched in patterns of cynicism, self-criticism, resignation, emotional numbness or negativity, their ability to initiate movement was more erratic or radically worsened after the adrenaline from their foot injury was gone.²

¹ The hellish situations that erupted among the PDers who were taking antiparkinson’s medications when they started to experience recovery are discussed in my 2003 book, *Medications of Parkinson’s or Once Upon A Pill: patient experiences with dopamine-enhancing drugs and supplements*. This book is available for free download at www.pdrecovery.org

² Both adrenaline and dopamine are neurotransmitters that can trigger mental and motor function. For the muscles that perform motor function, fear-based thoughts release the adrenaline that activates these muscles during times of wariness, danger or injury and joy- or contentment- based thoughts release the dopamine that activates these exact same muscles during times of calm, curiosity, or other “seeking” behaviors. (Continued on next page.)

Before we could say that we'd found an effective treatment for idiopathic Parkinson's, period, we also needed to find a way to help the PDers with these emotion-based patterns.

To say that The Little Project did not follow a straight path would be understatement. A far-reaching search for some method of emotional healing that could be effective across the entire spectrum of PDers led us down many avenues before we found a simple, common-denominator treatment. We had false leads, tragic, drug-related deaths (among the medicated PDers), and periods of pure bafflement, to say nothing of the overt hostility from some MDs and some fellow acupuncturists. But there were also curious and supportive MDs and acupuncturists. And best of all, I knew the joy of getting to know and work with some of the most intelligent, sincere, selfless and hardworking people I've ever met: the hundreds of people with idiopathic Parkinson's disease who joined us in our research. And what was the result of The Little Project?

JUMPING AHEAD TO THE PRESENT – THE YEAR 2006

An unhealed foot injury combined with a fear-based attitude

After working with hundreds of people with Parkinson's disease, I can say that, in all likelihood, idiopathic Parkinson's disease is set in motion by a foot injury that fails to completely heal.¹ The *reason* for the failure is also significant: the foot injury fails to heal because, at the time of injury – in some cases, even prior to the injury – the injured party has decided, consciously, that he must, for whatever reason, pretend that he is incapable of being hurt, of feeling physical (and sometimes emotional) pain. To keep this self-deception in place, an injured foot must not be allowed to exist: the injured foot must therefore be mentally and emotionally

Generally, when we talk about “adrenaline release,” we are thinking of the terrific surge of adrenaline that occurs during emergency. In fact, we *always* have some amount of adrenaline flowing. Whether the body is experiencing rapid heart rate and fully opened bronchial tubes – typical signs of emergency-based, high amounts of adrenaline release – or experiencing a resting heart rate and normal breathing depends on the *amount* of adrenaline being released at any given time.

Dopamine is also flowing in the body at all time, although, in the case of Parkinson's disease, the clusters of dopamine-producing cells in the midbrain become partially dormant. These dormant (no longer darkly pigmented, but still very much alive) cells are often referred to by the western medical community as “dead cells.”

These clusters of dopamine-producing cells which, in a healthy person support conscious mental and motor function, cannot be fully accessed by a person with Parkinson's: hence the dormancy.

¹ I must explain what I mean when I refer to “working with patients.” With local patients, I usually meet once a week for an hour. I may meet with the patient for months or even years. With visiting patients, we often maintain email contact with the patient and practitioner after we have worked with them in Santa Cruz. The long-term nature of our treatment program reflects our desire to root out and remove the cause of Parkinson's disease.

As an aside, our treatments are *not* oriented towards providing a temporary cessation of symptoms for the PDer. Programs and treatments that give immediate, short-term relief abound. PDers, especially those who have become accustomed to using dopamine-enhancing medications, are notoriously susceptible to a short-term placebo effect. Nearly all PDers, even those who have never used medication, can experience the temporary tremor-calming effect of any soothing therapy. However, these therapies do not address the root cause of PD and they do nothing to slow the progression of the illness. In fact, based on western research and our own observations, we suspect that inappropriate acupuncture and other stimulating treatments that give a temporary boost of energy may, like the drugs that provide “unwanted” dopamine, actually accelerate the disabling of dopamine receptors in the brain, thus accelerating the progression of PD.

disconnected from the body. However, once the foot is perceived to be either non-injured or non-existent, full healing of the foot injury cannot occur.¹

Electrical confusion in the area of the foot injury

Asian medicine recognizes a system of electrical currents in the body, the correct organization of which is crucial for the maintenance of health. In most of my writing, when I talk about electrical patterns, I will be talking about the largest rivers of currents in humans, usually called “channels,” from which all the smaller electrical patterns in the human body derive. The phrase “electrical patterns” can also refer to all the electrical schema in every living system; this includes the micro currents around each cell that regulate cell behavior and DNA expression, the larger currents that integrate the functions of the various organs in the living system and direct the development and maintenance of organs and nerves, and the major currents that, in brain-based organisms, allow the brain and body to grow and behave as an integrated unit. (Although western medicine theorists assume that nerves control the brain-body relationship, the growth and function of the nerves themselves are directed by the electrical patterns in the currents.)

In PDers, over decades, the long-standing injury in the foot causes the electrical patterns in the area of the injury to grow increasingly irregular. Eventually, whether changing at a glacial pace or a rapid one, the snowballing electrical disarray becomes large enough to present electrical resistance to a major electrical current that is supposed to traverse the top of the foot. This resistance sets in motion deleterious changes throughout the flow pattern of this particular current.

The center of the foot: injury site, terminus of the Stomach Channel

The unhealed foot injury that causes the electrical disarray of Parkinson’s disease, which can almost always be easily detected by hand (using a technique that will be explained later in this book), is at the center of the foot, at or around the 2nd cuneiform bone.²

¹ In cases of milder emotional detachment, some degree of awareness of the foot may continue, but with the understanding that the injury “never occurred” or “didn’t really hurt.” In the more extreme cases, even the mental self-image of the injured person changes in order to exclude the existence of the foot. When such a person is asked to mentally picture himself, his mental picture of himself may end at the ankle, the knee, or sometimes even the waist. He usually *cannot* mentally picture his foot (or feet). This truncated form of mental self-image or a complete inability to mentally picture one’s own body is common in Parkinson’s disease.

Also, a very common healing aid for *any* injury is to visualize light or feel energy in the injured area. This technique helps one to mentally focus on the area and increases vitality in that spot. However, most of our PD patients can only with reluctance and great difficulty, if at all, bring themselves to mentally picture their own bodies – particularly the very center of the feet – as being full of light.

² The electrical aberration at the center of the foot can be detected by machine. A visiting acupuncture professor from China, the renowned Dr. Ju-Yi Wang, considered a “Chinese National Treasure” and Master Teacher of Channel Theory, brought a machine he had invented to the acupuncture college where I teach. The home-made machine measured electrical forces in the skin via wires that emerged from a plugged-in black box, the ends of which terminated in damp cotton swabs. The swabs were placed against the skin at the beginning points and end points of the major channels. The computer screen to which the machine was hooked up graphed some aspect of the electrical forces being picked up by the leads. The electrical inputs from the skin were measured *not* in absolute value, but compared energy at various Source Points on the body to detect *relative* weaknesses and strengths in a person’s many channels, thereby locating objectively the most likely sources of a person’s health problems.

Through American translators, I asked dozens of questions but I remain uncertain as to exactly what the machine was measuring. One translator was pretty sure that Dr. Wang’s word that translates as “voltage.” The other

The significance of this location, smack in the center of the foot, is this: this spot is the terminus of a major electrical current. For reasons that will be explained later, this current is known, in the field of Asian medicine, as the Stomach channel.

In people with Parkinson's disease, an unhealed injury can be detected at the spot that *should* be, in a healthy person, the end of the Stomach channel.

As an aside, I talk of the "end of a channel," as though little bits of electrical current are isolated in the body, with distinct beginnings and endings. This is not accurate. All the channels actually connect to one another in a complex schematic that allows all parts of the body to communicate with each other. The most important channels, the named channels, are segments of current the locations of which are fairly uniform from one person to another. The named channels pass over very specific parts of the body and lie close enough to the skin that they can be detected by hand. The route of a given channel is usually referred to as its "path."

For example, the path of the Stomach Channel begins at the eyelid, flowing along a route that is sometimes narrow, sometimes wide, down the face, down the front of the neck, over the mammary line down to the pelvic bone. From there, it crosses to the anteriolateral side of the leg, and then flows down to the top of the foot. At the top of the foot, right over the 2nd cuneiform bone, the width of this channel narrows; the energy in this current converges right at this spot, before breaking up into a fan of currents that flow over certain toes and over to the big-toe side of the foot. Don't memorize the preceding, but the significance of this channel's location and the location of the structural symptoms of Parkinson's disease will arise before this book is done.

Now, back to the foot. In people with Parkinson's disease, the area around the 2nd cuneiform bone on the side of the body that first presents the symptoms of PD is electrically contorted. The area around the 2nd cuneiform bone in PDers feels energetically somewhat unresponsive, sometimes even "dead" to the touch. It may or may not also be physically unresponsive, contorted, jammed up or subtly twisted.

I've already mentioned that the Stomach Channel is supposed to flow down from the face and end at the 2nd cuneiform bone. But in people with Parkinson's disease, the Stomach Channel (on the side of the body that first manifested symptoms of Parkinson's) is running *backwards*: from the foot up towards the head. I repeat, the Stomach Channel, in people with Parkinson's disease, is running backwards. If the foot injury is fixed, the Stomach Channel resumes, spontaneously, in most cases, its correct flow. But that is looking ahead. For now, I need to explain why a backwards-flowing channel can be a very good thing or a very, very bad thing.

Backwards-flowing energy: a perfectly normal thing

It is not unheard of for a channel to flow backwards *for a very short time*. Electrical patterns in the body can get deranged due to injury. Take, for example, a healthy non-PDer who receives a highly significant injury, one severe enough to immediately derange an electrical

translator had no idea. Dr. Wang speaks no English. It seemed to me that amperage might be a more likely thing to measure since voltage, I imagine, should be somewhat consistent throughout.

I had Dr. Wang use his computer on my Parkinson's patients. He was stunned when the computer showed that my patients had almost *no* electrical signal at the center of the foot on whichever side of their bodies first manifested PD symptoms. He thought that something must be broken in his black box. He had never before seen a presentation that caused his machine to essentially flat line at a crucial channel terminus. Whether we were dealing with voltage or amperage, I still found it encouraging that my manual findings could be supported objectively.

pattern, such as a complex bone fracture, anywhere along the Stomach channel below the neck. In response to a significant injury, current reversal may occur in the part of the electrical system that is supposed to traverse the injury site. Energy in a downward-flowing channel may, starting from the electrical obstruction at the site of injury, flow *up* the path of the channel (in reverse of the usual direction) and then short-circuit at the head into the channel that allows deep sleep. This deep sleep channel is usually, in a non-injured person, activated only at night. This injury-induced short-circuit allows the wounded body to slip into a healthy, healing, resting phase (a phase of sleeping hard or napping a lot).

This resting-healing phase, in which dopamine release is inhibited, need not occur immediately. For example, if the injury occurs during a situation of on-going danger, the resting phase will not be evident: adrenaline-based motor function will dominate. A healing rest, and healing itself, will *not* occur during emergency conditions. The body will not slip into a phase of extra sleep and healing as long as an emergency is on-going.

For example, a person being pursued by a rhino does not need to know that his leg has been broken in the chase. He can run on a broken leg. He may not even feel leg pain or know that he has broken the leg until he comes to a safe place and reconnoiters with his body. His body cannot go into symptoms of shock or injury until he gets himself out of the immediate danger. Once he is out of immediate danger, he *should* become aware that his body has been badly hurt. It may be appropriate for him to seek help. He may even go into shock at that time.¹ Then, after he has been comforted and his injuries have been treated, when the pain eases up and the adrenaline climbs down, he will be able – thanks to the backwards-flowing energy having tripped the “go to sleep” circuit – to slip into a deeply calm, even predominantly motionless, physiological phase of “lots of rest and sleep.”

Healing sleep

The mechanism for the “healing sleep” phase of injury recovery is this: electrical channel reversal in the Stomach channel causes a short-circuit when the backwards flowing Stomach channel backs up all the way to the head. At the head, the Stomach channel short circuits into the Gall Bladder channel. The short-circuit causes increased electrical flow into the head portion of the Gall Bladder channel: the channel that ordinarily activates sleep processes. The Gall Bladder channel ordinarily only has an elevated level of current in it at night. But when energy in this go-to-sleep channel is pumped up during non-regular sleep times via the short-circuit from the injured-and-therefore-running-backwards channel, the sleep channel performs its usual electrical function: it triggers an electrical brain signal that sends the sleep message to the cells and structures of the brain.

Among these sleep messages are the usual sleep-time electrical signals that inhibit dopamine release in the midbrain (including the substantia nigra area).² This inhibition remains

¹ Symptoms of shock can include tremor, poor body temperature regulation (especially cold hands and feet), motor inhibition (slow, shuffling movement), weak voice, low blood pressure or poor blood pressure regulation. These symptoms are not uncommon in people with Parkinson’s disease.

² It is now recognized that only a very low amount of dopamine is released during sleep. In fact, people who take even slightly excessive levels of dopamine-enhancing antiparkinson’s medications at bedtime often suffer from insomnia, excessive movement during sleep, and sometimes even sleepwalking. (The narcolepsy experienced by people who take certain dopamine agonist medications is a completely different problem. It is caused by the

in place until the injury that triggered the directional shift is healed enough that the energy in the area of the injury can resume its normal flow pattern through the injured area.

If a person needs to do any motor activity during the time that the electrical pattern is reversed – that is to say, while he is in “injured mode” – the person will need to use the adrenaline-based, wariness-activated neurotransmitter system instead of the joy-activated, dopamine-based system.¹ After the injury is healed enough that the electrical system – including the channel that was running backwards – reverts back to normal, dopamine production and release are once again possible.

In the above scenario, the backwards-running electrical pattern is a good thing: it turns off dopamine production and release, thus enabling the injured person to rest or sleep deeply, for many more hours a day than he would normally have been able to.²

influence of dopamine agonists on the Stomach Channel and the stomach itself, an influence also known as “the overly large meal effect.”)

In the 1960s, in part because of PDers’ “inability to relax,” because they often slept poorly, and because they were known to be dopamine deficient, it was announced, based on no research whatsoever, that dopamine “must be” the neurotransmitter that caused sleep and/or muscle relaxation. However, thanks to new, excellent research on dopamine, now considered the neurotransmitter of joy and addiction, we now know that dopamine is *not* released in significant amounts during sleep.

However, many doctors who were educated in the 1960s through the 1990s, including neurologists, may not be aware of the new research. Maybe that’s because the best research on the role of dopamine is currently being done by the National Institute on Drug Abuse, a department somewhat removed from the path of most neurologists and general practitioners. (Continued on next page.)

For in-depth information on this historic science blooper, please read Appendix 6, “Dopamine fallacies,” in my book, *Medications of Parkinson’s Disease or Once Upon A Pill: patient experiences with dopamine-enhancing medications and supplements*. This book is available for free download at www.pdrecovery.org.

Here’s a sample of one of the research mistakes written up in the above appendix: the only test from the 1960-1970 era which actually measured amounts of night- and day-time brain dopamine, by abruptly chopping off the subjects’ heads, putting the brains in a blender and then measuring dopamine amounts, found that dopamine levels were higher at night. Amazingly, none of the researchers seemed to be aware that rats, the animals used in the study, are nocturnal. Thus, higher dopamine levels at night corresponded to the rats’ times of higher physical activity. Lower levels of dopamine in the daytime reflected the fact that rats usually sleep in the daytime. In other words, dopamine levels are lower during sleep.

All dopamine measurement studies of humans (measuring blood levels of dopamine rather than shoving brains in a blender) have shown that, in humans, dopamine levels are lower at night, and especially low during sleep. But because the rat study provided the answer that was expected and desired, the rat study was flaunted as “proof” that *human* dopamine levels are supposed to be *higher* during sleep – a completely wrong conclusion, but one that lived for over thirty years, and is still subscribed to by many MDs who went to school between 1960 and the late 1990s.

¹ Technically speaking, dopamine is not just released in the brain, but is present throughout the body, and is also used, in the heart, as a trigger even for the sympathetic, fear-based system. A person without enough interest in living, for example, a person who is on his deathbed or in deep despair, cannot, because of dopamine insufficiency in the heart, be stimulated to respond even to emergencies. However, for purposes of simplification in these early chapters, I will ignore this heart-based source-of-life aspect of dopamine use and refer to the two main nerve systems as the adrenaline and the dopamine systems, more commonly known as the sympathetic (fight or flight) and parasympathetic (curiosity and cud-chewing) systems.

² The Stomach channel, more than any other, is associated with the parasympathetic nervous system (the system activated when feeling relaxed or when enjoying food) and with dopamine release. When the Stomach channel runs vigorously, dopamine flow is increased. Oppositely, the Stomach channel in particular is laid out in such a way that a severe injury to this channel is able to shut down the brain portion of the dopamine system. For

Backwards flowing currents can be very, very bad

In the PDer, though, the injury never fully heals because, emotionally, it “never happened” or “didn’t even hurt.” However, the subconscious mind is keenly aware that something painful has occurred – an emergency has occurred. In terms of body chemistry, if not in terms of conscious awareness, the *emergency* never ends.

One consequence of this emotional denial is that the injured person’s call for adrenaline never gets turned off. You will remember how, in the previous fleeing-from-rhino scenario, the person used adrenaline until he could get to a safe place, acknowledge the injury, and maybe even get some help. Then, when his adrenaline levels eased off, healing and the concomitant healing-sleep could set in.

Well, in the case of the PDer, there may never be a “safe place.” So the PDer remains stuck in the adrenaline phase. Sometimes the PDer’s injury “never happened.” Sometimes it “wasn’t important,” because of an ongoing emotional sense of emergency with regard to the injury or to some other life situation. For whatever reason, the “emergency” that necessitates an pro-adrenaline, anti-dopamine mode never ends.

In other words, the PDer may live his whole life as if he is still running away from the rhino. The healing-sleep, anti-dopamine brain pattern may have become established – a brain pattern that *would* allow the PDer to fall into healing-type sleep if he ever turned off the adrenaline – but he can never avail himself of it. He may also be waiting to manifest shock, to set in motion the tremoring, lying down and curling up that wordlessly communicates to the others of his species that he is in need of help, warmth and comfort, but he cannot yet manifest it. He is still on adrenaline; he is still running away.

Note carefully: for *some* PDers, the mental block against starting the healing process is only tied up in the foot injury. At the other end of the mental-block spectrum, some PDers cultivate this dynamic, adrenaline-based emergency mindset until every aspect of their lives is approached via adrenaline: with careful wariness, a sense of chronic importance or even emergency, relentless self-criticism and/or negativity, or even an emotionally immature belief in the “virtue” of utter self-reliance or self-protection.

The PDer, ever running away – at least with regard to the foot injury, or maybe with regard to every potential threat in life – may not be able to manifest these symptoms of injury (the sleep of purposeful dopamine shutdown, a dragging leg, or the tremor of shock) until either life-style relaxation or exhaustion allow them to peek through.¹ And when these symptoms do appear, the PDer will not see these early symptoms of injury or shock as indications that he needs to find a safe place and be comforted. Instead, the symptoms of tremor, cold, or light-

more information about the relationship between the Stomach channel and dopamine release, please see chapter 24 in *Medications of Parkinson’s Disease, or Once Upon a Pill*.

Also, in Temple Grandin’s excellent book, *Animals in Translation*, she points out that dopamine is the dominant neurotransmitter for “seeking” behaviors: curiosity, interest, appetite and anticipation. These are all qualities that need to be temporarily stifled if a serious injury is going to be allowed to heal in peace and comfort.

¹ “Life style relaxation” refers to this: some PDers’ symptoms begin to manifest as soon as they retire, when the last of the children finishes college, or after a long-desired extended vacation comes to pass. Others first manifest symptoms while recovering from serious surgery or an illness that forced them to take a long rest and be waited on by others. While these situations would ordinarily cause a decrease in adrenaline and a concomitant increase in dopamine-based relaxation types of behavior, a person with PD merely experiences the decrease in adrenaline; dopamine release is still being prevented by an electrical “emergency” pattern caused by foot injury and long-time emotional habits of wariness.

headedness-when-changing-from-seated-to-standing usually stir the PDer to try to resurrect his failing adrenaline-based control over his body. The PDer is, in many cases, not ready to ask for help – he may imagine he needs to keep running from the rhino.

The PDer's injury never heals (except on a superficial level of skin healing and possibly some minor attempts at sealing off the injury). The electrical reversal pattern, set in motion either immediately or over years, by the disarray at the injury site, never goes back to a correct flow: it becomes chronic. The dopamine-system inhibition pattern, the healing-sleep pattern, set in motion by backwards-flowing current and its short-circuit into the Gall Bladder channel at the forehead, becomes chronic.

Physical, mental and emotional reliance on adrenaline, instead of dopamine, becomes chronic. After decades, the results of the electrical current reversal through the leg, torso, neck and head, and the pattern of adrenaline-dominance and concomitant dopamine-inhibition manifest respectively as the physical (structural) and the movement-inhibition and tremor (emotional and mood- or expectation-dependent) symptoms of Parkinson's disease.

Parkinson's is curable. When the foot injury is healed *and* the PDer resumes using dopamine-releasing thought patterns instead of the adrenaline-releasing thought patterns (negative, vigilant, word- and logic-based or self-preoccupied patterns) to which he has become accustomed, the symptoms of Parkinson's disease melt away.

The foot problem and the mental attitude that allows it to thrive are, evidently, the root cause of Parkinson's disease. These *are* evidently the root cause because, when both the foot injury *and* the life-long mental attitude that held it in place are treated and healed, a person with Parkinson's disease then permanently recovers from his PD symptoms. The foot treatment is not difficult and can be performed by a layman. In some cases, channel-blocking scars and rerouting of incorrect channels may require treatment with a special style of acupuncture. The difficulty in changing the mental attitude varies from person to person. While this change in mental attitude can be supported by friends and health practitioners, the onus of this change ultimately rests on the PDer himself.

IN CONCLUSION

We now feel confident that we understand the cause of idiopathic Parkinson's disease.

The cause has two parts. In all the cases of confirmed idiopathic Parkinson's that we have seen, an unhealed foot injury has been present. To varying degrees, mental/emotional dissociation from the injury has also existed. In all cases, the (originally intentional) dissociation has prevented the PDer from having full awareness of his foot. In some cases, the dissociation has expanded beyond the denial of foot injury, and has become a dominant force in shaping the PDer's personality, causing the PDer to have presented, even prior to his illness, an adrenaline-based, healing-inhibiting emotional posture to the world.

Over decades, this denial of foot feeling may grow and spread into additional mental arenas. In many cases, the PDer has created a mental/emotional condition in which he behaves as if he is not mentally or emotionally associated with his injured body part, or even, eventually, the positive (pleasant) types of sensations his own body. This condition can feel, literally, as if the heart is empty or else walled off with regard to feeling one's *own* physical or emotional pain.¹

¹ Sensitivity to the feelings of *others* is not necessarily effected one way or the other by Parkinson's disease. With regard to other people's physical or emotional pain or joy, a PDer might have any sort of attunement, any learned or intuitional sensitivity and caring that falls within the spectrum of normal human behavior.

This physiological process, a process that involves a shift in heart neurotransmitter levels, is called dissociation. Dissociation can occur spontaneously during a traumatic event. People with high intellect and enormous self-control can also induce a state of dissociation intentionally. Eventually, this denial of self-feeling can lead to anxiety and/or depression: manifestations of a heart response so diminished that neither adrenaline *nor* dopamine can be released in quantities large enough to provide for healthy mental or motor function.

Treating the PDer's foot injury in an extremely gentle, non-threatening manner allows the attention to be gently brought back to the dissociated body part: the injury can heal. When the foot is healed enough that the disrupted electrical currents can resume their normal pathway, the anatomical symptoms of Parkinson's, including the injury-type inhibition of dopamine production and release, melt away. In some cases, the healing of the foot also serves to erase the "unable to feel," the "closed-off heart" response, restoring normal emotional capability for triggering dopamine release.

In other cases, though, even after the foot injury is healed and the brain is anatomically *capable* of releasing dopamine, dopamine may still be hard to access consistently, if at all. When a person has an emotional or mental habit of sustaining a dissociation response, dopamine release is inhibited. Even if there is no ongoing injury, the mental or emotional habit of dissociation can inhibit dopamine release. This short-term inhibition of dopamine occurs in any *anyone* experiencing or maintaining a mental or emotional dissociation response – not just a person with idiopathic Parkinson's. The mechanism for this diminished lack of sensory feeling and diminished nerve signal activity between the brain and the heart, which in turn may cause diminished levels of neurotransmitter release, will be explained further in later chapters.

If the PDer whose foot injury has recently healed still cannot experience dopamine-releasing heart-based *feelings* and their resultant dopamine-releasing thoughts that lead, in turn, to dopamine-based motor function, he must try to fall back on his habitual adrenaline-based thoughts and their related motor functions. However, once the PDer's foot injury is gone, *adrenaline will be harder to come by*. As the heart, in long-term dissociation mode, sends ever-diminishing signals to the brain, the body's ability to have *any* neurotransmitter release, even adrenaline, will continue to decrease. With the adrenaline from the foot injury no longer contributing to the neurotransmitter mix, the ability to initiate his customary adrenaline-based movements and hide his tremor will decline even more rapidly than would be expected from the normal progression of Parkinson's.

The diminished heart signals of PDers can be seen in PET scans. When researchers measure the response of the heart's sympathetic nerves (the nerves that, among other bits of information, also carry information to and from the brain about what sort of heart rate is needed at any given moment), PDers' hearts show a diminished response, a dormancy in these nerves.¹

¹ Goldstein et al, "Cardiac Sympathetic Denervation in Parkinson's Disease," *Annals of Internal Medicine*, Vol. 133, No. 5, Sept 5, 2000, pp. 338-347 and Kaufman, Horatio. "Primary Autonomic Failure: Three Clinical Presentations of One Disease?" *Annals of Internal Medicine*, Vol. 133, No. 5, 2000, pp. 382-384.

This research was done using G-dopa as the radioactive analog in SPECT scans of the heart. The unexpected discovery of diminished heart sympathetic nerve function in people with Parkinson's resulted from studies inquiring into the orthostatic hypotension (light-headedness upon changing abruptly to a raised or standing up position) seen in Parkinson's and in several other health conditions. The researchers found that most people with orthostatic hypotension had normal hearts, indicating that the problem was coming from somewhere else in the body. But in people with Parkinson's disease, an inexplicable, measurable decrease in the heart's sympathetic nerve

Following recovery of the foot injury, if the heart's sympathetic nerve and vagus nerve responses remain diminished via a dissociation response, a partially recovered PDer's physical movement may become even *more* difficult than it was before the foot injury healed. Although the body may have become limp instead of rigid and some sensory functions such as taste and smell may have returned, frailty may rapidly set in, in an accelerated time frame compared to the normal progression of Parkinson's disease.¹

For full recovery, any attitudes that cause emotional emptiness and/or emotional inhibition of dopamine release must be overcome and new emotional habits put in place. The

signal was seen. This decrease was not related to the use of dopamine-enhancing drugs. This result was so specific to Parkinson's disease that subsequent researchers have proposed that this test may someday be the definitive diagnostic test for Parkinson's. Current diagnostic tests for PD are based on ruling out everything else: a diagnosis by default. But the heart research found that this type of diminished action of the heart's sympathetic nerve was unique to PDers.

Subsequent researchers in Italy have named this condition, in which the heart's dopamine receptors show diminished activity levels, "The Parkinsonian Heart." see: *Current Medicinal Chemistry* [2007] 14 (23) : 2421-2428; Fornai F, Ruffoli R, Soldani P, Ruggieri S, Paparelli A. "The Parkinsonian Heart is characterized by a severe loss of the physiological noradrenergic innervation and a slight impairment of central autonomic control,"

We know from the responses of recovering PDers that these nerves are dormant, and not "lost" or dead. During recovery, many PDers have been astonished to feel a sensation in the chest that feels as if "Something just clicked on in my heart!" or "Lately, I can feel a new sensation in my chest: it expands when I feel emotion!" This rapid shift in heart feeling and heart awareness during recovery suggests that the heart nerve cells have merely become dormant, not dead, just like the dopamine-producing cells of the brain's substantia nigra are dormant. These brain cells used to be thought of as dead until research proved that they had merely reverted to undefined, embryonic-type cells. In both cases, in heart and in brain, these types of cell change reflect, not illness, but the healthy body's efficiency, run along the lines of the "use it or lose it" principle. When I use the words "lose it" in the preceding sentence, remember: the cells themselves are not lost; the differentiation of the cell into a dopamine-producer is temporarily lost until such time as the cell is once again called on to be a dopamine-producer.

¹ "Frailty involves exhaustion, weakness, weight loss, and a loss of muscle mass and strength," reports Dr. Harris, head of geriatric epidemiology at the National Institute on Aging. See: Gina Kolata's article, "Old but Not Frail: A Matter of Heart and Head," *New York Times*, Oct. 5, 2006.

These symptoms of frailty are often associated with aging, but they can also occur during recovery from Parkinson's disease if the foot injury heals but the heart remains preferentially oriented towards dissociation and the inhibition of the heart's brain-connection system via the sympathetic and vagus nerves.

The above article on frailty, based on new research, reports that the onset of frailty can be the result of expectation and attitude: "Rigorous studies are now showing that seeing, or hearing, gloomy nostrums about what it is like to be old can make people walk more slowly, hear and remember less well, and even affect their cardiovascular systems. Positive images of aging have the opposite effects. . . . [In this double-blind controlled research, groups of older people were exposed to short videos in which words of either a negative or positive nature were subliminally inserted. One video had words such as decrepit and feeble; the other had words such as sage, wise, and respected. The groups performed physical and mental tasks after being exposed to the short videos. The performances of the tasks clearly showed a significant influence from exposure to the subliminal messages.] 'I am changing my initially skeptical view,' says Richard Suzman, who is director of the office of behavioral and social research programs at the National Institute on Aging. 'There is growing evidence that these subjective experiences [thoughts and attitudes] might be more important than we thought.'"

Dopamine release is known to be expectation and attitude dependent. Dopamine is the neurotransmitter that regulates the activities that go into decline in times of frailty. Considering that dopamine inhibition can be induced via a dissociation response and maintained via a negative expectation or attitude, one can begin to see how a PDer who mentally clings to a habitual dissociation response can experience a rapid increase in frailty-type symptoms after the adrenaline-producing injury begins to heal. Even if brain dopamine production and release is once again anatomically possible, a heart oriented towards the wariness mode for most of life's situations may be unable to register the heart-based *feelings* needed to trigger dopamine release. If a recovering PDer does not orient himself to positive feelings, his Parkinson's disease symptoms might decline but severe frailty can ensue in its stead.

PDer who has cultivated certain negative attitudes may need to learn how to trust positive emotional stimuli and how to acknowledge his body's internal sensory experiences. After the foot is healed *and* any necessary mental/emotional healing occurs, then dopamine release – the healthy, correct physiological response to positive emotional stimuli – will occur.

We use a very gentle technique of Asian massage to remove the foot injury. We also figured out an extremely simple technique of visualization and affirmation that can retrain the heart's sympathetic "emergency" nervous system to revert back to parasympathetic (content, joyful) mode. When the PDer learns to do this, his heart is able to once again trigger healthy emotional responses, which result in the release of dopamine. Although the retraining technique is simple, some PDers need months, if not years, to learn how to use adrenaline or dopamine in a healthy way rather than opting to mentally dissociate from the body at the first sign of worry or negative thinking.

The above is all very breezy and casual and probably does not even begin to answer most of your questions. Be of good cheer: this book is going to go over all of this again in glorious detail, complete with diagrams and supportive evidence. Also, even though many of the principles for this theory are based on Asian medical theory, I will use everyday English to the greatest extent possible, and provide user-friendly translations for any Asian terms that I simply must use. And I'm sure I won't need to translate the following statement: idiopathic Parkinson's disease is not – and never has been – an incurable illness.



“Cry out! Don’t be stolid and silent with your pain. Lament! And let the milk of loving flow into you.”

from Rumi’s “Cry Out In Our Weakness” (Bark’s translation)

CHAPTER TWO

FEELING NO PAIN: A FEW CASE HISTORIES

To demonstrate what I mean by unhealed foot injury, dissociation from foot injuries, and dissociations that linger even after the Parkinson’s is gone, I’ve selected a few representative case studies.

FOOT INJURY CASE STUDIES

Katya

Katya, age 56, was my very first patient with PD symptoms. She came to see me for gallstone troubles. After terrifying, brutal experiences with obstetric doctors in her native Russia in the 1960s, she was determined to avoid all MDs. Therefore, she had never seen a doctor about her increasing immobility, legs that felt like wood, gnarled feet that stuck to the floor, cogwheeling wrists and ankles, slow fingers, increasingly hunched posture, loss of voice and loss of sense of taste and smell. I learned of these problems as I used Asian medicine to treat her, successfully, for gallstones. She mentioned her slow shuffling walk and the profound rigidity in her legs and torso, both of which she could overcome for a few steps by using sheer will power, when she needed to. But what worried her even more was what she called “the woodenness” of her legs.

I hadn’t started this study yet. Because she didn’t have a tremor, I assumed she did *not* have Parkinson’s. I have since learned that 15 to 35% of people with Parkinson’s do not have tremor. The numbers vary depending on whom you read.

Had I known she had Parkinson’s, I most likely would have done the modern Asian medicine treatment for Parkinson’s disease: acupuncture needles in the scalp. This very modern treatment, which only provides short-term relief from symptoms, was designed to conform to the modern *western* understanding of Parkinson’s – the idea that Parkinson’s is caused by insufficient dopamine-producing cells in the brain. At this time, my knowledge of modern Asian medicine would have pointed me in this direction only.

After inserting acupuncture needles to treat her for gallstones, I was exploring the bones of the leg and feet to see if there was a reason for the “wooden feeling” in her lower limbs. I found an area on the foot that was deathly rigid, absolutely devoid of vibration and responsiveness. Because Katya was quite afraid of having her feet touched (a not uncommon PD characteristic), I used the extremely firm, supportive, slow-moving technique of FSR, a type of Yin Tui Na, which enables the practitioner to support an injured area without intruding on the patient’s desire to not be touched or “messed with.” The technique is so relaxing that Katya, like many recipients of FSR, ended up dozing, even though I was touching her usually-off-limits feet.

I slowly worked my hands over her more rigid foot until my hand came to a complete stop at the strange deathlike feeling in the center of the foot. I held that spot on her foot. I did nothing at all. I just held her foot at the acupuncture point known as ST-42 while I contemplated this unusual deathlike sensation for about fifteen minutes. And then her foot slowly began to

shake. The bones under my hands began to jostle and vibrate. And then Katya, still deep in her dozing, began to talk softly in her sweet Russian-accented English.

“She was wading in the Volga River,” Katya murmured. “She wasn’t supposed to be there. Her grandmother told her never to go down by the river. She was wading in the river. She hurt her foot. It was very bad. She wasn’t supposed to go down to the river. She wasn’t supposed to go there. She was only five years old. She hurt her foot in the river.”

Tears rolled down her cheeks. Her foot shook and her body shook. Her breathing shook as she whimpered. I continued to hold the place on the foot for over an hour. After the treatment, we discussed the long-forgotten injury.

Katya had been so terrified of being found out by her grandmother that she was not surprised that, even in her dozing memory of the event, she spoke about the injured child in the third person. She could not admit, in the beginning, even in her subconscious, that she was in fact the child with the injury. Consciously, as we spoke, she did recall the event. The faint but distinct scar where she had sliced her foot open was visible, in a good light, on the bottom of her foot. But her mental grip on the denial of her guilty secret was so tight that, even in her sleep-talking, she had attributed the entire event to some “other” little girl. It is no wonder Katya’s body had not been able to address that injury.

Little Katya had already learned to maintain a vise-like mental grip on her thoughts and emotions: at age three, she saw her father taken at gunpoint, at night, from the family home and killed in the street outside by army soldiers. Her mother had died violently at the hands of soldiers when she was a few years older. She was raised by her epileptic grandmother whose seizures terrified her and whom she was careful not to disturb in any way for fear of bringing on an epileptic event. Katya was a brilliant and talented woman who had overcome terrific obstacles in her life. Her ability to maintain her composure and leave the past behind was crucial to her success in her very rewarding life as a professional musician and choir conductor. I guessed that her ability to blot out the negative was probably also the stumbling block that prevented her body from recognizing and healing the old injury on her foot.

Lynne

Lynne’s foot was smashed in the car door. The injury was not lurking in the subconscious; Lynne had never forgotten about the injury, although it hadn’t hurt. She had assumed that the injury had healed normally. During treatment, it became evident that the foot had not healed.

Lynne was five years old when the older sister had accidentally slammed the heavy station wagon door on Lynne’s bare foot. Her tiny foot had been completely encased, the door had closed all the way. When Lynne’s father came around to see why she wasn’t getting out and opened the car door, she was sitting there, unmoving, silent. When the father began to yell at the adored older sister for being careless, Lynne insisted that her foot didn’t hurt. She stayed as still as possible and didn’t cry. Father carried her into the house and plunged her foot in ice water.

About ten minutes later, an electrical storm came up and the power went out. Father had to go looking for flashlights in the garage and then got caught up in the usual madness of a five-children family. Lynne was forgotten, sitting on the bathroom counter in the dark: motionless; not crying; with her foot in a sink of ice water for over an hour.

She never cried, but it was a memorably strange day in her young life. So many unusual things: the electrical storm, the guilt-stricken older sister, the smashed foot. Subsequent to the

injury, the foot never swelled, Lynne never limped. Except for one toenail falling off, it was as if the injury had never happened.

Forty years later, during Lynne's recovery from Parkinson's disease, her foot ached for weeks. The entire top of the foot was tender to the touch and the sole hurt to bear weight.

Curious about the injury and her recollection of the event, Lynne asked another sister about it. The sister recalled the day, confirmed the car door accident, remembered the frightening freak electrical storm, agreed that the oldest sister had felt horrible and that young Lynne had not cried.

It is worth noting that Lynne's mother could not bear to hear the children crying; she would beat the children if they cried. The mother also reacted violently to demonstrations of emotion. Lynne learned at a very young age never to feel pain or emotion. She never cried.

Lynne told me of another incident she'd had two years later, at age seven, in which she broke her right arm during a Girl Scout outing. She never mentioned the accident to anyone. Later that evening, when her mother accused her of eating with the wrong arm, Lynne lied; she mumbled that she *wanted* to eat with her left arm. After a grilling by her mother, she confessed that could not use her right arm since falling off the play structure.

Mother was furious; it was now after-hours and they could not see the regular doctor. After the after-hours visit to the doctor and the X-ray that showed the arm was clearly broken, Mother called the Scout leader and demanded to know why they had not mentioned the injury earlier.

The leader was amazed. She said, "Lynne broke her arm? But she never cried or indicated that she was hurt from her fall. She just looked a bit thoughtful. It didn't even swell up. How could we have imagined it was broken?"

Lynne had learned to dissociate from her pain so completely that, except for a one-day preference for using the other arm, she had no somatic reaction to a broken bone. Forty years later, she had every symptom of Parkinson's disease.

Hjalmar

Hjalmar, when asked about history of injury, whispered with pride, in his muffled PD voice, "I've never been hurt. Never!" During his intake interview, he had already told me that he'd served in the Navy for many years and had seen active duty in the Pacific during WW II. His nickname from his lumberjack days was 'Give 'Em Hell' Hjalmar.

Hjalmar had twinkling eyes and a jaunty bearing despite his walker and his shuffle. I was endeared to him from the start. It seemed incredible to me that he had *never* been involved in some sort of horseplay or risky event that might have caused an injury.

He was sixty years old. He'd been diagnosed with Parkinson's disease seventeen years earlier. His head was scrunched down on his torso and his hands were useless flapping fins. His voice, when available, was a gasping whisper. His right foot was a shapeless, purple-gray mass. He usually insisted on shuffling along without his walker, with the result that he fell down several times a day. His knees were twice as wide as healthy knees, the result of thousands of falls, which he always broke by dropping to his knees. "Nope," he whuffed. "Never been hurt."

This got my curiosity up. "No broken bones? No surgeries? No black eyes? No falling off a bicycle? Sprained ankle? Whiplash? Car accident? Tripping in a gopher hole? No one ever pulled a chair out from under you?"

"Nope!" Proud defiance and unquenchable good will beamed forth from the mischievous eyes, which sat, ludicrously, in that expressionless face.

After the fourth session, as his shapeless, doughy foot began to respond to the FSR, he cleared his throat. “Now, when you asked about injury,” he whispered, “you might have been thinking about the time I got hurt when I was three years old. I forget exactly what the situation was, but my granddad was visiting. Maybe I took a pretty bad fall or something, because I remember my granddad put my arm in a sling. I don’t recall exactly what it was, I think I hurt my arm and my shoulder, and maybe my leg and my foot. It must have been pretty bad for them to put my arm up like that.” He soon recalled another severe foot injury from falling off a naval cruiser ladder with his foot caught in a rung. This injury had badly twisted his foot and ankle, but it had never hurt.

The next week, he reported that a few days after the last foot treatment a dark bruise had appeared on his right foot. But more interestingly, two bruises had appeared on his right arm as well. We suspected that when the foot injury got dislodged and released that ancient memory of injury, the body spit on its hands, hauled up its slacks and decided to take care of the other injuries to the body that were lurking: a sort of package deal. At any rate, his rigid, flipper-like hand became much more flexible shortly after that visit, even as his foot began the long road back to health.

Hjalmar’s childhood

Hjalmar was outgoing and chatty, but he stiffened perceptibly when I asked about his childhood. He told me there was nothing to talk about there. “I’ve come to terms with it,” is all he would say. Hjalmar’s wife interrupted: “What about your mother?” She turned to me and continued, “Cold as ice. She was so cruel to those kids. We don’t even like to talk about it. But there’s a story there, if you want it!”

I never did probe for details. It was telling enough that my simple question “How was your childhood?” evoked such a response. As I got to know them better, both Hjalmar and his wife alluded, many times, to the heartless “parenting techniques” that Hjalmar’s mother had enforced.

Norm

Although sometimes an injury is remembered, or partly remembered as with Hjalmar, the injury history just as frequently remains as a sealed book. Norm never did remember what he had done to his right foot that caused it to be rigid, unable to bend at the ankle or even the toes. He had never even thought that it was strange that his feet moved like robot feet, unflexing in any part.

He grew up in the Midwestern U.S. and he had run cross-country in school. He’d been very good at sports, so we concluded that his foot had probably not always been so rigid. But by the time he came to me, shortly after his diagnosis at age 48, there was no question that there was something strange about that foot. He guessed that the rigidity had come on slowly, through the years. His best guess remains that it was the high school broken ankle, sustained while running cross country, which had caused the foot to become, to coin a neat simile, stiff as a board.

He did allow, “I don’t think about my feet much. My feet hurt all the time, sure, but I try not to think about them. Feet aren’t very nice, after all.” It was the first time I had heard that feet weren’t very nice, so that stuck with me, but later I wondered which came first, the injury, which caused him to conclude, eventually, that “Feet aren’t very nice,” or the idea of feet not being nice, so that when he hurt his foot he hadn’t wanted to dwell on the injury. I never went deeply

into this with him, but it was a curious sentiment coming from a sportsman whose feet had served him faithfully for so many years.

Norm came to Santa Cruz three times a year for treatment in our program. He stayed for a week each time. He was treated for at least two hours a day while he was here. It was three years before Norm's big toe suddenly was able to move, following a rotation of his 1st metatarsal. A few days after that, another bone shifted and his second toe was able to move. A few days later, his entire foot could flex and extend.

During those three patient years, he continued to fly to Santa Cruz from across the country. I asked him if he was discouraged at how long it seemed to be taking. Those feet were the most rigid of any feet I've ever seen, before or since. In reply to my question, he said, "Waal, ah figure there's Plan A and there's Plan B. Y'all are Plan A. And there ain't no Plan B."

So Norm stuck it out. More than three years after he started coming to Santa Cruz, he could once again play tennis and go for long walks.

After his cement-like feet finally became flexible and pain-free, Norm continued to be severely disinterested in being able to feel or be aware of his body. He wanted his body to be functional, but he didn't want to have to think about its existence. He was adamant that he did not want to work on anything having to do with emotions.

Even though he regained his ability to walk and play tennis, his right arm tremor never did go away. When I asked him, many times, if he wanted to keep working with me to see if he could get rid of the tremor, he always said, "No, I don't need that arm. I use my left arm now." He refused to discuss the matter.

After he was once again playing golf and tennis, I asked him, on his way out of my office, to use his trembling right hand to carry the small airline pillow that he always brought with him. He was somewhat puzzled by the request. As soon as I put the pillow in his right hand, he transferred it to the left hand. I asked him to put it back in his right hand. He did so, but within moments it was back in his left. I asked him to concentrate hard on holding the little pillow in his right hand. He found it amusing that he didn't seem to be able to leave it in his right hand. He tried very hard to keep the pillow in his right hand, but it was hard for him to simultaneously walk and hold the pillow in his right hand. He got out the doorway of my office with difficulty and approached the stairs leading to the parking area. He was baffled by the stairs. He turned to me, pillow in right hand, and asked me in all seriousness, "What am I supposed to do?"

I told him that he was supposed to go down the stairs. He asked, "How?"

I told him to just go down the stairs one at a time. This problem with stairs was utterly unexpected. He stood at the top of the landing. Slowly, with his wife's help, he negotiated a few steps. After several steps, he was breathing hard and sweating, and looking desperate. He switched the pillow to his left hand and finished going down the steps.

When he got to the bottom of the steps, I asked him to please put the pillow back in his right hand until he got in the car. He did so, and approached the car. He got to the door of the car and again became baffled. He had no idea what to do next.

I told him to open the car door. He looked painfully confused. He started to reach for the car door with his left hand – the hand that he'd been using for several years now – but with the pillow in his right hand, he couldn't figure out how to simultaneously push the door handle release and also pull the door open. He struggled with the door for several seconds, then gave up: he put the pillow in his left hand and, with his left hand, opened the car door.

I asked him to put the pillow back in his right hand after he got the car door open. With the pillow in his right hand, he could not figure out how to get into the car. After that, I never

met with him again. His wife emailed me several times: his tremor continued to get worse, to the point that it seemed as if his right hand was shaking his whole body. He sent his love, but he did not want to do anything that might involve working on emotions having to do with his right arm. The last I heard, he decided to try antiparkinson's medications to help with the tremor. He tried all of the various drugs, and none of them worked. Some of them actually made the tremor worse. After experimenting with various agonists and L-dopa, his tremor had become a monster. He got no relief from the drugs; instead, they made him dyskinetic within a matter of days (which, we have seen, is typical, once a PDer's foot injuries are gone).

In all the years that I knew Norm, with his gentle, loving smile and his gentle, polite drawl, his response to my medical inquiry, "How are you feeling today?" was always the same: "I don't know. I never really think about how I'm feeling." Probing with more specific questions could never elicit any answer other than "I'm fine" or "I don't know." Norm's wife used to laugh, "Don't ask him how he's feeling. He never knows what to say!"

Norm was never interested in being able to feel or be aware of his right arm. More accurately, he feared such feeling and awareness. Although his structural symptoms of Parkinson's rapidly evaporated, Norm's fear, tremoring, and dissociation from his right arm continued to worsen.

As an aside, I had to wonder if a person who has actually *lost* a limb but who still, now and then, imagines using the limb or who feels phantom pain in the missing limb is a mentally healthier person than a person who, terrified of acknowledging a body part, lives in ever-increasing fear of that body part or fear the pains that caused him to dissociate. The former still has a complete body image; his consciousness is healthy even though the physical body is lacking. The latter has an unhealthy consciousness: his conscious mind can only perceive himself as if is standing outside himself, or as if he is terrifyingly incomplete – even though his subconscious mind knows that he has a body, and knows that the body is wracked with pain.

TJ

I had been holding TJ's foot once a week for an hour, for about three months. Her previously numb foot had already gone from pale purple gray to a nice pink. She'd had horrible pains in her feet when feeling first returned to them. She said it felt as if "every time I've ever walked barefoot over sharp rocks, showing off that I didn't feel anything, well, now I'm feeling every one of those rocks. I was holding her foot when she started having excruciating pain half-way down her tibia (the bone on the front part of the lower leg). Within a day after the tibia pain began, it was crippling. She was unable to walk. Pain pills didn't touch it. After two weeks of her calling me every day, screaming into the phone that she was in agony, it suddenly occurred to me that she must have broken her leg. I asked her to go to the hospital and have it X-rayed.

Sure enough, the tibia was broken clear through and displaced. The radiologist told her, "You should have come in two weeks ago, when you broke it." He told her that two weeks worth of healing had already occurred at the broken ends.

TJ told him that if it was broken, she had broken it seven years ago. At the time, she had been carrying her baby while hurrying across a busy thoroughfare, dodging traffic. She suddenly saw that she was about to be hit by a bus. She had made a flying leap to the sidewalk and just missed: her leg, right at the point of the broken bone, had crashed into the curb. The baby had flown from her grip and landed in the shrubbery. Standers-by had asked if she was OK. Of course she was OK. She certainly wasn't hurt.

The radiologist told her, “You’re crazy. There is no way you could have been walking around on a broken leg for seven years.” That radiologist was wrong. I’ve since known several PDers with still-broken bones who’ve been unaware of the break until they started being able to feel.

It might be significant that, when TJ was four years old, her parents decided to get a divorce. Rather than subject TJ to the emotional strain of the divorce, they sent her away with no explanation. She had cried about something just before her parents had told her that she needed to go away. She’s assumed that the crying was the reason that her parents put her on the first airplane ride of her life, at age four. The flight took her, unaccompanied, to California, to live for many years with her grandmother, whom she had never met. TJ told me that, on the plane ride, she had made a solemn vow to herself that she would never cry again.

About a month after arriving in California, four year-old TJ was playing in grandmother’s back yard. A heavy cement table fell down on her foot. She kept her secret promise. She never cried. Almost thirty years later, I was working with TJ’s foot injury: an injury consistent with a foot being smashed by a cement table. Soon after the foot began to experience terrible pain, eliciting screams of fear and agony from TJ, she also began to experience her more recent tibial fracture.

As an aside, TJ was a horse trainer. Three of my first fifty PDers were horse trainers. I do not live in a particularly horsey area, so these numbers are somewhat curious. These three each said that they got along better with horses than with people. Also, they told me they were not afraid to punch an ornery horse in the ribs if it lashed out with teeth or hooves. “If a horse kicks me, I punch him back. It’s just playful. It doesn’t hurt. We get along.”

Chuck

After working with a few dozen PDers, I thought I had seen feet in every stage of deformity, discoloration, and distortion. But Chuck’s right foot was ghastly. When he was six years old, his right foot had been chopped off by a hay mower and then sewn back on. Fortunately the doctors had sewn it back on quickly enough so that the tissues and toes had all survived. The foot had been clumsily reattached in a pair of surgeries that would be considered primitive by today’s standards.

After it seemed like the re-attached foot would survive, the doctors did a second surgery. In hopes of giving the foot some flexibility, surgeons harvested tendons from the *good* foot and taken whopping skin and muscle grafts from his good leg. When I met Chuck, both of his legs and feet were rigid. Scars seemed to be everywhere on both feet and both legs.

When I met him, Chuck’s right leg was an inch and a half shorter than the left; he used an elevated shoe. With the limping and poor surgical alignment, his foot had become increasingly distorted through the years. When I saw him first he was fifty-nine, ten years after his diagnosis with Parkinson's disease. His foot stuck out to the side at a sharp angle; he was using what should have been the proximal (closer to the heel) part of the arch of his foot for bearing the weight that should have been borne by the ball of the foot, to the extent that it was hugely callused; and the outer ankle bone was rotated so that it was at the front of the ankle instead of on the side. The bones had grown into their new positions. His foot had the flexibility of granite.

He hadn’t let the injured foot slow him down; one of his favorite pastimes, before the PD, had been hiking. Why not? He had no feeling in his feet so hiking wasn’t a problem.

The palpable-to-my-hand Qi flow through the right foot was nonexistent. The toes were purplish grey. Some weight-bearing parts of the foot were bright red, as if mildly inflamed. He had no feeling in his feet. The Qi in his legs was running backwards. The Qi flow in the left was, if anything, even worse than the right. The right foot had been usable only as “flipper” for nearly 53 years.

I had never before been confronted with anything on this scale of physical and energetic distortion.

After many months of treatment, the foot no longer jutted to the side. The ball of the foot was weight bearing, and sensation was returning to the toes. After nearly two years, his right foot was nearly correctly shaped and could bear weight in the normal fashion, including on the newly formed callus at the ball of the foot. The callus in the arch was melting away. The bones were reshaping themselves, as evidenced by their obvious changes under the skin. He had recovered sensation in all five toes of the hay mower foot, the skin had good color and even the grafted areas had changed from blue-gray to pink.

Qi started moving through the feet after less than a year, but sensory nerves in the foot were slower to recover. It was nearly two years before he could feel the sensation of Qi moving through his big toe in response to a needle on the jaw at acupoint ST-6.

Chuck regained smoothness of movement and finesse in his left hand. He also regained the ability to smile, a matter of great importance to his six-year old granddaughter.

The above merely suggests a relationship between foot injury and subsequent foot numbness. Of greater interest to me was that his *left* side was the side that first developed symptoms of Parkinson’s. His *right* foot was the one that had been cut off and sewn back on.

Chuck’s Parkinson’s disease developed on his left side. His severed foot was on his right side. He was able to cognize his right leg and foot. The grafted skin and tendons used in the two repair surgeries had been removed from his left side. Chuck had no proprioceptive awareness of his *left* foot and leg.

Chuck had received an enormous amount of love and support after his foot accident. He had been told to be brave and not cry when he had the two subsequent surgeries that mutilated his left leg and foot while harvesting skin, muscle, tendons and ligaments. It was his left side that he could not cognize or visualize.

Rebecca

Most injuries are less shocking and traumatic than Chuck’s. For some reason I am always drawn to Rebecca’s story; Rebecca is just so sweet. She initially had no recall of any foot injury, but during our first session, as I was holding her foot, she drifted off into a sort of reverie and began telling me about her childhood, and, it turns out, her injury.

“My mother had seizures,” she told me, “and, you know, I think I was born knowing that I couldn’t make loud noises or do anything that might startle Mother. They tell me I was a perfectly behaved little girl, and that it’s a good thing, too; any sort of noise might trigger one of Mother’s seizures. I was always able to behave, and I never made noise.”

At this point, her voice grew softer, and she continued her story. “I remember it so well now. I remember exactly what I was wearing. It was a green and white striped skirt. I was wearing a white blouse with buttons down the front and a Peter Pan collar. My hair was cut in bangs, straight across the front. I can picture that green and white skirt like it was right here.

“I can just see myself, that day, playing at jumping back and forth over the railroad tracks that ran by, not too far from our house. I wasn’t ever supposed to go play by the railroad tracks. So of course, I did.

“I was five years old. I was jumping back and forth, back and forth. And one time, when I was jumping, I missed, somehow, and instead of landing on the bottom of my foot, I came down on the top of my foot. The front of my toes stubbed under and my foot bent right double under me, and my full weight came down on my bent foot so that I was standing on the top side of my toes and top part of the foot instead of the bottom of the foot.

“That foot was bent right double. You wouldn’t think a foot could bend like that, or that a little girl could hurt her foot so badly and not cry, but I always knew not to cry or make noise. Especially if I was doin’ somethin’ I shouldn’t...”

Lila

Sometimes, only rarely, in my experience, the injury is almost imperceptible except to a fairly skilled touch. The cause may remain unknown, as might the emotional reason for the failure to heal.

Lila was a forty-eight year old yoga teacher. She was devoted to healthful habits and yoga exercise, so it seemed strange to her that she was having trouble holding her arms in certain postures, and even lately having trouble getting up off the floor. All her adult life she had worked at keeping every part of her body flexible.

She did not yet have a diagnosis of PD, nor did I give her one. When, after learning about her other symptoms, I asked if she had tremor in her hand. She was a little surprised, but admitted that lately, when she held her arm up in certain postures, her right index finger tremored.

She must have thought I was asking random questions when I asked her my usual PD intake inquiries. Yes, her voice had been getting fainter and raspier, even though she sang weekly in a choir: she was starting to cut back on the solo work due to unpredictable vocal hoarseness. She assumed that, at forty-eight, her voice change was due to aging. I let her keep thinking that. (Four years later, I attended a service at which she did all the solo singing. Her voice had returned.)

Depression? Yes, despite her daily meditations and her wonderful life she was fighting to keep at bay a deepening depression.

I could see that her cheek muscles hung down limply on the right side of her face, making the characteristic PD line along the side of the nose and lips that appears when the ball-of-the-cheek muscles stop functioning.

As for her stiffness in her yoga postures and difficulty getting up from the floor, she said that it was almost as if she couldn’t think of what muscles to use when the time came to get up off the floor.

A quick exam showed me that Qi was running backwards in her right leg.

Unlike most of my patients, her feet appeared to have no flex or extension limitations whatsoever. I could not imagine that there was an injury lurking inside her graceful right foot even though she had early, but classic, signs of Parkinson’s on the right side of her body. However, despite the apparent health of the foot, I held it at ST-42 for an hour each week for several weeks. I told her only that I was treating the backwards-flowing Qi in her leg.

Despite no obvious sign of injury or a palpable sense of chaotic Qi in the vicinity of ST-42, after several sessions she had the characteristic static electricity release, followed by almost imperceptible shaking and vibrating in the foot. At that point, I had a palpable sense that something small, round, shadowy and viscous, about the size of a large pea, was inside the foot at ST-42. Whatever it was, it felt as if it was dissipating as I kept holding the foot.

Next, the bones felt as if they were moving in little circles, as if they were unwinding. If I had to guess, I might say that it was fascia tissue responding to a relaxation of micro-muscle in the area. Nothing was overtly displaced, but some tissue or energy was moving in that foot in response to the firm, nearly motionless holding technique of FSR.¹

During the next few weeks, her right foot became quite painful. She saw an orthopedic doctor who told her that she probably had sesamoid bones in her foot. He assured her that she would be in pain for the rest of her life. He suggested orthotic support devices for her shoes and told her to avoid walking uphill for the rest of her life.

When I saw her again, the next week, she asked me with some heat, “What, I can walk downhill but never uphill? What sort of nonsense is that?” Growing more expressive, she continued, “How am I supposed to walk downhill if I can’t walk back by going uphill? Should I find trails and paths that go down but not up? I should restrict myself to flatlands for the rest of my life? That’s ridiculous!”

Since she was one of the patients that did not have a diagnosis, I couldn’t tell her exactly what was going on: that she was experiencing the foot pain and other neural symptoms that were to be expected during recovery from Parkinson’s disease. I merely told her that I’d noticed some tension in her foot that had released, and that possibly there had been an old injury at that site. The place where I had been holding was the place that now hurt, after all. I suggested that the pain would go away as soon as the injury healed completely. I asked her to stay in touch and come again in a few weeks.

The pain did subside after three weeks. Her symptoms that had matched the symptoms of early Parkinson's disease began to ease up quickly and then disappeared.

She never had any idea how she had hurt her foot. Her foot had never appeared to be physically impeded in any way. But backward Qi flow originating at ST-42 had been flowing up her leg, and there had been an unmistakable release of energy and static, characteristics of injury release, from the center of her foot during her treatment.

¹ I am often asked if I have heard about the newest or latest light-touch therapies. Yes, I am familiar with many of the types of light-touch therapies, including “unwinding” – which might be used to describe what happened in Lila’s foot. However, I was not using any special “unwinding technique.” I merely used the word “unwind” in trying to explain Lila’s response to the treatments. I am intentionally not referring to any particular “named” therapies in this book.

Nearly all of the light-touch therapies are based on the exact same principles, even though their “discoverers” create special vocabulary and sometimes even insist that they have “invented” some aspect of gentle support. Also, these “discoverers” sometimes teach the student to be focused narrowly on a particular location or type of tissue that the discoverer has found to be “the key to nearly all problems,” thus preventing their techniques from having an adaptive universality. I prefer to keep the theory more generalized and adapt various supportive holding methods to the needs of the patient. Besides, all of the light-touch techniques use the exact same principles. I have to wonder, what sickness have we incorporated into our society that we need to take classes to learn how to gently hold and support a person who has been injured? All of the light-touch techniques are just stylized forms of hugs, and of the grooming, holding, and licking that “dumb” animals do for themselves and each other. By giving a technique a name, we make it “medical,” and therefore socially acceptable, I suppose. But this process also makes these techniques the domain of those who have the Special Training. The truth is, we all have an inherent knowledge of how to do these “newly invented techniques.”

Lila's case is an important one, for it demonstrates that the bones may be in what appears to be their correct positioning even though some element of retained injury remains in fascia, micro-muscle, or tendons. It is also important because Lila recalled a contented childhood and parents who were supportive and attentive even though they both struggled with severe depression. Lila never remembered any fearful event associated with her foot injury or any reason for inhibiting her pain, although she clearly had had a foot injury at some point and it had failed to heal. She recovered easily, with no emotional outbreaks or any behaviors to suggest that she'd been harboring any dissociation from her foot.

Gus

Two years after he was diagnosed, Gus came to the free PD clinic (1998 to 2002) at the local acupuncture college. Gus was treated by Doreen, an acupuncture student.

Gus was 78 years old. He had no idea of when or how he might have hurt his foot. During his first treatment session, he wondered out loud what he might have done.

During his second session, while Doreen was holding his foot, he suddenly said, "Ouch! I dropped an ammunition box on my foot right at that spot. It was during the war." He didn't say any more about it.

During the next session, he mentioned the war injury again. Then he started shaking: not in a Parkinson's disease tremoring way, but like someone who is severely chilled. Then his head started to hurt and his stomach was in terrible pain. He asked her to stop working on his foot. He felt nauseous. He began to panic. He got right up off the treatment table, shaking violently and holding his stomach. There was a look of horror in his eyes. He lurched to the front office and telephoned his wife to come pick him up immediately. After she arrived, he sat for a while in the outer office, clutching his stomach and shaking violently, trying to steady himself enough to walk to the car. He was terrified.

He called Doreen the next day and said that the dizziness, nausea and shaking was getting worse. He didn't think he could come back. Doreen was concerned and called him each day to follow up.

Finally, after several days had passed, he told her over the phone about his foot injury. That day in Japan, when he'd dropped the ammunition box on his foot, was the day every single person in his platoon had died except for him. He was the only survivor. He had never talked about it, not even with his wife. He never thought about it. He had not remembered it since the day it happened. He didn't want to remember. He couldn't bear to remember. As the sensations in his foot, triggered by Doreen's work, came to the surface, they were accompanied by the memories of that fateful day. He thought he would die from the pain and fear.

Over the next two weeks, he discussed his future options with Doreen. He never shared details of the war events. He wanted to know what he could do to stop the memories that were still overwhelming him. He was not afraid of worsening Parkinson's disease. He was afraid that he would keep remembering more and more details. He came to a decision: he would rather have Parkinson's than risk bringing up any more memories. "I'm old," he told Doreen. "I was only diagnosed two years ago. I may not live too long, and my Parkinson's is pretty mild. I would rather have the tremor and the stiffness and go out that way. I'd rather live with Parkinson's than live the last years of my life not being able to escape from the war. My only fear is that, now that you've opened that door, I won't be able to shut it."

He and Doreen agreed that he had a better chance at re-closing the door to his war-time memories if he did not seek further treatment for his Parkinson's disease.

We heard from him a few years later. His Parkinson's was worsening *very* slowly. He didn't say anything about his war memories.

Summary of the above case studies

The people in all the above cases had unhealed foot injuries. More curiously, most of them also volunteered some mental decision or emotional reason for having “not felt” their foot pains at the time of injury.

RECOVERY SYMPTOMS

In all the above cases, when the injury started to heal, each of the patients experienced some number of symptoms that we have come to call “Recovery Symptoms.” These symptoms included: tingling or stinging sensations in the toes and foot, similar to the stinging that occurs while recovering from frostbite; visible improvements in circulatory function in the injured foot; improved temperature regulation in the foot. All of the preceding symptoms that might occur in the foot might also occur in the face. Muscles that had been somewhat numb sometimes went through a short period of spontaneous spastic or dyskinetic muscle function until brain-to-muscle coordination had been restored. They experienced an improvement in sleep patterns and the relaxation and even temporary limpness of previously rigid muscles. They experienced effortless, lasting improvement in posture, increased sensitivity to bladder signals, resumption of normal-speed movement initiation, and cessation of tremor.

The cessation of tremor was sometimes preceded by the fleeting sensation as if the left and right brain hemispheres were vibrating and rotating position relative to each other. At the time, these sensations led people to presume that they must be dying. The utter stillness that they felt inside after the vibrating ended seemed to confirm that they were indeed dead. They soon realized that they were still alive, usually by noticing that they were still breathing – and also noticed that their *ability* to tremor was permanently gone.

These changes were often painful and not at all what the patients were expecting – or hoping for. They had assumed that, if the Parkinson's went away, they would just feel great, the way they used to. Instead, they often found that they were far more susceptible to physical aches and pains. They felt everything, including their overall body awareness, far more deeply. As they regained more awareness and sensitivity in their fingers, arms, neck, torso, and skin, it became evident that, in many cases, the PDer had been numb to more than just the foot injury.

Also, many recovering PDers experienced dramatic changes in personality: crying more easily; feeling “touched” more easily by charming or beautiful sights, sounds, or experiences; being far less worried about committing social solecisms such as tardiness. They often became less self-conscious and more forgiving of their own imperfections. Sometimes, as they became more aware of their bodies, they also became more aware of a physical sense of positive responsiveness to the presence and behaviors of others. Their understanding of how they were perceived by others also became deeper.

Not everyone went through these personality changes. A few patients, such as Katya and Lila, were unlike most of the other PDers, inasmuch as they had never lost their ability to feel a sense of expansion in the chest in response to beauty or grace, or their ability to cry. Their personalities were the same before and after recovery.

However, in some cases, although the PDer's body seemed to experience unmistakable, visible recovery symptoms, the personality did not. In these cases, the PDer retained characteristics of the Parkinson's personality: harm avoidance, inability to cry, excessively analytical behavior and/or inability to *imagine* himself moving. These cases suggested that the dissociations that had allowed the foot injuries to stay in place were still alive and well, even though the foot had been allowed to heal. These lingering dissociations or tendency for dissociation seemed to evoke symptoms of parkinsonism during times of stress or fear. The cases in the next chapter will demonstrate.



Cowards die many times before their deaths; The valiant never taste of death but once.

- Shakespeare's Julius Caesar

CHAPTER THREE

STILL FEARING PAIN

PARTIAL RECOVERY CASE STUDIES

Chip

Chip experienced most of the foot, face, and body recovery symptoms, including recovery from the constipation that had plagued him for years. He had not yet felt the “brain shifts” that can occur during recovery, but his tremor was greatly reduced. He was once again able to chase his nephews around the dining room table. He was certain that he was recovering.

However, the morning that he had to see the dentist for some serious tooth repair work, his wife found him lying on the living room floor, curled in a fetal position, trembling violently – far more violently than he had ever tremored before. He was unable to initiate movement, unable to open out from the fetal position. He relaxed slightly after his wife canceled the dental appointment but he remained extremely shaky and barely able to move for most of the morning. Over the next three days, he slowly came out of his panic. When he saw me the following week, he announced, “The Parkinson’s is back. You obviously didn’t get rid of all of it. There must be more foot injury that you didn’t clean up. Or maybe there’s another injury somewhere else, maybe in my arms or neck.”

After our session, he felt fine once again. The next week he said that he’d been able to chase his nephews over the tidepools at the beach, a feat that requires excellent balance and timing. Except for a faint tremor now and then, his Parkinson’s symptoms were gone. But two weeks later, on the day of his rescheduled dentist appointment, he was back on the floor, fetal position, trembling violently.

The next time he saw me he complained that I still hadn’t gotten to the root of the Parkinson’s disease. I suggested that maybe there was a psychological angle, something to do with dentists. He disagreed. He insisted that he had *no* fears.

I said that Parkinson’s disease wasn’t something that came and went. I pointed out his improved posture, gait, arm swing, facial expression, everything related to Parkinson’s, and he confirmed that he didn’t have Parkinson’s except on his go-to-dentist days. He also pointed out that, when he was lying on the floor, immobilized, trembling, he had *all* the symptoms of advanced Parkinson’s disease. Parkinson’s is defined by its symptoms. If he had the symptoms, then he had Parkinson’s. I was the Parkinson’s specialist; I should fix him.

He had the dentist reaction several more times. He also started to have a new trigger for his bouts of parkinson-like symptoms: *thinking* about still having Parkinson’s. Whenever his mind strayed to the thought that his Parkinson’s wasn’t gone after all, he would start to tremor and stiffen up. His feet stuck to the floor and he could barely walk. If he got distracted from those thoughts, the symptoms went away. Over the next year, he became almost constantly preoccupied with the fear that the Parkinson’s would never heal. Every time his mind touched on

this subject, he stiffened up and tremored. But even so, if he “forgot to have Parkinson’s,” as he expressed it, he could move normally.

Team members and I continued working with him for more than a year. But then he applied for disability pay and stopped seeing us.

Hope

Hope experienced a collection of recovery symptoms, including a small spinning sensation in her head, but not the full brain shift. Some of her symptoms cleared up completely, others were greatly reduced. She still walked with the slight limp in her left hip that she’d had all her life. Her small, intermittent tremor departed her left hand and settled in her right hand. Her vigor and facial expression returned completely, and she started riding her bicycle again for the first time in years. For several years it seemed as if the Parkinson’s was on its way out.

However, within hours of learning that her best friends and their daughter had died in a car crash, her tremor became larger and nearly constant. Over the next two years, she found herself moving a bit more slowly.

But one week, when she showed up for her weekly session with me, she reported something odd: on the previous Friday, she’d woken up with a tiny bit of a head cold. She wasn’t sure if she would go to work or call in sick. She puttered around the house for an hour and decided to go back to bed. As she was climbing back into bed, she realized that she’d been moving perfectly normally and hadn’t had any tremor the entire time she’d been puttering around the house.

I asked her if she had any idea why she’d been able to move normally for that hour.

“I wasn’t really sure I was going to get up for the day. I was still wearing my bathrobe, so it didn’t matter.”

“What didn’t matter?”

“Anything. Since I might not be getting up, I didn’t need to be worried about anything.”

“What are you usually worried about?”

“Nothing. I’m not afraid of anything. But I notice that when I first wake up I don’t have any symptoms until I think of what I need to do that day. And of course, I didn’t tremor much on that Sunday when the power was out.”

This was news to me. “Why not?”

“There’s no way anyone is going to call me from work and expect anything of me on a Sunday when the power’s gone out.”

“Are you usually afraid that someone’s going to call you from work? Would that be a problem?” (Hope owned the business that she worked at.)

“Well, you know how it is. They always expect something of you.”

“If no one expected anything of you, would you not need to tremor any more?”

“I don’t know. There’s *always* going to be something they’re going to want from you.”

Hope had another symptom that was steadily worsening. Like many other PDers, she had cramping in her toes once in a while. The cramping usually lasted up to ten minutes. But recently, it would sometimes last all day. She had a breakthrough when she realized that it only occurred when she wore her nice boots. She started wearing only shoes. Within a few months, her toes cramped up after a few hours of wearing shoes. She started wearing only sandals. Within a few months, her toes cramped after a few hours in her sandals. She started wearing cheap flip-flops to work. Her toes didn’t cramp in the flip-flops except for short periods of time, while she

was worried about something specific. She still wore shoes once in a while, for short periods of time, but she noticed that her toes started cramping while she was starting to put the shoes on! A few months after noticing this, she realized that something really strange was going on: her toes cramped up when she *thought* about shoes. That's when she finally realized that it was the *idea* of shoes, especially *nice* shoes, that was causing the toe cramps.

Hope told me about her mother, a cruel woman who openly resented having had children. For example, the father had never been allowed to give little Hope a hug because he "belonged" to the mother. Mother had more money than she knew what to do with, and she collected shoes. She had hundreds of pairs of shoes, many of them never worn. When Hope was a young child, she had regularly thought to herself, "You should love your children more than your shoes." Together, we managed to work through her mixed feelings about shoes, and her toe cramping decreased significantly, but she was still bitter about her mother.

Hope's tremor continued to worsen. It got so that it only let up when she was certain that "no one was going to expect anything" from her.

As an aside, she had no memory of ever having cried prior to recovering from Parkinson's. But even though she could now cry and felt much more open to feeling emotions and expressing emotions, she remained emotionally guarded. As she informed me one day when the tremor was severe, shaking both arms and her chin, "The whole point of life is to protect yourself from feeling strong emotions."

In Hope's case, her intermittent plunges into worsened tremor or stiffness were clearly triggered by thinking about work, car problems, or responsibilities in general. Her toe cramping was related to thoughts of shoes, especially nice or expensive shoes.

But unlike Chip, she never again had the full-blown collection of PD symptoms that she'd had when I first met her. Most of her symptoms never returned. Her expressionless face, dragging foot, bent arms, inability to open doors, carry things, or go up stairs without keeping a death-grip on the handrail were permanently gone.

And at some point, she told me that she'd sat up in bed in the middle of the night and said out loud, "I've been making this way harder than it needed to be." As of this writing, she is working on changing her "protect from strong emotions" credo, and she is starting to see a decrease in her tremoring.

Duke

Duke moved as slowly as molasses even though his foot injury had healed. He'd had some recovery symptoms in his face and feet. However, even though he now had facial expression and could once again initiate normal-speed *speech*, he still had difficulty initiating normal-speed large motor movements.

Before I can go into details of Duke's case, I need to mention that some patients continued to dissociate from their entire bodies in times of stress, even after their foot injuries were gone. As we saw in the case of "Chip and the Dentist," ex-PDers very often cannot move easily during these dissociative periods. During these intervals, they feel as if they are outside their bodies, observing themselves from the outside but unable to *feel* that they exist *inside* their own bodies. By the time I was working with Duke, we'd developed some exercises that had helped partially-recovered PDers learn how to stop dissociating so that they could completely recover. However, the biggest stumbling block for many PDers was forcing themselves to do the sometimes painful work.

Getting back to mild-mannered Duke, he'd just finished doing one of the more carefree and pleasant of the dissociation retraining exercises when he exploded with a rare burst of emotion. The technique he'd been doing involves lying down, relaxing, while pretending to *feel* what it's like to walk or lie in the sun while pretending to be some animal such as a kitten, pony or elephant. Most PDers enjoy this technique. After they finally master the concept that "feel the sensations" does *not* mean "*observe* yourself from the outside," they are usually able to imagine what it might feel like to be or move like an animal – even if they are as yet unable to imagine what it might feel like to be or move like a human.

After an hour of being various animals, I asked Duke if he'd enjoyed the experiment. He snapped at me almost viciously, "No! I don't *want* to feel!" Duke was stunned by what he'd just said and how he'd said it. He apologized and meekly assured me that he truly did want to feel. He added, "I had no idea. I guess I've got my work cut out for me."

For his homework, I asked him to figure out some animal or person that he was willing to feel like. The next week he told me that he'd chosen Sammy the River Otter, a favorite book character from his childhood. Sammy is happy. Sammy likes to have fun. That weekend, while "feeling" like Sammy the River Otter, Duke had easily driven the winding mountain highway that connects our beach town to inland California. He'd felt so good after driving in a relaxed and responsive manner that he drove another twenty miles to visit some friends. He'd had a great time and moved better than he'd moved in years.

But the following week, his movement was once again maddeningly slow. I asked him if he'd tried being Sammy the River Otter. He became angry again, and said, "I don't want to be anything but me. I want *me* to be able to move. I don't *want* to have to be a river otter.

I suggested that *he* didn't like feeling his own body, and Sammy did. As long as he was determined to be his old personality, a personality that, by his own admission, didn't want to *feel* anything, he might not be able to move well. Except when using adrenaline, a person has to be able to anticipate the *feeling* of moving his body, or at least be able to *imagine* movement, before he can make it move.¹

As an aside, the degree of numbness and lack of proprioception in many PDers is very high. Prior to recovery, many PDers insist that they are not numb because they are able to feel externally applied stimuli. However, when they recover, they are usually stunned to discover that they can have awareness of, that is to say, *feeling*, in body parts even when there is no external stimulation or pain coming from the area.

I suggested to Duke that maybe his current personality was just as false as Sammy the River Otter's. Maybe his *real* personality *did* like to feel his body and was also able to move perfectly normally. He started crying, and said, "But I had a perfectly happy childhood. Sure, my father was in the Navy so he left for sea when I was six months old and I didn't see him much while I was growing up, and my mother always needed everything to be just so, but I was fine with that. I'm not afraid of anything. And I *do* feel. I'm not numb."

He called me a week later and left a message: "I just realized that I *am* numb, physically and emotionally. I was really observing my wife, and I noticed how much she *feels*, physically

¹ Dopamine-induced movement, as opposed to adrenaline-induced movement, must be processed through the imaging area of the brain before it moves to the actual motor function area of the brain. People with Parkinson's disease do not activate the imaging part of their brains when they try to move. This part of the brain is also shut down immediately following severe trauma or severe blood loss. Citations supporting this new research appear in chapter xxx, which discusses brain processing during dissociation.

and emotionally. She gets excited about everything and she feels every little thing in her body. *I don't feel anything. I have my work cut out for me.*"

Rudolph

Rudolph recovered quickly from Parkinson's disease. When I lectured in Germany, he translated for me. On the second day of the talk, when the PDers in the audience got to introduce themselves and share a bit about their symptoms, Rudolph stepped up, last of all, and introduced himself as a person who had recovered from Parkinson's. Everyone in the audience was stunned. Some did not believe that Rudolph had ever had symptoms of Parkinson's disease, or else were determined that that his symptoms must have been mild.

But within a year after being fully recovered, Rudolph told us that he still had Parkinson's disease in his right arm. It was only a problem when he was shifting the gear level in his car. Still he was concerned: his greatest fear, after all, was losing his ability to drive a car.

Aside from having Parkinson's disease in his arm when he was in the car, he could move beautifully. He could run after his two sons when they played at football (soccer). But his arm stiffness while moving the gear shifter worsened. After a period of years, he was barely able to move the gear shifter. As his arm's "Parkinson's disease" increased, his fears started to build. Whenever he caught himself thinking about never recovering from Parkinson's, he felt his facial expression fade and his movement became labored. When he forgot about the Parkinson's, he could move easily. But as he became more and more preoccupied with not being able to recover, his stiffness appeared more and more often.

During his visits to Europe, Chris often treated with Rudolph. Chris Ells is the member of our team that represents our work at the Yin Tui Na Centrum of Amsterdam, a non-profit that was organized, in large part, for the purpose of supporting our work in Europe.

When Chris worked on Rudolph's arm, the arm felt perfectly healthy. Chris started to suspect that Rudolph's lack of arm use had more to do with the fear of not being able to drive than it had to do with the arm. On one of his visits, Chris led Rudolph through a long series of movement exercises. Cleverly, without drawing attention to it, Chris led Rudolph through every one of the movements that are used while moving the gear-shift lever. Rudolph did all of them easily, fluidly. Rudolph was sitting down, exactly as he would be in a car. Chris, pretending to be assessing strength, had Rudolph do the same gear-shifting moves while Chris pushed against him, creating resistance.

Rudolph did all the gear-shifting movements beautifully, effortlessly.

When Chris was finished, he explained to Rudolph that his arm was fine, and that the problem had nothing to do with the arm, and nothing to do with Parkinson's disease. Parkinson's disease symptoms *do* worsen or lighten up with mood, but not to the extent that Rudolph's symptoms manifested. What Rudolph had was an arm that didn't work when he was in the car. Rudolph admitted that his greatest fear was losing his ability to drive, but he didn't see why that was related. It was the Parkinson's, and not the fear, that was causing his arm to freeze up in the car.

Chris suggested to Rudolph that his fears and negative thoughts were causing him to manifest symptoms of psychogenic parkinsonism. Rudolph asked how often Chris would need to perform Yin Tui Na on his already healed foot in order to make the fears and negative thoughts go away. Chris, and then later, I, assured Rudolph that we could treat him for anxiety and fear, and guide him through some psychological exercises that might help him. But we also made the

point that, ultimately, overcoming his fears was his own job. He was actively creating fears. He needed to stop doing that.

Rudolph was stunned. He told us that it had never occurred to him that he was responsible for his own thoughts. He said, “I just assumed that, if thoughts showed up, they had the *right* to be there. I assumed that I was *supposed* to pay attention to them. It never occurred to me that *I* was responsible for the nature of my own thoughts.”

Rudolph was well educated, a published author, and a long-time practicing Buddhist and, like most people with Parkinson’s, extremely intelligent. Also, like most of our patients with Parkinson’s, he assumed that his *actions* should be under his own conscious control but that his *fears* were *real* things: his fears were legitimate; his fears were *not* mental constructs. His fears, like this thoughts, simply *were*: he could not be expected to have any amount of control over them.

We walked Rudolph through some of the exercises we’ve developed for learning how to experience physical or emotional feelings without reverting to dissociation at the first sign of pain. We started by encouraging Rudolph to feel the physical pains of experiences that he had already “processed” mentally and that he hadn’t considered traumatic at the time. He cautiously allowed himself to actually feel and then neutralize the physical knot in his stomach that he’d *never felt* when his girlfriend dropped him for his best friend back in high school. As he *felt* and neutralized this and an assortment of others pains, he started laughing.

While slapping himself on the forehead several times in a gesture of sudden realization, he exclaimed sheepishly, “I’ve been doing this to myself! I’ve been doing this to myself!”

Summary of the above four case studies: Chip, Hope, Duke, and Rudolph

These cases, and many others like them, suggested that some people with Parkinson’s who’d recovered from their foot injuries, who’d experienced unmistakable recovery symptoms, nevertheless still had a strong mental or emotional trigger that caused them to tremor or freeze up in some body part now and then, or to even have a body-wide episode of immobility in certain circumstances. Sometimes these people moved perfectly when they weren’t having an “attack” of Parkinson’s, as some called it. Of these, many noticed that their worst times were when they started thinking about having Parkinson’s – which they thought about more and more. Many also used the exact expression, “Forgetting to have Parkinson’s” to describe those times when they move perfectly normally.

Many of them noticed that the PD symptoms that are directly caused by the electrical chaos set in motion by the foot injury, such poor circulation and muscle function in the foot and face, a decrease in sense of taste and smell, poor temperature regulation, and foot dragging, did *not* return. And when they *were* moving easily, they often moved better than they’d moved in years.

When they were moving easily, they often reported that it was the first time in *decades* that they’d moved with dopamine. Movement with dopamine is somewhat effortless, and *feels* distinctly different from adrenaline-based movement. If one hasn’t experienced dopamine-based movement in a long time, the difference is quite distinct, even “impossibly” effortless.

For example, one recovering PDer told me that when she’d gotten up off the sofa recently, it didn’t take any work. She had merely thought about standing up, and the next moment, she was standing up, as if “by magic.” She burst into tears and bitterly asked out loud of the empty room, “Is this how easy it’s always been for everyone else?” Although she had not developed Parkinson’s until she was in her forties, she had *no* adult memories of ever having

moved without first consciously willing herself to activate her limbs. She had always moved with intention and will power. She'd had no idea that most people moved by imagining the feeling of movement and then letting the body express that thought.

The changes that we saw in recovering PDers suggested that they had healed from their injuries *and* that they *could* access dopamine for movement initiation.

However, when some of the recovering PDers started to manifest the more "mood influenced" symptoms of Parkinson's, such as tremor and immobility due to a fear-based inhibition of dopamine, their symptoms sometimes became far more problematic than they had been previously.

We hypothesize that this abrupt worsening of symptoms during times of fear may have been due to a decrease in adrenaline levels. When the foot injury healed, it was suddenly much harder for many recovering PDers to summon up the adrenaline and the adrenaline-based personality that they'd always used to make themselves "get going."

After the foot injury healed, if they panicked, they felt as if they had neither adrenaline *nor* dopamine. Thus, when some of them, like Chip, were panicked or anxious, they were *far* worse off than they'd been before, back when they'd merely had idiopathic Parkinson's disease and a vigorous habit of relying on adrenaline.

We slowly came to appreciate the significance of some of the partially-recovered PDers protesting vigorously that they did *not* want to learn how to feel and they especially did not want to learn how to feel pain. Several PDers pointed out to us that the "whole point of life is avoiding pain." But when these people did overcome their tendency to dissociate at the first hint of unpleasantness, they were delighted, amused, and usually a bit sheepish. Everyone who recovered from the dissociation component admitted that it was simple, and that they had been solely responsible for setting it in motion.

As an aside, we found that those people who were determined to blame parents, politics, or circumstances were also unable to change their mental habit of dissociation. Only when they started taking responsibility for changing their own thought habits were they able to stop dissociating from their bodies.



“Chiefly the mould of a man’s fortune is in his own hands”

Francis Bacon

CHAPTER FOUR

ABOUT THE CASE STUDIES

SOME GENERALITIES

While working with hundreds of PDerS, I saw many examples of the same injury-denial pattern that I’d seen in the first dozen people with Parkinson’s. The injuries described by my patients were not usually the typical childhood or young adult foot injuries that cause whimpers, tears or screams and which are answered with hugs and kisses. These were *not* injuries from which a person *limped* away with a cheery determination and a defiant cry of, “Don’t worry, I’m OK!”

These PD-causing injuries, even at the moment of injury, may have behaved as if they had never happened. They may not have bled outwardly or internally. These injuries may not have swelled up.

The person receiving such an injury may have said nothing at all *or* he may have called out calmly, “Nothing happened.” The difference between the PDer and the typical person who calls out with bravado, “Don’t worry, I’m not hurt,” is that the PDer may have *truly* believed that nothing had happened.

The person who immediately dissociates from an injury may not even be able to cognize that he was injured. And even *if* he was aware of the injury, he may have dissociated from his ability to *feel* the injury: if he had conscious awareness of the injury, he might have felt as if the injury happened to someone else – a numb version of himself.

All of the PDerS that we’ve seen had unhealed injuries in their feet. Very often, the pain, and in some cases, the event, of these injuries had been blocked out of the mind. The injuries have never healed. In *many* cases, the injury had not followed a normal injury pattern of swelling, bruising, bleeding or pain. Instead, the injury froze in place and remained, ever since, as if in a state of suspended animation.

In some cases the complete follow-through of the injuring force never even made it all the way through the injured foot. I have worked on feet in which I found bones that were displaced, frozen into an incorrect position. After such a bone loosened up a bit, *prior* to resuming its correct position, it moved even further in the direction of the injury as if it was completing the follow-through pattern of the blow, before it eventually rebounded back to its correct position, started bleeding internally, and started to hurt.

Lack of a sympathetic hug

Many Parkinson's disease patients feel that they did not have any person in their childhood to whom they could go for sympathy and commiseration in the event of an injury. Others recall sympathy from parents but either hostility towards weakness or praise for “sucking up the pain” from one or both parents.

“Don’t show your weakness” was a common childhood theme for the majority of my Parkinson's disease patients. A not uncommon threat in the childhood of many PDerS is “Don’t cry; if you do, I’ll *give* you something to cry about.”

Others had a kind and loving guardian, like Katya. As an adult, Katya was very loving towards others and felt strongly the power of the music that she conducted. Still, the violent experiences of Katya's youth and her fear of her grandmother's epilepsy may have been enough to teach Katya to not feel her *own* physical or emotional pain.

Cruelty in the home

Other PDers had situations much worse than lack of sympathy. Many of my Parkinson's patients have shared blood-chilling stories of violent parents, stepparents or guardians.

One told me he had frequently been thrown head first into the wall starting at age five.

Another told me about a game that her father used to play with her in which he would pin her on her back and then force her legs apart, holding her bent knees all the way to the floor until she cried. He called it "tickling" her. The goal of the game was to make her cry. Only when she cried would he stop. She remembers refusing to let him have the satisfaction of seeing her cry. During her recovery, as Qi started flowing through her legs, she became nearly paralyzed for over a year by excruciating pains in her hips.

Finally, after a year of agony, she had a scan done of the hips. The psoas muscles were torn laterally (from side to side), an incomprehensible location for a psoas tear. Though they were not torn all the way through, the area all around the tears was badly inflamed. She took muscle relaxants and pain pills for nearly a year while the psoas muscle rips healed.

Her memory of her beloved father is that he was a good, kind man.

Histories of PDers with wonderful childhoods

Some PDers have told me that they had wonderful childhoods. They too, however, have evidence of an unhealed foot injury. Why did they dissociate from the injury to the extent that the foot was unable to heal? Sometimes the dissociation has nothing to do with family or upbringing: Gus, the PDer with the ammunition-box accident during the war, recalled a particularly wonderful childhood.

One patient who recalled only positive experiences about her childhood did remember not wanting to show injury when she hurt her foot. She was at college, partying with the guys from the swim team. They were trying to do some prank involving a railroad tie. When a car unexpectedly appeared down the street, the boys abruptly dropped the tie. It landed squarely on the center of her foot. She didn't want the boys to think that she was a wimp. She told herself that the injury hadn't happened, and it was as if she'd never hurt it.

Her case had a quirky touch to it: when I first saw her foot, I noticed a tiny shark tattoo on her foot – right over the end of the Stomach channel at acupoint ST-42. I asked why she had chosen that location, and why a shark. She laughed, "Oh, I don't know. I guess I think of sharks as protectors." I asked her why she wanted her foot protected at that particular spot. She replied, "I have no idea. I just did."

Finally, some PDers had no memory of any reason to dissociate from an injury, plus no *overt* indication of injury, such as displacement of bones. Nevertheless, in our experience, he *did* all have some kind of soft tissue knot, muscle tension, or some other sort of foot problem that hadn't healed.

Nails and bicycle spokes

PDers sometimes assumed that their unremembered foot injury must have been something profound or dramatic. This was not the case. Mundane events like nail punctures and bicycle accidents were at the root of many of my PDers' unhealed injuries. Many, many times a PDer has suddenly recalled a nail through the center of the foot while I was holding his foot at ST-42. I can't even guess at how many times I've heard the nail story. What made these nail injuries live on to cause Parkinson's was the manner in which the recipient thought he needed to behave *in response* to the injury.

Another common injury in Parkinson's disease case histories is the foot-in-the-bicycle-spokes accident. Spinning bicycle wheels can exert a tremendous torque on any object that accidentally slips between the whirling spokes. Many patients, during treatment, have suddenly recalled a foot in the spokes injury. But more importantly, they have often recalled a simultaneous embarrassment, shame or lapse into attitudinal training that caused them to deny the very existence of the injury.

The significance of the event isn't necessarily related to whether or not the injury was bizarre or horrendous. The significance is that the PDer dealt with the injury by dissociating from the pain. The result of the dissociation was that the injury could not heal.

A FEW QUESTIONS THAT ARISE

Before closing this chapter and beginning the chapters that describe how, exactly, reversed Qi flow can cause dopamine inhibition, I want to assuage the most common fears and answer the questions that most often arise when people learn that a foot injury is a causative factor in idiopathic Parkinson's disease.

I hurt my foot once. Am I at risk for Parkinson's disease?

People often ask me if they will develop Parkinson's disease because they recall having received a foot injury at some point. I tell them that it is most unlikely. Everyone has injuries to the feet and ankles, but most people do not develop Parkinson's disease. The problem with the feet in PDers is that they've never healed; the PDer can't visualize his injured foot, he can't imagine light in his foot, he can't really *feel* his own foot – and hasn't really felt it since he *decided* that he should deal with his injury by having it not hurt or not exist.

In and of itself, a foot injury does not cause Parkinson's disease. The inability to process the foot injury and the subsequent electrical changes that occur over decades because of the unhealed injury are causes of Parkinson's disease.

In further answer to the question, "If I hurt my foot will I get Parkinson's someday?" I reply that, even if a person has a foot injury that never healed correctly, if he was *able* to fully feel the pain of the injury, he probably doesn't need to worry. His injury is probably not the type that we see in people with Parkinson's disease. A person who *feels* his long-term injuries and does his best to heal them or favor them or send a little love or attention to them will always have energy flowing to the site of the injury in an attempt to fix the situation.

The problem in Parkinson's is that the injury cannot heal because the mind is pretending the injury doesn't hurt, or doesn't even exist. This is a very different situation than a foot that never heals quite right from a frightening or painful – and acknowledged – injury.

As a purely theoretical aside, the Heartmath Institute of Boulder Creek, California, does research that involves measuring brain and heart electromagnetic wave patterns and heart rate

variability patterns. They have found relationships between these measurable patterns and emotional states. They have developed various techniques of imagining the heart and paying attention to how one *feels* in response to pleasant memories. These imaginings and the increased heart awareness help to entrain the heart and brain wave patterns. When these patterns are entrained, a person is in parasympathetic mode instead of sympathetic: dopamine-releasing mode instead of adrenaline-releasing.

This highly respected organization teaches these techniques to groups ranging from CEOs to fourth graders. They have a very high degree of success. *Ninety-six percent* of the people in their programs are able to perform the simple heart-brain integration exercises, and notice the benefit: a sense of calm and well-being.

I have to wonder about the other four percent. By age seventy, four percent of Americans manifest symptoms of Parkinson's disease. When I asked my patients with Parkinson's disease to try the Heartmath techniques, most of them were utterly unable to understand the very simple instructions. People with Parkinson's tend to be extremely intelligent. But most of my patients had no idea how to even begin *thinking* about following the instructions. As soon as I said the words "heart" and "imagine" in the same sentence, I lost most of them. I have to wonder if the four percent who cannot perform the Heartmath exercises grow up to become the four percent who have parkinson's disease by the time they are seventy.

It may well be that nearly everyone gets foot injuries. The people who get foot injuries who have dissociated from their ability to feel pain may be the ones whose foot injuries fail to heal. These might be the ones, who, if they live long enough, also go on to develop Parkinson's disease.

"I remember hurting my ankle, but not my foot."

The center bone of the foot, also known as the second or "intermediate" cuneiform, is the bone that "travels" the most during every footfall. Due to the interconnected configuration of the foot, injury to almost any part of the foot ends up displacing, at least temporarily, the center bone of the foot. This spot bears the brunt of many foot injuries, whether the point of impact occurs at the toes, ankle, or the sides of the foot.

Due to the elegant precision of the current diversions that should take place at the center bone of the foot, an unhealed foot injury at the very center of the foot can, over time, set in motion a dangerous collection of electrical aberrations. These electrical aberrations mimic the electrical pattern of a severe injury: an injury that requires inhibition of dopamine so that the injured party will be immobilized during the early stage of healing.

While a moderate injury to the thigh, the torso, or the upper arm may be problematic, they will not set in motion backwards flowing currents. If these larger areas are injured, the local currents are able to flow around and past any electrical glitches. An injury in these areas will not set in motion the electrical aberrations that cause Parkinson's disease. An unhealed center-of-the-foot injury, however, might spell long-term trouble. The reasons for this will be detailed in chapter xxx.

"My foot bones are fused and/or wired together. Is there any point in trying to recover?"

The problem is not actually the foot bone displacement, per se. The problem is that the currents that run through the foot are distorted. We've worked with many patients like Chuck, whose feet are not completely "curable." By using Yin Tui Na, we've been able to bring the patient's attention to the foot while not actively doing anything to the foot. We've found that,

when a person's awareness starts to go to an injured area, the body's natural tendency is to *first* heal the patterns of electrical flow in the area. Whether or not the bones or tissues can ever completely heal is secondary. As soon as the electrical pattern ceases to run backwards, the brain's inhibition of dopamine can cease. In other words, if the body can create some satisfactory way for currents to flow past the injured area, the dopamine-inhibition and other symptoms of Parkinson's will cease. But the body cannot create those healed patterns until the mind is willing to pay some attention to the problem.

What if I don't remember the injury? Can I still recover?

Not everyone could remember the injury that set the Parkinson's disease in motion anywhere from fifteen to sixty years after the injury occurred. Many patients racked their brains trying to remember a significant injury. They asked old friends and family members, but come up blank. Some admitted that there were just so many injuries, and they never did pay much attention to any of them, that it would be impossible to single one out as the most likely culprit for triggering the Parkinson's disease.

Some patients felt bad about not being able to remember. Others were concerned that, if they couldn't remember, the Parkinson's disease wouldn't go away.

Actually, remembering the injury didn't seem to matter. If I had to guess at the numbers, I would say that about three fourths of my patients have, sooner or later, remembered their foot injury. Some remembered the injury during treatment. Others have always remembered it. About one fourth never did recall anything in particular. Apparently it is not a requirement of recovery that one must remember the injury that set the channel Qi running backwards.

It appears, thus far in our research, that as long as the Qi of the foot is restored to its correct flow pattern, the electrical currents will be able to run correctly again. Even PD patients who could not recall a specific instance of foot injury are walking around today completely relieved of any symptoms of Parkinson's disease.

Is the injury always visible by X-ray?

Is there any way of proving that there is a physical displacement?" The answer is "sometimes."

I included Lila's case because she had absolutely *no* displacement of bones whatsoever in her graceful foot. The foot moved easily in every position. The tension was in the soft tissue, not in a wrong bone articulation. It was only by holding the center of her foot patiently for a few hours (spread over several weeks) that the holding pattern in the soft tissues relaxed.

As in Lila's case, it is possible for the bones of the foot to be more or less in the correct position relative to each other, even if there is an injury in the area. Sometimes there is only a very subtle displacement. An X-ray may not reveal anything out of place in these cases.

Some PDers feet have glaring displacements. In our patients, the most common displacements were in the bones around the intermediate (2nd) cuneiform bone: the navicular, the proximal ends of the 2nd and 1st metatarsal, and the 1st and 3rd cuneiform. Some of these might have been visible in an X-ray. Other displacements are not uncommon, or may exist in combination with the above "most popular" sites for displacement.

If a person wants radiological proof of displacement, he might want to pay close attention to whether or not any of the cuneiforms have shifted too far towards the dorsal or the plantar side of the foot. These bones are supposed to be able to move up and down. If one gets jammed, it is usually after moving too far up or down and then not being able to drop back into place. Then

again, an X-ray may *not* show proof of injury even though the foot is jammed stiff and the foot bones are clearly unable to articulate correctly.

And while discussing X-rays, another PD patient, during the course of treatment, began experiencing terrible pain in his ankle. He finally had it X-rayed. There was a large *displaced* break in the ankle. He knew how it had happened: he had dropped a box spring mattress on his ankle while trying to muscle the mattress up a narrow stairwell. He and the mattress had both gone crashing partway down the stairs. This had occurred six years earlier. The doctors who did the ankle X-ray after the severe pain started could not understand how he had been bearing weight on that ankle for six years with the bone in that condition.

Isn't denial of injury and emotional pain a sign of maturity?

People with Parkinson's disease typically have tremendous mental control or did when they were young. It requires ferocious mental mastery to *choose* to not feel physical or emotional pain, a mental stance that allows the PDer to “not be hurt.”

The PDer may imagine that when he says, “I shall not be hurt!” his body *is* not hurt. But in fact, if an injury has occurred, the body *has* been hurt. Even before we came to understand the dissociation response that makes this denial possible, I pointed out to many PDers that inability to acknowledge an injury is not the same as *not having had* an injury, and it is not the same as mentally *healing* the injury. The denial of the pain of the foot injury that appears to be a commonality in Parkinson's disease can *prevent* the healing of the *very real* damage.

The person who adapts this mental posture is building upon the survival mechanism of holding on to an injury until such time as it is safe to let go. However, he takes is a step further: he never lets go. This is *not* a healthy, long-term solution to injury.

In the days when people only lived thirty or forty years, denial of pain may have been an effective short-term solution. But now we are living longer, long enough that our bodies can exhibit the slow-to-develop side effects of unhealed injuries. Our research suggests that using selective dissociation to inhibit the ability to feel a very real injury can lead to the type of dissociation that shuts down neurotransmitters and makes the body increasingly rigid: ultimately leading to the Parkinson's syndrome, which is far more devastating than the original injury might have been.¹

¹ Researchers have pondered the increase in the number of Parkinson's disease cases in the last few decades, and some have made the blind guess that this can be attributed to environmental pesticides. They should look closer to home, on the bodies of the patients themselves. The telltale signs of backwards-flowing Qi have other pathological manifestations besides Parkinson's disease. *Years before the PD was diagnosed*, many of my PD patients and a relatively higher percent of my younger PD patients have had a tumor, cancer, sarcoma or melanoma removed that was located exactly on the narrow route of the Stomach channel, on the same side of the body where the PD eventually appeared.

Irregular electrical patterns of Rebellious (backwards-flowing) Qi can create aberrations in DNA expression. This leads to irregular cell growth. It may well be that the faster-developing types of PD are so electrically powerful that the Rebellious Qi generates electrical aberrations potentially more dangerous and lethal than PD: cancer.

Possibly, until recently, many people with subclinical PD died of cancer long before Parkinson's symptoms were discernible. Now, in modern times, these superficial (skin based) growths are usually detected early and removed. The patient assumes that the growth was just a spontaneous, strange event, which occurred out of nowhere.

In fact, it may be that the reason that there seems to be more PD in these recent years is that other side effects of rebellious Qi, such as cancer, which used to be fatal in the past, are now successfully treated, so that the

Then again, if parents or caregivers for the child were violent enough that the child feared for his life, the child did the right thing, possibly a life-saving thing, in dissociating from his ability to feel pain, and activating in himself an extreme rigidity or stillness during those times when his slightest movements might have triggered or increased the rage or violence.

Some PDers have, during recovery, remembered that they practiced consciously dissociating from their physical and emotional pain as a response to the non-life-threatening pain of school-yard rejection. Whether the dissociation was in response to a life-threatening injury, fear of parental response, or the emotional agony of childhood rejection doesn't seem to matter. If a person practices cutting himself off from his ability to feel his own physical and emotional pain, he may well activate the same dopamine-inhibiting processes that occur during severe trauma. If he practices it enough, it can become a habit.

And if he practices it with regard to an injury, the body will not be able to heal that injury.

CONCLUSION

In our limited experience, every person with in idiopathic Parkinson's disease had an unhealed foot injury. We now assume that denial of the physical or emotional pain of the foot injury allowed the injury to remain unhealed. The gentle treatment allowed the patient to part the veil of dissociation that he had placed around his foot injury. Then, his foot could begin to heal.

Unexpected, even painful, and very often counterintuitive recovery symptoms were not predicted, but patients developed them nonetheless. These *recovery* symptoms, as much as the symptoms of Parkinson's disease, helped us figure out the electrical disarray that causes idiopathic Parkinson's disease.

However, some patients developed intermittent, psychologically triggered symptoms of psychogenic parkinsonism after their foot injuries healed and the symptoms of idiopathic Parkinson's started to go away.

Our researches suggested that these intermittent symptoms had a purely mental or emotional origin, and were being set in motion via dissociation from the body or parts of the body during times of conscious or unconscious stress.

The case study excerpts in the preceding two chapters hopefully showed why we now hypothesize that a foot injury plays a role in the development of Parkinson's disease. Hopefully, they also show why, from the earliest days of our research, we began to suspect that most PDers have, to varying degrees, a mental/emotional component to their illness that may or may not have been set in motion at the time of the foot injury.

person lives longer. He is thus able to develop the long-term consequences of rebellious Qi in the Stomach channel, which are the symptoms of Parkinson's disease.

Based on our findings, not only is Rebellious Qi in the Stomach channel a problem now that we are living longer, but Rebellious Qi in the Stomach channel is a cancer-causing killer in its early stages, decades before it causes the syndrome known as PD. No wonder the Asians used the word "Rebellious" to describe this treacherous type of Qi.



“It is by virtue of the twelve channels that human life exists, that disease arises, that human beings can be treated and illness cured. The twelve channels are where beginners start and masters end. To beginners it seems easy; the masters know how difficult it is.”

- The Chinese medicine classic, Spiritual Pivot, chapter 17¹

CHAPTER FIVE

CHANNEL THEORY

The next few chapters involve channel theory – a crucial part of Asian medical theory and the key to understanding the anatomical changes that occur in a person with Parkinson’s disease. This chapter will explain how the pertinent channels behave in a healthy person – or at least a person who does not have Parkinson’s.

INTRODUCTION TO CHANNEL THEORY

What are channels?

Channels are pathways of energy that circulate through the body. The energy in the channels is referred to as channel *Qi* (pronounced chee).² The term “channels” refers to the *directions* and *locations* in which the electricity-like channel Qi is most likely to flow, as it traverses the various tissues of the body. The channels are *not* structures. They have no tangible existence if no energy is moving. There is *no* physical, conduit-like tube through which the channels flow.

Channels in living organisms are rather like shipping lanes on the open sea. Shipping lanes are specific, preferred routes. They can be drawn on a map. But if you fly over the ocean and there are no ships in sight, no shipping lanes are visible. Their presence cannot be measured. As far as the ocean is concerned, shipping lanes are only a theoretical construct.

Likewise, the channels only exist when moving current is present. A dead person, a person in whom no energy is flowing, has no apparent channels. In a living person, when the largest, closest-to-the-skin, electricity-like channel Qi flows, it *tends* to flow in specific, detectable routes through the body’s tissues, especially the fascia tissues. These routes are called channels.³

¹ The translation is from *A Manual of Acupuncture*, Peter Deadman and Mazin Al-Khafaji, 1998. *The Spiritual Pivot* is thought to be approximately 2000 years old.

² Qi is defined in depth in appendix #xxx. For now, consider Qi to mean “energy.” If you have a background in Vedic studies, Qi means the exact same thing as the Sanskrit *Chit* (pronounced “Chee” with a nearly silent “T” at the end). Chit is one of the three attributes of God. For example, “Sat-Chit-Ananda” (Wisdom-Energy-Bliss), is one of the many definitions, or “names,” for God. While there are few books expanding on the full meaning of “Qi,” a mysterious, undefineable force, there are tomes explaining the physics and straightforward ramifications of “Chit,” all of which mesh perfectly with our most modern understanding of modern physics and quantum theory.

³ When western scientists first learned of channel theory, they were highly dubious. One doctor did a careful dissection of a corpse and announced that there was no evidence of any structures that corresponded to the description of the primary channels. Therefore, channel theory was, for nearly a century, mocked and reviled. Channels still cannot be detected in a corpse. When the body is dead, no electrical currents flow.

Is channel energy the same as electrical energy?

For the most part, the Qi that flows in channel routes behaves like electrical currents: the Qi is subject to resistance and is influenced by parallel currents. It can even “short circuit” into other, nearby channels if its own route is blocked.

The currents that run in the channels are unidirectional: direct currents rather than household-type alternating currents. The correct direction of channel flow is crucial to maintaining health.

Qi in channels is *not* absolutely the same as the electrical currents that flow through our kitchen wall sockets. One recently conjectured theory holds that Qi flow may not be the same as electrical flow because Qi flow is the movements of *waves* generated by electrons (or photons, in some theories) but not the movement of discrete, measurable “*bodies*” of the electron or photon. Other reasons that Qi is not considered the same as electricity (or light) per se are somewhat esoteric, and not necessary for understanding what I call the “electrical” aberrations that develop in Parkinson’s disease.

However, to appease the acupuncturists who may be unhappy even if I say that channel Qi moves *like* electricity, here’s an oversimplified explanation of why channel Qi is not exactly the same as the energy that flows through your car battery.

Thoughts and feelings generate electromagnetic waves (brain waves). These waves, in turn, necessarily influence the body’s electrical currents. These electrical currents, thus influenced, are affected along the length of their route. Thus, thought waves and the wave patterns generated by feelings influence the body-wide electrical currents. Also, ancient Vedic

Since the late 1970s, some western researchers have been preoccupied with proving the western hypothesis that acupuncture channels are dependent on the nervous system. While some studies have shown that the nervous system can be, in fact, affected by stimulation of the channels, and anesthetized nerves are not responsive to acupuncture, no studies have been able to prove that channels need the nerves in order to exist. The Asian theory would say that even single-celled organisms have channel Qi flowing over the surface of the cells. Nerve structures develop in response to the channels; at the smallest, cellular levels of channel electrical flow, the channels influence DNA expression and the formation of cellular structures, including the nerves.

Even though a few experiments are finally being designed to look for Qi flow patterns that are not directly related to nerves, these studies are few and far between. There is simply not much profit to be made in objectively proving the existence of channels. The existence of acupuncture points has been objectively proved: machines can easily detect decreased electrical resistance in the skin at the locations of the known acupuncture points. (In fact, some doctors who play at being acupuncturists use these machines to locate the points instead of memorizing the general vicinity of the point locations and honing in on the exact location by a trained sense of touch.) But the currents of moving energy that *connect* those points cannot yet be definitively measured by machine. The best way to feel the *directions* of the currents that run in the channels remains the seemingly subjective method of feeling the Qi by hand. It is quite easy to learn. It is like learning to differentiate the difference between the feel of velvet when stroked with the nap or against the nap.

While western researchers sneer at this subjectivity, I am reminded of a cartoon strip in which Doonesbury character Alex tries to assess the electrical engineering professors at the colleges to which she is applying. She asks them this question: “Two black boxes, each hiding an internal circuit. Using workbench tools, how do you tell which is the current source and which is voltage?” Several teachers are stumped. Finally, one professor gives her the correct answer: “Well Alex, they’re Thévenin and Norton equivalences, so tools are useless. You’d have to hold the boxes in your hand. Since the current source has a resistor, it’d be warmer.” (From the cartoon anthology collection, *Heckuva Job, Bushie*, G.B. Trudeau, Andrews McMeel Publishing, Kansas City, 2006, p. 223.)

At this stage of scientific research, electricity-measuring tools are still useless to single out the *direction* of Qi flow amidst the various bits of electrical forces that can be detected on the skin. At the present time, if you want to detect the dynamic flow of Qi, tools are useless; you have to feel it with your hand.

theory explains that there are five types of electron vibrational patterns. Chinese medical theory recognizes that these five *types* of electricities (usually referred to as Five Elements) direct different aspects of cellular and organ functions.

In Asian medical theory, one could say that the energy that moves through the channels is a life force- and brain wave-driven transition from electromagnetic fields into moving electricity-like current. These currents have specific electron-vibration patterns.

Your car battery or an electric generator is only concerned with getting a charge to move from point A to point B. The type of subtle electron vibratory movement is of no consequence in a car battery.

In bodies, the *type* of electron vibration pattern in each channel helps make up the characteristics of that channel. The type of electron vibration pattern determines how the currents will influence on the cells over which they flow.¹

A spot of theory

When it comes to studying Asian medical theory, the western medical paradigm tries to understand Asian theory using archaic western medical constructs. The western medical model turns a blind eye to the new findings in modern physics, and quantum theory in particular. The 19th and 20th century paradigms of western medicine hold that the *structures* of the body – nerves, organs, blood vessels – must be creating and determining the paths and mode of effectiveness of any electrical currents that may or may not exist. Further, they insist that, if any electromagnetic fields exist alongside of the currents, they are the *result* of the currents – currents that are generated by chemistry in the cellular structures. There is no “which came first, the chicken and egg” mystery in western clinical science. The structure is assumed to be the dumb template on which chemical and electrical events take place.

In Asian medicine, we recognize the *mutual* effects of the electromagnetic fields (such as those generated by thoughts and the various electrical events in the body), the currents (directed by the fields), and the cells (created based on the instructions provided by *both* the currents (channels) and the materials provided by the structures (cellular chemistry, including DNA). Working together, the somewhat *stable* electrical properties of physical structures of the body and the electromagnetic fields with their fast-as-a-thought-wave ability to *change or influence* moving electrical currents, provide both stability and dynamism to the underlying electrical nature of the chemistry of living systems.

The vibratory (electromagnetic wave) aspect of a person, not the chemistry, is the ultimate driving force behind the ever-changing structures and molecules that make up a person’s physical being. Again, the vibratory forces – the electromagnetic fields, thought waves and electromagnetic heart waves – direct the currents of Qi in the channels. The currents then direct the cellular performances.

Oppositely, cellular injury, illness, or toxins, can cause changes in the cells, which then cause changes in the electrical currents. These changes potentially can, in turn, cause changes in organ function and in organ electrical wave patterns, and even in one’s thought waves. The extent to which illness or injury can change one’s thought waves depends on the opposing vigor

¹ The five different natures of electricity flowing in the various channels helps keep the channels running true even when they intersect with each other. Also, in case you’re curious, the five senses – vision, hearing, touch, smell and taste each use only one of the five types of electricity. The nerves and neurons that process a particular sense only respond to the type of electron movements that apply to that sense’s electrical type.

with which one mentally resists yielding to the “vibrations” of illness or pathology. The ability to oppose changes in thought waves in the face of insult and injury depends on the strength of one’s consciousness, one’s sense of self.

Ironically, in order to maintain his composure and self-control, many a PDer has opted for dissociation, a psychological stance that denies him full awareness of his “self.” Thus, he renders himself *less* able to consciously feel the pain and the eventual pathological changes set in motion by his ignored injuries. He cannot consciously resist the damaging changes caused by his injury because he cannot feel the injury. He can summon up his will power in the struggle to keep functioning *in spite* of worsening illness and injury, but so long as he is dissociated from his ability to feel his injury, he cannot summon up the consciousness that will trigger a *healing* process.

In summary, the flow of energy in the body, while obeying the laws of electricity, is considered to be far more than just a mindless flow of electrons. This electricity-like energy is referred to as channel Qi. When acupuncturists refer to channel Qi, they most often are referring to that portion of body-regulating electricity that flows in large, detectable streams near the surface of the skin. Healthy current flow is unidirectional, not alternating. The electrons for any given physiological task are vibrationally specific.

Channel Qi behaves like electricity, but it is much more than mere voltage and amperage. Therefore, in the field of Asian medicine, we do not say that channel Qi is electricity. We tend to say that “channel Qi is the *energy* that flows in the channels.”

Next, to futher appease any sticklers with a background in Asian medicine who may insist that Qi is too mysterious to explain or that Qi is *not* the same as electricity but they can’t say what it is, I repeat: when I refer throughout this book to the currents being “electrical,” I mean electrical in terms of being made up of electron movement *but also* derived from and driven by electromagnetic thought waves and forces related to brain waves, consciousness, subconsciousness, and superconsciousness. Channel Qi is a transitional form of energy in which thought vibrations, having created electromagnetic fields, are being condensed into electrically charged flows of tangible energy – moving energy that can be felt. These currents influence and are influenced by the chemistry and structure of the body.

What size are the channels?

There are currents of varying size and strength. The largest currents integrate the bigger parts of the body: the head and limbs and the internal organs. The very smallest branches of the currents are made up of the ever-fluxing electrical charges (on molecules) that surround each cell, thus determining DNA expression and other cellular functions.¹

¹ “DNA expression” is a term that refers to activity in small sections of the huge DNA molecules. Small sections of the molecule are called on now and then to manifest a few of the thousands of bits of genetic instructions that reside on the DNA molecule. Most of the DNA molecule is inactive most of the time. Electrical charges that bathe the outside of the cell contribute to the regulation of chemical activities in the cells, including activities in the cells’ DNA. These electrical charges are manifestations of the very smallest branchings of the channels.

To appreciate just how small the channels can be, consider the one-cell organisms; in single celled organisms, their surface-of-the-cell electrical patterns *are* their “channel Qi:” their shape- and function-determining electromagnetic structure.

The largest currents that are accessed in Asian medicine run just under the skin. These larger currents have enough force that they can be felt by hand, by simply holding one's hand directly over the course of the current. It takes a bit of training to feel them, but not much. Nearly everyone can learn to feel the current in the larger channels.¹

The larger currents branch into smaller currents, and then into still smaller currents, and even smaller currents. The larger currents traverse and integrate the whole body. The smallest currents are mere electrons shifting back and forth. These tiniest electrical currents and their associated electrical fields can direct cellular chemistry. These smallest electrical "switches" and their matching electrical/magnetic fields give instruction to cellular DNA. These switches signal the cell's DNA to express genomes (execute particular genetic instructions) at the appropriate times.

Overall, the current-generated electrical signals trigger necessary biological events in the cells, organs, and the body. Together, the physical and chemical structures of the body work side by side with the electrical currents that run over and through the entire body. The former, the structures, provide some structural stability. The latter, the currents, provide the ability to grow, change and respond to an ever-changing internal and external environment.

¹ When I teach students to feel these currents, they can often feel them within a few minutes of receiving instruction. It takes much longer for them to start to trust what they are feeling. I have found that the easiest way to help them learn to trust their perceptions is to have a group of students all feel the same patient's channel flow and write down what they perceive without telling the other students. Later, when they compare notes, they are amazed that they have all felt the same things, including the same aberrations in the exact same locations. This confirmation lends a sense of objectivity to their as yet "unprovable" measurements. In my years of experience, I have only had a few students who could not learn to feel the flow of Qi.

Possibly because of the very small amperage of the currents and waves that constitute the channel system, these electrical forces that course over the body have been, until recently, utterly disregarded in western medicine. This is starting to change, as the significance of micro-currents is exploding in our faces.

We now have tiny computer chips that can almost fit on the head of a pin – and these chips are directed by micro-electrical systems. In the world of computers, we see very small electrical forces directing very large and complex systems. Even ten years ago, the general citizenry might have expressed disbelief that micro-, even nano-electromagnetic signals might be elegantly regulating the tiny electrical bonds that hold together the chemistries of the body. Today, in the extremely small world of computer chips, we see how a one-electron switch can direct an entire cascade of information to move in a particular direction. This makes it easier to understand how, in a biological system, the electric fields generated by the currents running throughout the body, and the currents generated by fields and wave patterns, including heart and brain waves, might send significant signals to tissues and chemistries in the body.

Until very recently, even most doctors assumed that crude chemical interactions, rather than subtle electrical ones, determined all cellular functions. Even into the late 1990s, many older doctors assumed that the extremely large electrical impulses that run along the nerves were the only electrical processes in the body!

Just a few years ago, in 2001, a middle-aged neurologist sharing a table with me at a wedding got up angrily from her seat and announced that she refused to sit at any table with me or ever speak to me again. This astonishing explosion occurred when, in response to her asking me how acupuncture worked, I started to explain that acupuncture manipulated electrical currents in the body, currents much smaller than those used by nerves. She bristled. Possibly she had expected some esoteric statement about Qi that she could laugh off. She loudly refuted my statement. I reasserted it. She stood up from her assigned seat, announced loudly, "Nerves are the only part of the human body that use electricity! I refuse to listen to this." and stomped off.

Maybe she had forgotten that there is almost no chemical process that is *not* electrically driven. At the time this hostile interaction took place, the research on bio-electrics had been making strides for over a decade.

Where are the main channels?

The easily accessible and detectable portions of the main channels travel in the subcutaneous fascia, a membrane just under the skin. These currents influence the underlying muscles, tendons, and bones and the overlying skin in their vicinity. Branches of these channels travel inside the body, connecting with organs and with branchings from other channels. The body-wide looping system of electrical currents is heavily interconnected.

The twelve primary channels are bilaterally symmetrical: each channel on the left side of the body has a matching one on the right side. Therefore, one might say that there are actually twenty-four primary channels, if counting the lefts and the rights separately.

All of the twelve primary channels flow sequentially: channel one flows into channel two, channel two into channel three, etc, with the twelfth and final channel flowing back into the first one and starting over. All the channels have many points of intersection with other channels and the extraordinary channels, in addition to the over-all sequential flow pattern.

The electricity in our homes flows in very limited pathways: wires. The flow of Qi in the channels is not limited in this way. Channel Qi flows everywhere. At points of multiple channel intersections, Qi is influenced by several factors. It will flow into whatever pathway offers the least resistance while simultaneously being under the influence of the thought waves and the physical and chemical structures of the body.

Nomenclature of the channel system

Primary and extraordinary channels

The channel system's largest channels consist of twelve primary channels and eight "extraordinary" channels. The primary channels are so called because they actually make up one circuitous loop. The extraordinary channels are distinct currents of energy, but they do not form a continuous loop. Also, six of the extraordinary channels do not have named acupuncture points on them. In these six extraordinary channels, their major points of decreased electrical resistance (acupoints) are at intersections with the primary channels, intersection points that are already named via the primary channels. Acupoints, or "named acupuncture points" are areas of decreased electrical resistance. They are located along the primary channels and along two of the extraordinary channels.¹

The two extraordinary channels that have named acupoints are the *Ren* (translated as "Conception") and the *Du* (translated as "Governor"). These two appear to be in the "extraordinary" class because, like the other six extraordinary channels, they are not a part of the sequential flowing loop of the primary channels. Also, the *Ren* and *Du* are exceptional in that they are singular: they are *not* bilaterally symmetrical.

The *Ren* channel runs up the midline of the front of the body, from the genitals to the bottom lip. The *Du* runs up the midline of the back, from the genitals to the top of the head, over the top, and down the front of the face to the upper lip. The *Du* channel regulates energy in the spine and brain, and in the midbrain – and regulates the release of dopamine. The energy in the *Du* channel keeps the brain and spinal currents alive. If the neck is broken and the *Du* channel severed, death or a serious brain-body disconnect will ensue. Protection of the *Du* channel plays a large role in the electrical changes that occur during a severe injury. In the upcoming

¹ As a point of interest, western-medically recognized "trigger points," areas on the body that become sensitive when something is going wrong, are areas of *increased* electrical resistance.

explanation of what goes awry in Parkinson's disease, the Du channel, in addition to the Stomach channel, will be referred to many times.

As an aside, when I refer to "large" channels or "main" channels, I am referring to *both* the extraordinary and primary channels as the "main" or "large" channels.

Channel names

The name of each *primary* channel reflects an internal organ that is located along the course of the channel or along a branch of that channel. For example, one of the smaller branches of the Stomach channel diverges from the main, skin-level portion of the Stomach channel, as the Stomach channel traverses the area just above the stomach. This branch flows deep inside the body at that point and directs the development and function of the stomach. Therefore, this channel was named "Stomach channel."

Acupoint names

The acupoints, the locations on the body that are used most often in acupuncture, are named with the channel name and a number. The numbers are assigned in sequence, starting at the origin of the channel. For example, in the case of the Stomach channel, which flows from the face to the feet, the first named point, "Stomach 1," is near the eye, at the beginning of the channel, and the last point, "Stomach 45," is on a toe at the end of the channel.

In writing, the channel names are usually abbreviated. The point named "Stomach 1" is usually *written* ST-1. In speech, the point names are *not* abbreviated into initials. For example, ST-1 is *not* called "S" "T" "one." When spoken, the points are called by the full name of channel plus number: for example, "Stomach one."

In Chinese, the points are not numbered; they each have poetic names that relate more to the function of the acupoint than to the channel.

THE PHYSICAL BODY AND THE ENERGY CONTAINED THEREIN

"The Qi is the leader of the Blood"

The body's physical structures, whose creation was directed by channel Qi, help influence the channel Qi to stay, somewhat, in the physical path that was created by the Qi.

The Qi directs the growth of the body. Growth of the body produces more Qi.

Qi directs the creation of the body, and the body provides a path for the movement of Qi. In the same manner, the great rivers of the world stay in their river beds – or stray from them in times of changed circumstances. In the river analogy, the initial water flow digs a river bed. The river bed then guides the flow of subsequent water – until a flood or geological upheaval occurs. In the same way, channel Qi will tend to flow in a healthy pattern until a severe injury or scar tissue disrupts the flow of current. When disruptive events occur, the Qi, like electricity or water, will follow the path of least resistance, and may sometimes create a new pattern. In living systems, these "new" patterns are rarely as conducive to health as the undisrupted patterns.

In Asian theory, this famous principle of energy and chemistry's mutual relationship in living systems is expressed as "The Qi (vibrational energy that is transitioning from pure thought and pure vibration into matter, including the electromagnetic energy that is condensing into electrons) is the leader of the Blood (chemistry) and the Blood is the Mother of the Qi."

The first part, “The Qi is the leader of the Blood” might be translated: “Vibrational forces, including electromagnetic and electrical forces, *direct* and sustain chemistry and biological structures and their changes. The second part, “The Blood is the Mother of the Qi” might be understood to mean: “As the structures in living systems multiply or grow larger, they provide a ever larger substrate over which an *increased* amount of electricity can flow.”

The Chinese way of saying it is snappier: the Qi is the leader of the Blood and the Blood is the mother of the Qi. The expanded version, however, might make more sense to a person trained primarily in western sciences.

Hopefully, this is enough general information to be getting on with. The next chapter provides very specific information about the four channels that go haywire in Parkinson’s disease: the Stomach channel, and the three other channels that are affected when the Stomach channel flows backwards. I’ve included the details of their pathways. You do not need to memorize the paths of the channels. However, if you take a few moments to study these Parkinson’s-related pathways, you might sudden see that all the symptoms of Parkinson’s disease fit along these channels. Considering that a person has thirty-two channels, the realization that early stage Parkinson’s symptoms only appeared on one channel, with repercussions into a mere three of the other channels, helped create our initial hypothesis of the cause of idiopathic Parkinson’s disease.



"The river glideth at his own sweet will..."

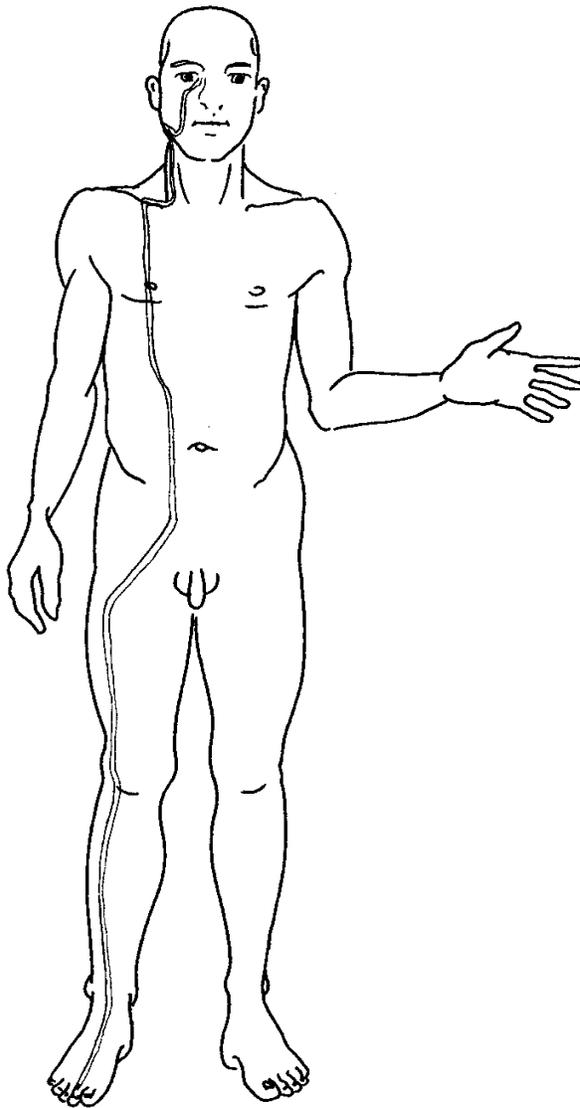
William Wordsworth's "Lines Composed Upon Westminster Bridge"

CHAPTER SIX

THE CHANNELS INVOLVED IN PARKINSON'S

The diagrams in this chapter show the *correct* paths of the channels that are involved in the pathologies of Parkinson's disease.

THE STOMACH CHANNEL



The acupoints of the Stomach channel begin at the inner canthus (meeting point of the upper and lower eyelids) of the eye. The Stomach channel then runs down the center of the cheek, spreading out widely over the cheekbone and narrowing again as it passes the corner of the mouth, loops around the upper and lower lips, and then resumes its path down the face, from the corners of the mouth down to the jaw. (Note: the branch that loops around the mouth is not shown on this diagram.)

The current then travels along the lower jaw to the back corner of the mandible. From the area of the lower back molars and the back of the jaw, the Stomach channel flows down the side of the neck and over the collarbone towards the nipple. The channel follows the mammary line (where a row of nipples would be located if we were dogs, and not humans) until it comes almost to the bottom of the ribcage.

Around the level of the fifth rib, the channel travels medially (towards the center line, or midline, of the body), drifting a bit closer to the midline but still continuing its downward (towards the feet) flow. When the Stomach channel gets to the pubic bone, it flows laterally (towards the sides of the body, away from the midline) over the inguinal groove towards the front-side of the hip.

Fig. 6.1. The correct flow of the Stomach channel.

From the hip, the Stomach channel flows down the anteriolateral (front-outer) side of the leg, over the center-front of the ankle, directly over the highest point of the dorsum of the foot (over the arch of the foot) and then the channel branches. One branch goes to the second and third toes. Another branch flows over to the medial side of the big toe after which it reverses direction, heads back up the leg, and is known as the Spleen channel. There are numerous other branchings and bifurcations along the Stomach channel, but the main path and its branching on the foot are the most important for understanding Parkinson's disease.

There are two symmetrical Stomach channels, one on the left and one on the right. Only the right side Stomach channel is drawn here.

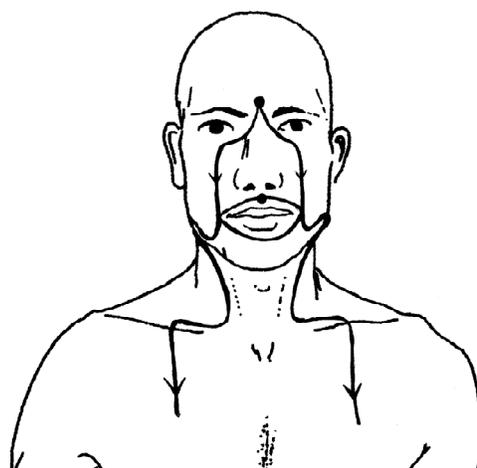
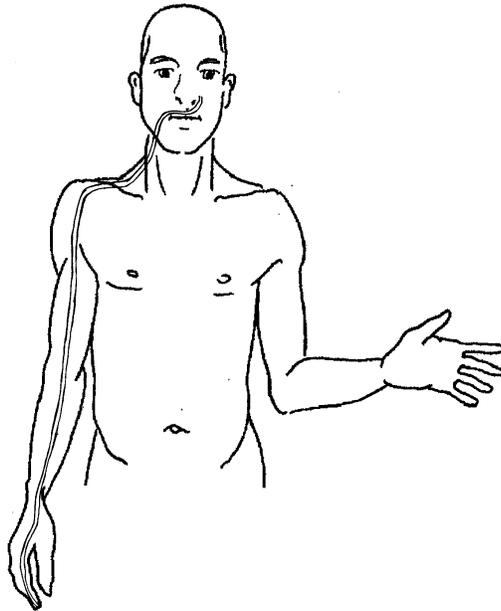


Fig. 6.2 Detail of the face portion of the Stomach channel

Notice that the Stomach channel connects with a branch channel that skirts the upper and lower lips. The Stomach channel current starts at the forehead, but the first named acupoint on the Stomach channel is located at the inner canthus of the eye.

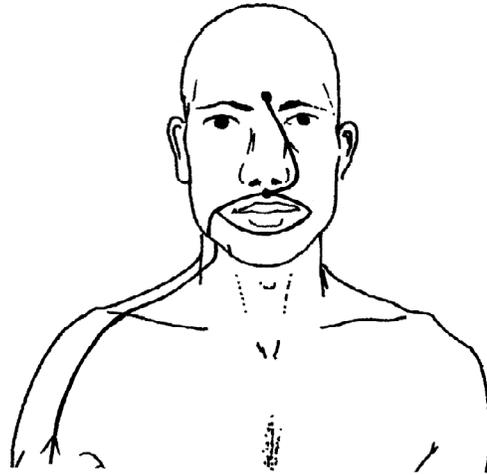
Fig. 6.3 The correct flow of the Large Intestine channel



The Large Intestine channel flows from the tip of the index finger up to the side of the nose on the other side of the face. From there, it connects with Yin Tang, the point between the eyebrows (not shown).

From Yin Tang, the current flows back down the face in the pattern known as the Stomach channel. Notice that the right arm channel crosses over to the left side of the face. The right Large Intestine channel flows into the left Stomach channel, and vice versa.

Fig. 6.4 The face portion of the Large Intestine channel



Notice that the Large Intestine channel crosses over to the opposite side of the face via the small channel that skirts the lips. The right-side Large Intestine channel flows up the right arm and crosses over to the left side of the nose. Oppositely, the left-side Large Intestine channel (not drawn) flows up the left arm and crosses to the right side of the nose.

Fig. 6.5 The upper portions of the Gallbladder channel, the Stomach channel, and the Large Intestine channel showing the correct flow directions

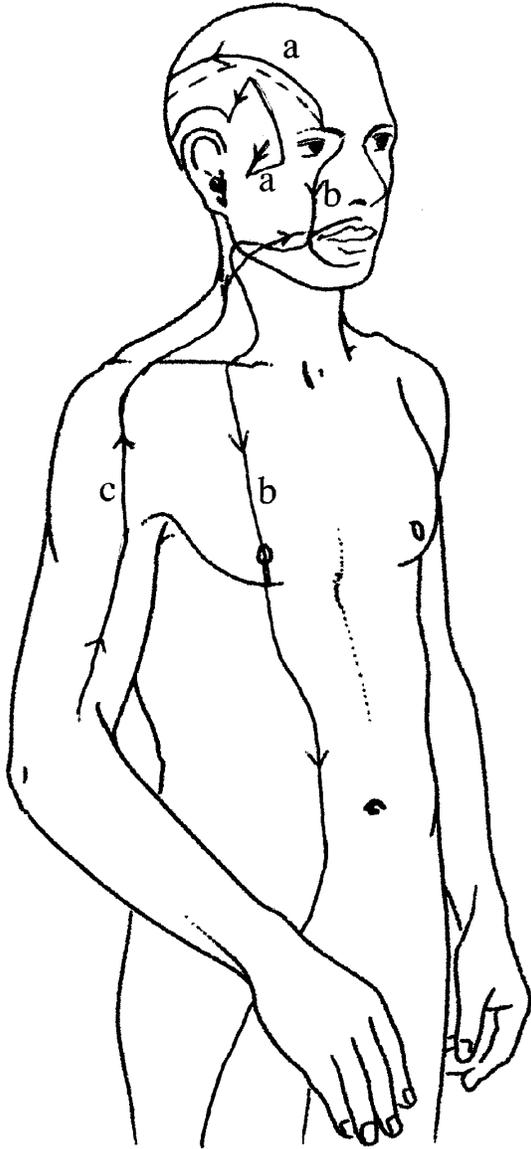


Fig. 3.5 shows with arrowheads the correct flow direction of the head segments of the a) Gallbladder, b) Stomach, and c) Large Intestine channels. (For clarity, not all segments of the channels are shown.)

a. The Gallbladder channel begins at the outer corner of the eye and traverses the side of the head.

b. The Stomach channel begins at the inner corner of the eye and travels down the face, along the jaw, down the neck, and down the torso.

c. The Large Intestine channel travels up the arm, up the neck, crosses through the Stomach channel at the jaw and then crosses over to the other side of the face via the upper lip. The Large Intestine channel then travels up the side of the nose to the inner side of the eye, where it then becomes the Stomach channel. The right-side Large Intestine channel flows into and becomes the left-side Stomach channel, and vice versa.

The exact location of the Large Intestine channel crossover from the neck to the jaw is farther back on the jaw. To show the exact location, the drawing would become too

cluttered at the jaw. This drawing is approximate.

Fig. 6.6 Correct flow patterns of three of the channels on the side of the head

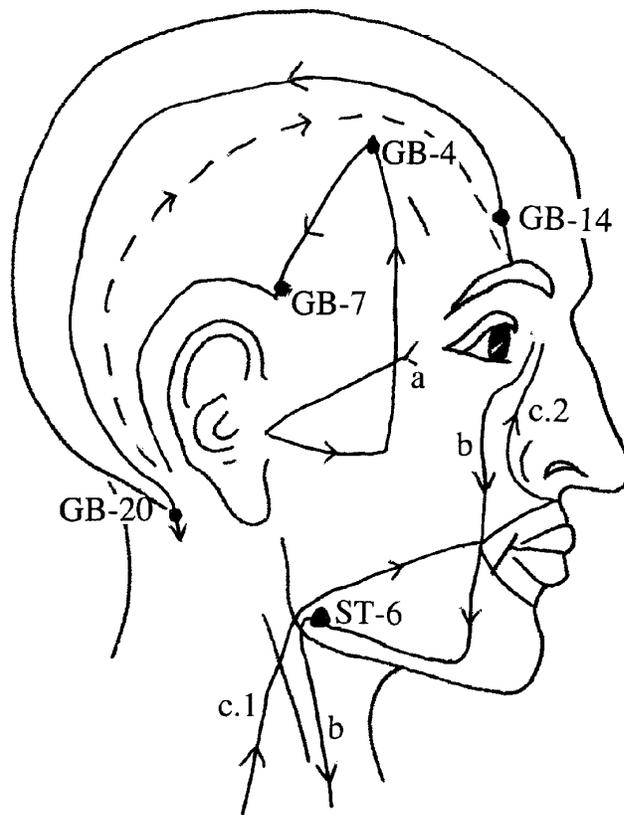


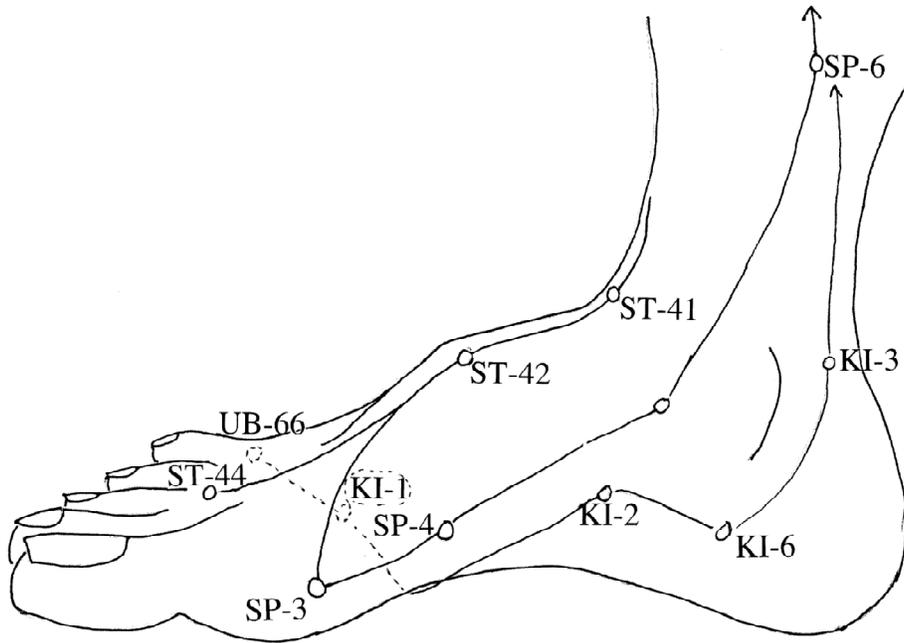
Fig. 3.6 is a close-up of the *correct* flow of the a) Gallbladder, b) Stomach, and c) Large Intestine channels. Note that the Large Intestine channel must cross over the Stomach channel at the neck or jaw in order to cross over to the other side of the face. In the above diagram, line c.1 designates the *right side* Large Intestine channel. Coming up the right side of the nose after having crossed over from the *left side* is line c.2. The dashed line indicates a deeper, subcutaneous segment of the Gallbladder channel that connects the more superficial (running through the skin) segments, segments which are shown with a solid line. This construction of the GB channel creates a sort of coil on the sides of the head, amplifying the electric field properties of this channel.

Key points shown on the Gallbladder channel are GB-4, GB-7, GB-14 and GB-20. At the corner of the mandible is shown the location of the Stomach channel point ST-6.

The Gallbladder channel sweeps back and forth across the side of the head. In ordinary circumstances, the Stomach channel and Gallbladder channel do not flow into each other. Note: to prevent things getting too messy, this diagram does not show the branch of the Large Intestine channel that connects to the Qi that skirts the lower lip.¹

¹ The drawings in this book are not intended to provide in-depth instruction on channel and acupoint location. The bibliography suggests a text that is well-suited for in-depth study in locating channels and acupoints.

Fig. 6.7 Correct Qi flow in the Foot in the Stomach, Spleen, and Kidney channels, and some key acupoints of the foot



Stomach channel Qi flows down from the face to a point at the high spot of the foot. At this point, named Stomach 42 (ST-42), the channel bifurcates into two lines of current. Qi flows both towards the toes (ST-44) and also over to the Spleen channel at acupoint Spleen 3 (SP-3). The dotted line indicates Qi flowing on the underside of the foot. Qi flows from the Bladder channel (at UB-66, on the smallest toe), underneath the foot to the first point on the Kidney channel, Kidney 1 (KI-1), on the sole of the foot, and then continues up the Kidney channel.

The most important point to note on this diagram is ST-42. This is the location of energetic disarray and unhealed foot injuries or some residue of unhealed foot injury in all the Parkinson's patients that we have seen.

The most important thing to get out of this chapter is the idea that electrical flow in the healthy body occurs in a fairly specific pattern and in a highly specific direction. Also, as you may have noticed, the channels are not distinct, separate bits of energy. The channels are actually parts of a continuously circulating electrical current. For example, you have seen above how the Qi that flows up to the face, across the upper lip and up to the forehead is named the Large Intestine channel. But when this current changes direction and flows down the face towards the legs, the downward flowing portion of the Qi flow is named the Stomach channel. The names change; the Qi never stops.

The Qi flow is a continuous stream of energy. One channel flows into the next, and from there into the next, and the “last” of the primary channels flows back into the first.



“No go through: pain. Go through: no pain.”

- A basic principle of Chinese medicine

CHAPTER SEVEN

BLOCKED QI: TROUBLE

Basic channel flow theory

When channels and all their bifurcations and subsets are running perfectly, all the body systems can grow, maintain, die, or respond correctly to external changes, in an optimal manner. That's health.

But sometimes, the flow of Qi goes wrong. The result is illness or pain.¹

This chapter explains just a few of the problems that can interfere with the correct flow of channels, according to the “rules” of Asian medical theory. It includes the snafus pertinent to the Stomach channel blockage seen in all people with Parkinson's.

Channel obstructions

When the electrical flow of channels is blocked or stopped due to a break in the tissue or the presence of some non-conductive tissue, the flow diverts around the blockage. Scar tissue, with its rubber-like mass of short, crisscrossing fibers, is an example of non-conductive tissue. When the path of a channel is blocked by a bit of scar tissue, the Qi is diverted around the scar. The channel Qi resumes its usual pathway a short distance downstream from the scar tissue. The Qi diversion can be felt by a trained hand.

The absence of channel Qi in the scar tissue means that the cells of the scar tissue do not receive electrical instructions on what kind of cells to be. In the absence of instructions, cells become non-functional, adhesion-type cells. In most cases, scar tissue, left to its own devices, does not grow back into healthy skin. Also, scar tissue and its immediate surroundings often feel numb to the touch: nerve conduction, an electrical phenomenon, is also blocked by non-conductive tissue. The numbness usually corresponds to the Qi-free zones that are created as Qi skirts the blocked area and resumes flow somewhere downstream.²

¹ The Chinese maxim for stating this is “No go through: pain. Go through: no pain.” Although my professors stated this maxim to me nearly every time I asked a question about theory, I had no idea what they were talking about. Not until nearly a decade after graduating from Asian medical school did I realize what this statement actually meant: if Qi is flowing correctly, the body will manifest health. If Qi flow is inhibited, obstructed, or flowing in any direction that is not consistent with the vibratory pattern (the thought wave and electromagnetic wave determined by that thought) that provided the original pattern for the creation of that body, the result will be “pain”: sickness, poor health, pain, moodiness, wrong thinking, shortened lifespan.

I think the reason that it took me so long to understand this maxim is that any discussion of channel theory was not acceptable during the years when I was in school. Because, as a westerner, I only heard the words of the maxim and had nothing to connect it to, it was meaningless. Nevertheless, I heard it a *lot*. It wasn't until I understood channel theory that I began to suspect the significance of this maxim.

² Acupuncture needles can be used to reintroduce electrical flow through scar tissue. When correctly needled a few times, scar tissue slowly reverts back to healthy tissue and the numbness usually goes away.

Sometimes, if the scar traverses a wide area, the Qi gets shunted into the path of a nearby channel. In this case, if the nearby channel is flowing the same direction as the blocked channel, the displaced Qi may or may not resume flow in its own, correct channel after it gets past the blockage. If the Qi does not, at some point, flow back into its own channel, significant weakness, numbness, or pathologies may develop along the portion of the channel that is “downstream” from the blockage, which has minimal or no Qi flowing in it.

Even if Qi is diverted into the path of another channel, the flow pattern often self-corrects somewhere downstream of the blockage. Remember, Qi is not brainless electricity; Qi flow patterns are stabilized by the body’s chemistry and structure and by the mind’s brain waves. These forces can help Qi to resume its correct pattern after getting past the trouble spots. Also, throughout the system, smaller “connecting channels” link the main channels. These connectors can bypass or help correct small Qi glitches. The diverted Qi can resume running correctly somewhere further downstream of the blockage, or it may remain in a neighboring channel into which it was diverted. In the latter case, the amount of Qi in the original channel is reduced, and the amount of Qi in the neighboring channel is increased – and possibly confused: each channel’s electrical vibratory patterns are specific for one of the five electron movement variations.

If a channel is blocked and the nearby channel into which it flows is running the *opposite* direction of the diverted channel, the diverted current changes its direction, and flows along with the nearby channel’s current. (Actually, the neighbor current will be somewhat modified by this addition, but for now I’m introducing general principles, not the fine details.) In this case, the *diverted* channel is not able to resume its correct path farther downstream, but becomes caught up in the flow of Qi running in the opposite direction.

In some cases, some portion of the diverted Qi may run deeper into the body via a “connecting” channel rather than running laterally into a nearby primary channel. This submerged Qi is often able to resurface near the skin farther downstream into the original channel. In this case, the Qi may continue to flow correctly for the rest of the length of the channel, but the amount of Qi flow may be diminished in the area immediately downstream from the blockage.

Utter blockage in a channel

When channel blockers such as scar tissue, muscle tension, bone displacement, excess fat, mucus or other diversions, including the electromagnetic disruptions of mental blockages (unhealthy brainwave patterns), occur at the very end of a channel, the channel is not able to divert around the problem area and regroup farther downstream. A channel blockage at a channel terminus is therefore particularly problematic. The Qi flow pattern may become distorted in the vicinity of the blockage. Over time, the distortions themselves can contribute to a form of electromagnetic blockage. At some point, as the Qi flow at the terminus becomes increasingly stymied, the electrical resistance in the channel begins to build. Qi, like electricity and water, will flow in the path of least resistance. If the resistance at a channel terminus becomes too large, the Qi in the channel will begin flowing in whatever direction offers the least resistance – even flowing backwards.

Although the standard terminology is “backwards,” the actual movement of “backwards” Qi can be a back and forth, rapidly alternating movement. However, in a backwards-flow situation, if Qi distribution higher up the channel becomes diverted into another channel,

movement can become predominantly backwards (instead of back and forth). However, some amount of back and forth electrical movement might still occur in the area closest to the actual blockage.

When electrical currents run backwards, they are said to be running rebelliously. The idea that currents can run backwards is presented in the oldest book of Asian Medicine, the hoary Nei Jing.

Rebellious Qi was translated as “Retrograde” (backwards) Qi in the first English translations. Now, “Retrograde Qi” is the common translation in England. In the U.S., “Rebellious Qi” is the more common (and more metaphorically correct) translation.

What's in a word?

The word “rebellious” is significant. Rebellion, in China, is considered the most dangerous of political situations. In rebellious times, the political system is overthrown. Chaos and death ensue. No good can come of rebellion.

I had learned about Rebellious Qi in my Asian medicine classes. I had been taught mild examples of systems running backwards: a cough is a spasm of backwards-moving muscles in the lungs. Burping is backwards-movement from the stomach.

Never was I taught that an entire channel might run backwards. And yet, the very name “Rebellious Qi” suggests that Qi moving backwards might be more dangerous than a mere cough or a burp.

When I noticed backwards-running Qi in my PD patients and then discovered all the pathologies that derived from it, I began to understand the power and danger of Rebellious Qi. A channel running backwards can destroy the very underpinnings of the parent organism. Rebellion indeed!

A few acupuncturists have questioned my understanding of Rebellious Qi. They too were taught in school that Rebellious Qi refers only to vomiting or other short-term manifestations of systems running in reverse. I like to recall the following: several times during the last two centuries, Asian medicine was forbidden in China. These restrictions were political. In every instance, the government was trying to rid itself of the embarrassment of a medical system that, seen by western eyes, was superstitious and archaic.

After the revolution, the Chinese government re-embraced Asian medicine with pride primarily for economic reasons: it worked and it was cheap.

However, conflicts arose. The essence of Asian medicine is philosophical, even spiritual. Religion was anathema to the revolutionaries. To make Asian medicine politically safe, the more spiritual teachings of the ancient medical texts were stripped from the books. A sanitized version was produced.

One of the principles of Asian medicine that was relegated to the retired list was Channel theory. Channel theory, for reasons too long to go into here, is closely tied to the spiritual underpinnings of Asian medicine. Also, western researchers had long mocked the idea of unseeable forces flowing in channels. The Chinese scientists, sensitive to western criticism and determined to weed out discussions of Spirit and philosophy, denied the existence of channels. The medical books were altered accordingly.

Because, for political reasons, channels no longer exist, the theory of Rebellious Qi could no longer apply to Rebellious Channel Qi. Instead, “Rebellious Qi” now refers to hiccups and

sneezing: situations that might better merit the title Pesky Blurps of Backwards Qi, but certainly not the terrifying title of Rebellious Qi.

It was not until I stumbled across backwards flowing Qi in an entire electrical channel and saw the deadly repercussions of this phenomenon that I came to appreciate the wisdom of the ancients. When they named the phenomenon of backward-running channel Qi “Rebellious Qi,” they knew what they were talking about.¹

This has not been a complete discussion of what can go wrong with channels, by any means. But it’s enough to be getting along with.



¹ To suggest another reason why channel theory has been discarded by the Chinese government, and to show that Asian medicine was, originally, inseparable from a profound understanding of spiritual philosophy, I will return to the earlier discussion of the famous “medical” principle, the Qi is the leader of the Blood and the Blood is the Mother of the Qi: the *power* of Love (the source of Qi in its largest sense) is the leader, the director, the force behind the physical existence of the universe (Blood); the physical universe (Blood) is a growing, ever-changing, formed *substrate* that allows an increase in the *manifestations* of Love (Qi). In other words, Infinite Love, by creating – via thought waves that condense into light waves or electromagnetic waves, and then into photons or electrical phenomena – *physical* manifestations of its thoughts, its consciousness, can increase the amount of *Manifested* Love. Infinite Love can create manifested things. These things, especially living things, can, by loving, increase the amount of Manifested Love in the universe. Love directs the universe to create feeling, aware manifestations of itself; the created things of the universe create more love. Qi is the leader of the Blood; Blood is the Mother of the Qi.

As with most great poetry, the word Qi, in this medical principle, has two meanings. The first use of the word Qi refers to the power *inherent* in Infinite Love. The second time the word Qi is used, it refers to the energy, the wave patterns, generated by *manifestations* of Love. This interpretation is consistent with Asian medical appreciation that Qi (as the word is first used) can exist even where Blood does not exist. In terms of energetic creation, Qi must always precede the existence of Blood. Blood may provide a substrate for increased *manifestations* of Qi, but without Qi, there can be no Blood. In western spiritual philosophy, this might be translated as Love exists first: even the “mother” aspect of creation is an outgrowth of infinite, eternal, genderless and formless Love.

Bringing it back to channels, the channels are manifestations of (electromagnetic) *vibrations*. As such, they are closer to the purer manifestations of energy and the thought *waves* of individual or Divine consciousness than are the chemistry and organs, which are “cruder,” more temporal, more subject to change and the delusion of duality. The energy in the channels, on the other hand, is a representation of the more lasting vibrations, even pre- and post life vibrations, of consciousness.

While this understanding might be scorned by the current political leaders of China, this ancient understanding in which medicine is understood as an extension of physics is increasingly compatible with the findings of quantum theory, string theory, and modern philosophy.

This footnote may appear to have little to do with Parkinson’s. I have included it to explain why the channel theory that is so crucial to understanding Asian medicine in general and certain diseases such as Parkinson’s in particular has been officially rejected by the anti-religion leaders of the 20th century Chinese government. Today, most acupuncturists who received their training in China have little or no background in or respect for channel theory.

“Oh the toe bone connected to the (huh) foot bone, and the foot bone connected to the (huh) ankle bone, and the ankle bone connected to the (huh) leg bone... Oh! Didn't it rain!”

— old American song

CHAPTER EIGHT

THE DEVELOPMENT OF PARKINSON'S: CHANGES IN CHANNEL FLOW, PART I

Changes in the foot portion of the Stomach channel

In Parkinson's disease, the unhealed foot injury eventually sets in motion a disruption in the flow of channel Qi in the vicinity of the injury. This disruption builds on itself. When the snowballing electrical disarray in the foot becomes too large, it restricts normal flow of electricity through the crucial Stomach channel terminus at the center of the foot (ST-42). The electrical foot currents start experiencing high resistance at ST-42. Less current is able to get past the growing electrical obstruction. This leads to a decrease in Stomach channel Qi in the areas of the foot distal to the ST-42.¹ With the decrease in Stomach channel Qi in these distal areas, circulation in the middle toes and the medial side of the big toe decreases. These areas usually become somewhat numb over the passing decades.

STOMACH CHANNEL REVERSAL

As resistance at the terminus of the Stomach channel increases, over years, the Stomach channel eventually begins to flow backwards: when the Stomach channel current running down the leg hits the ever-enlarging electrical snafu at ST-42 on the foot, it backs up, creating a back and forth movement of current. Some of this “rebelliously” running current will get shunted into nearby branches or even into other nearby channels that are close to the foot. Over time, an increasing amount of the Stomach channel will be carrying Qi that moves back and forth, instead of flowing towards the foot. Eventually, after ten to twenty years (in most PDers) the back and forth pattern will be occurring in the part of the Stomach channel that travels up the neck to the back of the jaw. At some point, if the Qi runs Rebelliously for a long enough time, a new pathway will open up on the side of the face that will allow Stomach channel Qi to run upwards, instead of downwards. Once this happens, the Qi actually does flow in the reverse direction over most of its path.

As for the Stomach channel currents the overflow into nearby channels, the most common path I've seen for a sideways short circuit near the ankle shunts the Rebellious Stomach channel Qi into the Kidney channel, at the ankle. But even with a short circuit at the ankle, the Stomach channel Qi eventually starts to run backwards.

Current that alternates rapidly back and forth rather than flowing in the correct direction is called Rebellious, Retrograde, or “backward” Channel Qi. Sometimes, if the energy in a channel is sufficiently deranged, the direction of Qi flow will literally become backwards. The main source of Qi for the channel will not be able to flow into the channel, and will be diverted to some other channel or reroute itself to discharge out the fingers or toes. Qi flowing into the

¹ Distal means “moving in a direction away from the head.” Proximal means “going towards the head.”

Rebelliously running channel from the smaller branch tributaries along the length of the channel will flow backwards, or “upstream.” In Parkinson’s disease, over time, the overall feeling of movement in the Stomach channel is predominantly backwards even if the back and forth pattern is still occurring to a small extent in some portions of the channel. The predominant palpable (to a trained hand) sensation generated by the Rebellious Stomach channel current is that of a current running up the leg (towards the face) rather than down (towards the foot).

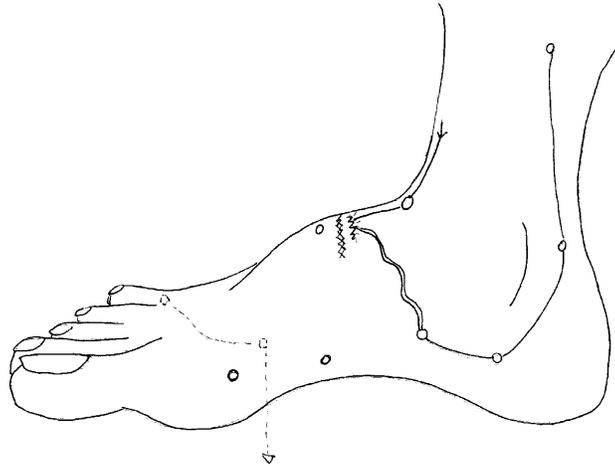


Fig. 8.1a

A common presentation of pathological Qi flow in the Parkinson’s disease foot

In Parkinson’s disease, Stomach channel Qi is impeded at ST-42 (marked with two rows of Xs in fig. 8.1). Because of the impediment, Qi flows backwards or sideways. Compare this drawing with drawing below that shows a healthy foot. Note the absence of Stomach channel Qi in the middle toes and the medial side of the big toe in the Parkinson’s foot. In the healthy foot below, most of the Qi in the Stomach channel becomes Spleen channel Qi after branching over from ST-42 to SP-3, on the side of the foot. A small amount of Stomach channel Qi goes out the second and third toes and then flows laterally across the tips of the toes to the big toe. Its name changes to “Spleen channel Qi” as it heads up the side of the foot to SP-3.

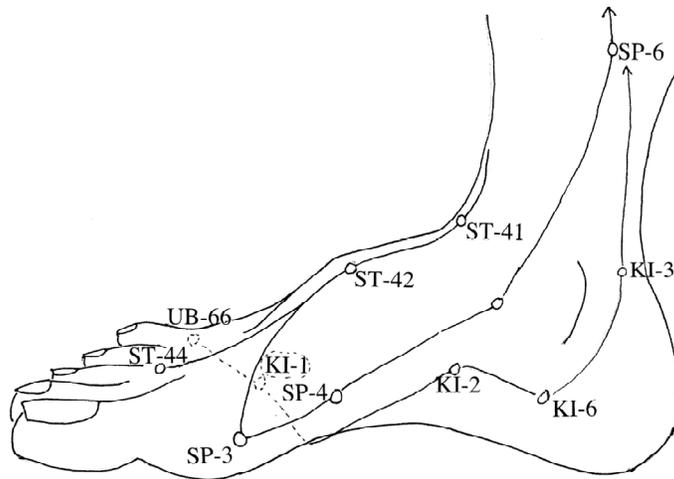


Fig. 8.1b

Figure 8.1 above also shows one of the many options for incorrect Qi flow: Stomach channel flowing into the Kidney channel at the ankle. This particular aberration and the static caused by it contribute to the weakened ankles, strange sensations in the ankles, and poor inner-ankle vascularization experienced by many PDers. Also, because the Qi from the Stomach channel is filling the Kidney channel, the Qi that should have gone from the sole of the foot over to the side of the ankle (shown with a dotted line) cannot fully flow into its already filled pathway. Because the Kidney channel Qi that flows under the foot from Ki-1 cannot flow into the channel at Ki-2, it grounds out to the floor. This grounding out pattern contributes to the feeling of magnetic pull that many PDers describe as “foot sticking to the floor.”

In PDers, after decades of disarray, Stomach channel Qi may even *cease* to palpably flow in the foot or lower part of the leg. Very often, by the time the Qi flow in the foot portion of the Stomach channel is no longer detectable by hand, the foot looks pale purple, or even gray. In these cases, where the foot feels almost dead, the area that feels blocked may extend farther back up the channel from ST-42, reaching up to the ankle or even the lower leg. In cases of advanced Parkinson’s, even when the lower leg portion of the channel ceases to have any *detectable* Qi flow, Stomach channel Qi may still be detected running rebelliously in the upper leg, torso, and neck portions of the channel.

In the lower leg, some of the Stomach channel’s current may short circuit into other channels. A not uncommon short circuit carries Stomach channel Qi into the nearby Gallbladder channel at or around acupoint ST-40, about halfway up the lower leg. Once the Qi begins to run backwards, nearly any aberration is possible.

Some PDers have foot injuries so broad that both the Stomach channel and the Gallbladder channel become blocked. If this is the case, Qi may run backwards in both of these channels.

Moving up to the head: blocking the Large Intestine channel

In a healthy person, the Qi flow known as the Large Intestine channel, on the arm, flows up the neck and over the face to the forehead. As the Large Intestine channel travels up the neck

and jaw, it must cross over the downward flowing Stomach channel at the top of the neck, near to ST-6, at the lower back corner of the jaw. When flowing correctly, the cross over schematic on the jaw quite elegant, energetically supports both the channels, and helps coordinated left-right arm-leg movement. At the forehead, the energy in the Large Intestine channel reverses course and is then called the Stomach channel.

When Stomach channel Qi is running backwards, the cross over point on the jaw where the Large Intestine channel Qi crosses over the Stomach channel becomes obstructed. Large Intestine channel Qi cannot traverse the Stomach channel if the Stomach channel is running Rebelliously. Instead, the Qi in the Large Intestine channel travels up from the hand to the neck, hits the blockage at the lower back of the jaw, and rebounds back down the arm. In PDers, the Qi that can be felt running Rebelliously in the arm's Large Intestine channel is running backwards because it cannot cross over the Rebellious Qi of the Stomach channel.

In a healthy person, the Large Intestine channel becomes renamed "Stomach channel" after it flows to the forehead and starts its downward flow. But if the Large Intestine Qi cannot cross over the Stomach channel at the back of the jaw, it cannot flow up to the forehead and become "the source" of the Qi flow known as the Stomach channel. Therefore, Stomach Qi flow is minimized. As the obstruction at the cross over point near ST-6 increases, the amount of Qi in the *face* portion of both the Large Intestine and Stomach channels diminishes. Note carefully: the amount of Qi on the face is not running Rebelliously: it is diminished. This difference plays a large part in the difference between the Parkinson's symptoms on the face and the Parkinson's symptoms in the arm, leg, and torso, where Qi is running backwards.

Extra sources for Stomach channel Qi

The Du channel, which flows up the skin covering the spine, flows up the back of the neck and over the top of the head, and then down the center of the face, provides a small portion of the Stomach channel's Qi. This portion of Qi continues to flow into the path of the Stomach channel even if the Large Intestine channel cannot get past the blocked cross over point on the neck. Therefore, the face will have *some* amount of Qi flow along the Stomach channel, but the amount will be much less than in a healthy person.

Also, one of the branch channels that flows from deep within the torso and which feeds into the Stomach channel from Ren 12 (several inches above the belly button) continues to supply Qi to the lower part of the Stomach channel. In Parkinson's disease, this auxiliary Stomach channel Qi, entering from halfway down the length of the Stomach channel, may have enough momentum to direct Qi *into* the Stomach channel. But if Qi in the Stomach channel is alternating back and forth, unable to flow down through the foot, this incoming energy will either move back and forth or, if the new channel has opened on the side of the head, will flow Rebelliously upward: headward.

Another source of input into the Stomach channel can occur – in a healthy person – where Heart channel Qi has a connecting line to the Stomach channel in the vicinity between ST-13 and ST-14, on the upper chest, along the mammary line midway between the collar bone and the nipple. This connecting line allows variations in the amount of Qi in the organs of the heart and stomach. When a person is in parasympathetic mode (awake and carefree, hungry, or curious), some Heart Qi flow towards the Stomach channel. Oppositely, during sympathetic mode (emergency, fearful, increased adrenaline-releasing mode), Stomach channel Qi flows into the heart, instead of flowing down the Stomach channel and branching into the stomach.

When in parasympathetic mode, this Qi flow diversion takes energy away from the heart, which slows down, and gives extra energy to the stomach to help with digestion. Oppositely, during sympathetic mode, the diversion gives the heart extra energy and stomach function decreases.

This example of Qi diversion can easily be felt in healthy patients who are asked to *imagine* themselves first in a relaxing, pleasant, scenario and then imagine themselves in a dangerous scenario that requires a strong, heroic response. As an interesting aside, this particular heart-stomach diversion is one that shifts quickly to the “emergency” position when a person has negative thoughts or even allows his subconscious to cultivate negative memories. This particular channel also plays a role in the dissociations used by people with Parkinson’s. We will return to this particular divergent channel later, in the chapters on treatment techniques for emotional blockages.

Variations in channel flow

In acupuncture books and in beginning level acupuncture classes, students are only taught one set of channel flow patterns for each channel. These lessons show patterns for primary and extraordinary channel Qi flow that occurs when a person is awake and in parasympathetic (*not* fearful) mode. However, the channel Qi flow patterns vary enormously depending on whether a person is awake, asleep, in “seeking” mode (curious, hungry, or playful), danger mode, or dissociated mode. The electromagnetic waves generated by emotions and thoughts also contribute enormously to activating the various electrical “switches” that direct the energy in divergent channels one way or the other.

In order to understand Parkinson’s disease, it is not necessary – or possible – to learn all the potential diversions and supplementary sources of Stomach channel Qi. I have introduced this material about divergent channels for two reasons. First, to help a person understand why the Stomach channel might have a perpetual source of new, incoming Qi, even if the normal origin of the Stomach channel (The Large Intestine channel) stops contributing. Secondly, to make the point that the Qi flow in a living system is fantastically dynamic, and changes in response to energy need, as well as in response to injury, and in response to thoughts and imagination. Channel Qi flows like water: it follows the path of least resistance. Some of the resistance is created by the very structures of the cells themselves, some is created by thought and heart waves, and some can be created by injury or illness. It is *not* necessary or even possible to learn all of the channel variations that might occur.

The patterns of channel Qi flow are *not* rigidly fixed. However, in a healthy person, whether sleeping, eating, or running for the bus, channel Qi always flows in the correct *direction*. If Qi actually becomes so disordered as to run backwards, this is referred to as Rebellious Qi. Rebellious Qi in the primary channels is *always* a sign that something is deeply wrong in the system.

Feeling the flow of Stomach channel Qi in people with Parkinson’s disease

In people with Parkinson’s disease, in the portion of the Stomach channel that runs between the foot and the belly, the Qi may feel as if it is going back and forth, or it may feel as if it is running distinctly upwards. Whether the Qi seems to be moving back and forth or straight up towards the head (running backwards) depends on the extent of the injury, whether or not there are injuries in other channels, and the extent to which the body is using some of the small connecting channels to circumvent the backwards-flowing Qi. However, in Parkinson’s, no

matter how many circumventing routes are established, the leg portion of the Stomach channel feels predominantly as if it is flowing backwards or back forth. The Stomach channel Qi does *not* flow in the normal fashion in people with Parkinson's disease. A trained hand can easily detect the perversions of Stomach channel Qi. Almost anyone can, with practice, learn to feel a patient's channel Qi as it flows in the primary channels.¹

¹ In my very limited teaching experience, I have noticed that people taking anti-depressant medications are sometimes no able to detect Qi flow. I have no idea as to why this might or might not be a general rule.

ELECTRICAL CHANGE IN THE HEAD PORTION OF THE STOMACH CHANNEL

The safety shunt at the jaw

Over the years, the Rebellious flow of Stomach channel Qi can affect nearly the entire length of the Stomach channel: Rebellious Qi in the Stomach channel flows backwards from the point of injury all the way up to the head. When this backwards-flowing Qi flows up the neck to the point at the back of the lower jaw, a point known as ST-6, the current is redirected – it can no longer flow backwards along the path of the Stomach channel.

A protective, one-way flow regulator exists at ST-6, on the jaw. This flow regulator is an electrical configuration that serves to prevent backwards-flowing Qi from flowing backwards in the face portion of the Stomach channel.

The face portion of the Stomach channel, in a healthy person, intersects with a consciousness-sustaining point on the forehead. The regulator on the jaw prevents backwards-flowing Qi getting into this point on the forehead. When *backwards*-flowing Stomach channel Qi gets to ST-6 at the back of the jaw, it is shunted away from the path of the Stomach channel on the front of the face, and redirected up the *side* of the face just anterior to the ear, and up to the forehead.¹

This shunted channel terminates at ST-8, a point on the side of the forehead to which flow is directed only when a person has a severe injury, severe stomach pain, or severe emotional pain: events that might block the flow of Qi in the Stomach channel for a finite (hopefully) period of time. The acupoint ST-8 on the forehead acts as a sort of reservoir or electrical capacitor for excess Qi; electrical charge can build up at this point. When Qi builds up at this point, it can cause a headache in the temple area. The headache is a signal that all is not well; the headache should make a person want to lie down. The headache is one benefit of this shunt; the nap taken because of the headache is sometimes all that is needed for the body and mind to process the physical or emotional injury.

¹ Although current teaching about the Stomach channel dogmatically states that the Stomach channel *must* flow in the sequence suggested by the new acupoint-numbering system, this new teaching can be easily refuted. Feel with your hand the Qi flow of the Stomach channel on the face of any *healthy* person: the Qi runs down the face, spreading out over the cheeks as far over as ST-7. Stomach channel Qi does *not* normally flow into that branch that visits ST-8. Qi can only be felt in the branch that runs from ST-6 to ST-8 when there is some problem in the Stomach channel.

As for the point numbering system, that is an extremely new addition to the lore – it was added in the 20th century in an attempt to modernize and westernize the acupoint system. This was around the same time that the existence of channels was disavowed. I have been extremely fortunate both in having teachers that were able to explain the correct course of the Stomach channel to me and in being personally able to confirm these teachings by feeling the actual path of the Qi flow.

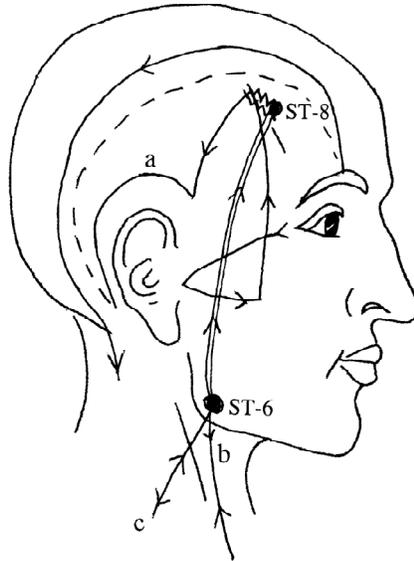


Fig. 8.2a The Parkinson's disease pattern of facial Qi flow

In Parkinson's disease, the (b) Stomach channel is running up the torso, rather than down. When the Stomach channel Qi gets to the face, it runs up to the short-circuit point at the top corner of the forehead, and from there it arcs over into the (a) Gallbladder channel. The zigzag lines at the top corner of the forehead indicate the location of the point where the Stomach channel Qi flows into the Gallbladder channel. The dark circle on the chin shows both the site of the shunt at ST-6 and the location where the Large Intestine channel becomes blocked as it tries to cross the path of the rebellious Qi at ST-6.

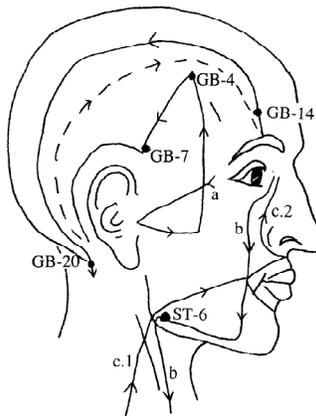


Fig. 8.2b The correct, healthy flow of facial Qi

Compare Fig. 8.2a with 8.2b. Note the fullness of electrical patterns crossing the face in the correct, healthy flow. (For the benefit of curious readers, the dotted line shows the erroneous, academically selected flow pattern for Gallbladder Qi flow, as of the 20th century.)

Note in 8.2a the absence of any Qi in the cheek, jaw, or mouth area in the face in the Parkinson's pattern. This absence corresponds to the PDer's decline in facial expression, lack of vigor in the lower eyelid, drooling, sinus problems, seborrhea, lack of control of the salivary glands, and decline in the senses of smell and taste.

Short circuit into the Gallbladder channel

If and when the build up of electrical charge at ST-8 becomes too great, it causes the backwards-Qi accumulation at ST-8 to “short circuit” into the nearby Gallbladder channel. With a spark from built-up charge, the Stomach channel Qi zaps across from ST-8 into the Gallbladder channel near GB-4. The burst of short-circuiting Qi can make a person dizzy, or cause a severe headache. From GB-4, the Qi flows into the Gallbladder channel, which runs across the side of the head.¹

If the Stomach channel short circuits frequently enough from ST-8 into the Gallbladder channel, a new, semi-permanent electrical pathway will become established. In this new, semi-permanent pathway, Qi can flow from ST-8 to the Gallbladder channel *steadily*, rather than in bursts.

Semi-permanent means, in this case, permanent until the electrical flow in the Stomach channel ceases to flow Rebelliously.

The reason for the safety valve on the jaw at ST-6

The electrical configurations at ST-6 at the back of the jaw serve as a directional regulator that prevents rebellious Stomach channel Qi from *ever* flowing backwards onto the face, into the critical facial junctions with the Du (“Governor”) channel (which runs up the spine and over the head to the face and upper lip) and Ren channel (which runs up the midline of the front of the torso, neck, and lower lip).

Ordinarily, when the healthy, *downward* (correctly flowing) Stomach channel departs the forehead and skirts the upper and lower lips, it intersects (and is in part powered by) the surging energy in these two major channels, the Du and the Ren. The Stomach channel intersects the Du and the Ren at acupoints located near the mouth, on the midline of the face just above and below the upper and lower lips, respectively. Even more importantly, the healthy Stomach channel also intersects the Du channel at the point between the eyebrows, a point named Yin Tang, a major point of the Du channel. (See Fig 8. below, xxx.)

¹ The Gallbladder channel is quite wide as it traverses the side of the head. The modern convention of drawing a linear path to sequentially connect the modern “numbered” points of the Gallbladder channel requires the invention of an awkward internal current looping back to the forehead from GB-12 behind the ear to get over to GB-13 on the forehead. No such looping path actually exists. The Gallbladder channel covers a wide swath that runs along the side of the head from the forehead, side of the temple, and front of the ear, all the way to the back of the head, in one broad panel. The numbering system of the Gallbladder channel is awkward, and the channel lines invented to connect the points in numerical order are misleading.

An acupuncturist with an inquiring mind can verify the presence of this wide band of Qi, as opposed to two skinny lines of Qi connected by an internal, backwards flowing skinny line of Qi, by feeling the Qi flow on the side of the head. Note that the *amount* of Qi in the side-of-the-head part of the Gallbladder channel feels somewhat smaller than the amount of Qi flow that can be detected in narrower portions of the channel, such as the flow of Qi at GB-20. This seemingly decreased amount of Qi in the channel on the side of the head is merely caused by the Qi being spread over a wider area. This feeling of “less power” is similar to the way that a river seems less powerful when it emerges from a narrow cut and flows into a wide flatland. The river still has the same amount of water and potential downhill energy when it is spread out, but at any given spot in the flatland portion, the amount of power in the river will *seem* to be less than the amount of power that was rushing through the narrow cut.

The Gallbladder channel, as it courses down the sides of the torso, spreads out again. Although the named points on the Gallbladder channel seem to define a zig-zag course down to the ankle, the channel actually flows in a wide, straight path. The points define the boundaries of both sides of the swath.

Points of intersection of the Stomach and Du channels on the face

Yin Tang is the point where the upwards-flowing Large Intestine channel reverses direction and starts flowing *down* the face. When the Qi starts its downward flow, it is no longer called the “Large Intestine channel;” it is called the “Stomach channel.” The Stomach channel has two points at which it connects with the Du channel: Yin Tang, and the point at the midline of the upper lip.

The Du channel, carrying spinal column energy, is the most powerful channel in the body. The Stomach channel’s two facial points of contact with the Du serve as energizing stations to keep the Stomach channel charged and moving.

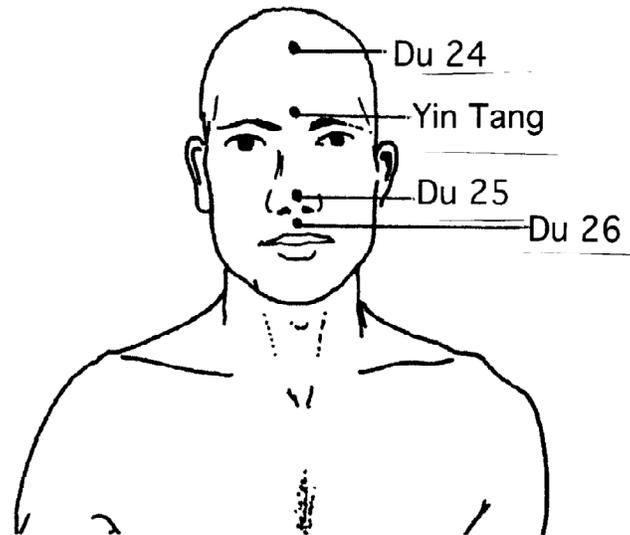


Fig. 8.3 Face points of the Du channel

The Du channel runs along the spine from the coccyx up to the neck and then into the midbrain, frontal lobe, and forehead. A branch of the Du runs over the top of the head and down the forehead, where it rejoins the main channel at Yin Tang. The Du then continues down the front of the face to the upper lip.

Yin Tang is a major point on the body: half of the body’s channels terminate or originate at Yin Tang. A few sequentially numbered points on the Du channel are also shown above, merely to demonstrate the direction of flow in the Du channel on the face.

The danger of backwards-flowing Qi in the Du channel

The main part of the Du channel runs through the head, from the brain stem area at the top of the neck, and into the center of the brain. When a person is conscious, the channel flows through the frontal lobe of the brain and emerges on the forehead at Yin Tang. This channel directs activity in the midbrain and brain stem – the seat of physical life and consciousness. If the Qi of the Du channel were to run *backwards* through the head, which is to say run from the forehead to the midbrain and then into the brain stem, loss of consciousness or even death might ensue.

If the Qi running through the head portion of the Du channel is impeded in any way, consciousness is affected.

This is why the directional regulator ST-6 is so important. If backwards-running Qi from the *Stomach* channel were able to flow backwards into its points of intersection with the *Du* channel on the face it could derange the part of the Du channel that traverses the middle of the head. Alterations in consciousness, and even death, might ensue.

Fortunately, the body has the shunt at the back of the jaw, at ST-6, to prevent backwards-flowing Qi from the Stomach channel from *ever* flowing onto the front of the face where the Stomach channel intersects the Du channel. If Stomach channel Qi *does* flow backwards as far up the body as the lower jaw, it is shunted to the side of the forehead, to ST-8.

As noted earlier, although the numbering system may suggest that ST-8 is linearly connected to the rest of the Stomach channel, the Stomach channel Qi does *not* flow up to ST-8 under ordinary circumstances: in a healthy person, Stomach channel Qi flows *down* from the Du channel at the forehead, down the front of the face to the jaw, and then over to ST-6 at the back of the jaw. From ST-6, it flows down to ST-9 on the neck. From there, it flows down to the feet.

But, as explained above, in the case of rebellious Qi in the Stomach channel, when the Qi flow in the channel path is reversed, the Qi is prevented from flowing backwards from ST-6 over to the front of the face and into the Du. Instead, it is shunted upwards to ST-8 on the side of the head, thus preventing a calamitous reverse flow through the critical life- and consciousness-maintaining Du channel.

As an aside, the activity of rebellious Qi at ST-6, the site of the shunt, can cause the pain in the lower jaw and/or lower back molar pain that is reported by some PD patients, and for which dentists can find “no cause.” Also, some PDers notice a dull pain, a pressure, or a buzzing sensation or sound at ST-8, where Qi accumulates. These sensations may be chronic or intermittent.

The Stomach channel short-circuit into the Gall Bladder: a rarity

As already noted, a *small* accumulation of Qi at ST-8 merely causes a headache at the temple, if anything. However, if the Rebellious pattern in the Stomach channel continues long enough or with enough severity, Qi can eventually over-accumulate at ST-8. If this happens, the Qi arcs into the nearest part of the Gallbladder channel, near GB-4. This short-circuit is not something that is supposed to happen every time that Qi builds up somewhat at ST-8. This rare and somewhat violent short-circuit only occurs when Stomach Qi is severely blocked, and the Stomach channel Qi runs powerfully in the wrong direction. Such a shift in Qi flow may occur as a healthy response to a severe injury such as a broken leg.

When the short circuit does occur, it sets in motion a collection of perfectly correct, “healthy,” injury-related events, including the inhibition of dopamine release in the midbrain. This inhibition replicates the midbrain dopamine inhibition that occurs during sleep. When this dopamine inhibition occurs in the context of an injury, it forces an injured person to have some mental and physical “down time” during which he does not have enough dopamine to perform the usual activities of daily living.

Then again, a person can always override the movement inhibition by using adrenaline.

The mechanism for inhibiting dopamine release

When the ST-8 to Gallbladder short-circuit occurs, the Stomach channel energy flows into the Gallbladder channel. This input causes the Gallbladder channel to carry a greater load of Qi, a higher amperage if you will, than it ordinarily would. The Gallbladder channel ordinarily only has higher amperage at night, starting at around 11:00 pm. This increased amperage causes dopamine inhibition and allows consciousness to ebb: in other words, it causes sleep.

The shunting of Stomach channel Qi into the Gallbladder channel causes higher amperage in the Gallbladder channel. Therefore, a go-to-sleep signal is initiated in the midbrain whenever this shunt occurs. If the shunting of Stomach channel Qi into the Gallbladder channel becomes chronic, the Gallbladder channel *constantly* creates a “go to sleep” signal and *constantly* inhibits the release of dopamine.

In a healthy person who has this pattern because of injury, the pattern is only temporary. As soon as the injury is sufficiently healed that Stomach channel Qi can again flow somewhat correctly, or at least not backwards, the Stomach Qi no longer shunts into the Gallbladder channel. Full alertness – a *non-emergency*, dopamine-based alertness – is once again possible. In PDers, because the injury never heals, the shunt into the Gallbladder channel becomes semi-permanent. The “go to sleep” electrical instruction becomes semi-permanent. The inhibition of dopamine becomes semi-permanent.

Most PDers have an unhealed injury on one foot only. Stomach channel Qi is therefore Rebellious on only one side of the head. Therefore, the brain only receives half of a go to sleep signal. Dopamine released is only *partially* inhibited. And remember, these go to sleep signals can be overridden by using adrenaline instead of dopamine.

The semi-permanent short circuit in PDers

In PD, the overflow into the Gallbladder channel becomes an established, semi-permanent pathway for flow of rebellious Stomach channel Qi.

Some of our patients have a distinct memory of a sudden memorable pain or noise in the temple on the more parkinsonian side of the body, or a sensation as if the brain or the room was spinning.

We hypothesize that these memorable moments occurred when the Qi first started short circuiting into the Gallbladder channel. In those of our patients who volunteered that such an event took place, it had usually occurred about ten to twenty years after the injury event. At the time, the short circuit may have only been intermittent, or it may have immediately become chronic: there is no way to know.

Evidence that supports the side-of-the-head short-circuit hypothesis

Some PDers recall a single unique incident, usually in their late teens or early twenties, during which they felt as if something inside their head “went into a spin,” “exploded,” or “received a huge electrical shock.” Some simply felt momentarily dizzy or heard a buzzing or a “zzzzzzt” sound on the side of the head in the vicinity of ST-8. Some felt the freak dizziness and then experienced a brief lapse of consciousness, finding themselves, a moment later, lying on the floor. Others merely grabbed a passing sofa or bureau and clung to it for a moment until the room stopped spinning.¹

¹ One patient was relieved to hear about this pre-Parkinson’s short circuit. His had occurred when he was riding his bicycle at age seventeen. He was riding along a country road, perfectly alert, and the next thing he knew

The Effect of the Gallbladder Channel on the Du Channel

To enable the reader to appreciate the elegance and biological perfection of this dopamine inhibition system, these next few sections will go deeper into the relationship between the Gallbladder and Du channels.

The main channel that regulates alertness, the Du channel, runs *from* the brain stem *to* the forehead. The Gallbladder channel runs *from* the forehead *to* the back of the head. In other words, the Gallbladder channel on the side of the head and Du channel in the center of the head run in opposite directions.

When the Gallbladder channel runs at its usual amperage, the Du channel is unaffected. When the Gallbladder channel has an increase in amperage, it causes a decrease in the amount of amperage flowing through the Du channel. This is a basic principle of physics.

When the flow of the Du channel decreases, dopamine levels decrease. Therefore, dopamine levels *decrease* when the Gallbladder channel runs at higher amperage than usual.

Each of the twelve primary channels runs at higher amperage for approximately two hours a day. This two-hour increase drives the circadian rhythms in the body. The Gallbladder channel in a healthy person runs with increased amperage every night, beginning around 11:00 p.m. (2300). The increased amperage lasts until 1:00 a.m. This causes the Du channel to be sedated, enabling a person to fall asleep.

After 1:00 a.m., even though the Gallbladder channel resumes its regular, lower level of amperage, the Du channel does not receive enough stimulation to resume flowing at full strength.

A healthy person will be able to stay asleep even after the Gallbladder channel resumes its lower level of flow. In the early morning, from around 5:00 a.m. to 7:00 a.m. the Large Intestine channel takes its turn at flowing with increased amperage. The Large Intestine channel flows up to the forehead and intersects with the Du channel at Yin Tang. When the Large Intestine channel is flowing with increased amperage, between 5:00 and 7:00 a.m., the Du is stimulated out of its sleeping mode via the increased stimulation at Yin Tang. The sleeper returns to alertness.¹

The main point of the above details is to make it clear that sedation of the Du channel by the Gallbladder channel is a perfectly normal, healthy event. It occurs every night. When the Du channel is somewhat sedated by an increase in Gallbladder channel Qi, dopamine release in the midbrain is inhibited. If it were not inhibited, we would walk, chew, and generally disport ourselves in our sleep. People who do walk in their sleep have a problem inasmuch as their dopamine is not being adequately inhibited during sleep. In other words, sometimes dopamine inhibition is a good thing. Another reason to introduce the subject of the two-hours cycles and the circadian rhythms is because the cycles helped us make sense of certain recovery symptoms.

he was a good quarter mile farther down the road with no recall of any consciousness during the last quarter mile. He'd felt something on the side of the head, and he felt somehow mentally altered. In the privacy of his own mind, he had always laughingly referred to the incident as "my abduction by aliens." He had been afraid to tell anyone about his head-shift experience at the time, as were all the PDers who told me of their "brain shift."

¹ These clock times are approximate. They are local times, not Greenwich Mean Time. Also, during summer and winter, the "two-hour" spans expand or contract a bit fit the increase or decrease in sunlight. If a person's life style conflicts with the clock times for increased amperage in each channel, the body is able to make some accommodations. However, optimal health ensues if a person honors the natural times of day that are best for waking, eating, and sleeping – the times when the channels for those functions are running at increased amperage.

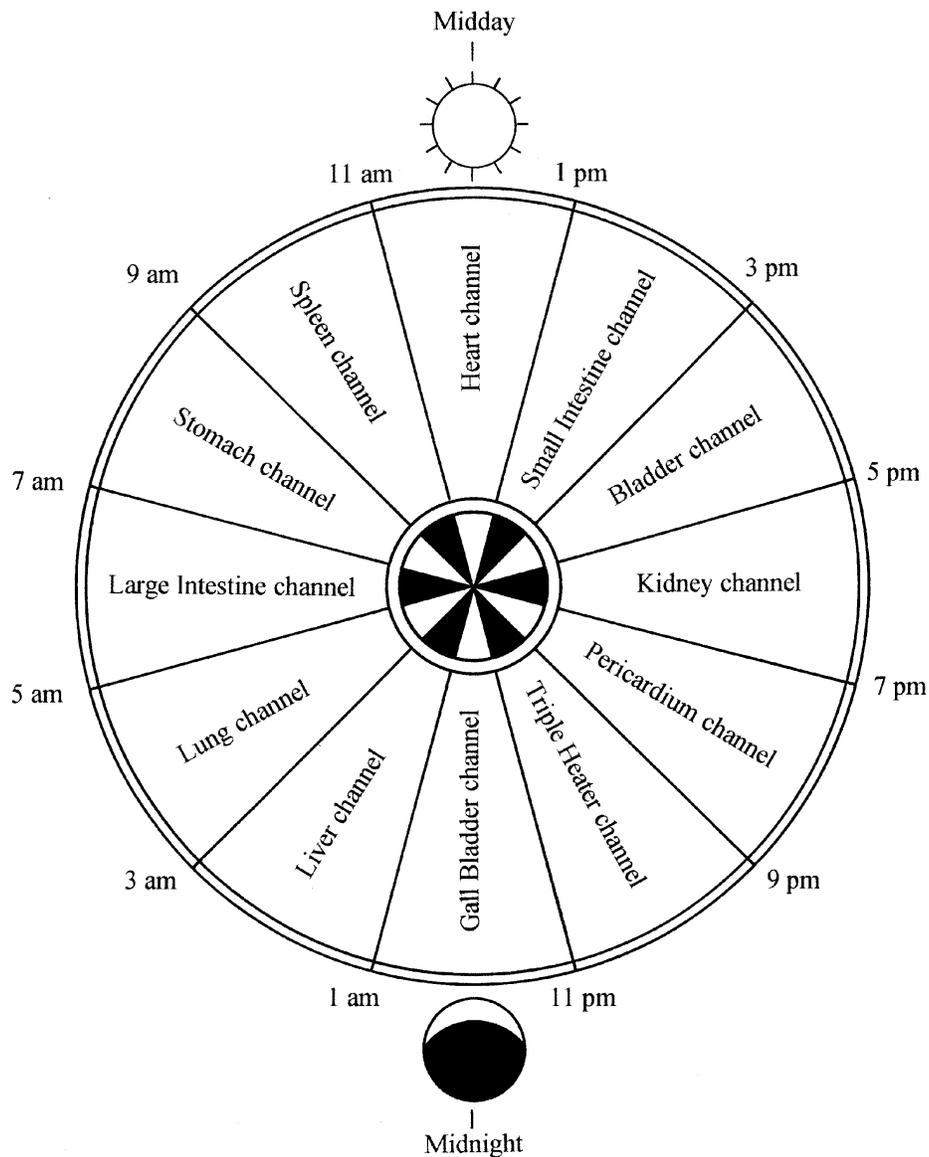


Fig. 8.4 The Asian “Law of Midnight-Midday”

It is recognized in Asian medicine that every channel has an approximately two-hour span during which channel Qi levels increase significantly. This period corresponds to the time when the related organ receives more vitalizing nutrients and when organ or channel repair work, if needed, is done.

This cycle *can* be moved forward or backward if a person regularly goes to sleep at a time other than 10:30 to 11:30. For example, during daylight-savings time, the cycle makes a shift to account for the later bedtime.

Interestingly, these time-based variations in energy flow in channels and their corresponding organs may account for the fact, recognized in western medicine, that surgery done on certain organs has a better outcome at certain times of the day. However, these anecdotal observations are not put to use: this information is disregarded when scheduling surgeries.

The secondary point is that this same dopamine inhibition can occur following a severe injury, when a person will benefit from peaceful rest. The mechanism for inhibiting dopamine following an injury is that Rebellious Qi from the injury short circuits into the Gallbladder channel on the head.¹

More about the Du

The Du (Governor) channel is the most important channel in the body. As noted already, it runs from the base of the spine and up the spine to the neck. It then flows from the base of the skull (at the brain stem, acupoint Du-15) and into the head. It passes through the brain stem and midbrain, directing physiological processes in the brain stem and midbrain. When a person is conscious, the Du channel emerges from the head at the center of the forehead at Yin Tang, the point between the eyebrows.



Fig. 8.5 The head portion of the Du channel, external and internal branches

The line that travels the midline of the top of the head shows the external branch of the Du channel. The dashed/solid/dashed line that goes through the middle of the head represents the main path of the channel. The latter branch flows, not along the surface of the skin, but through the very center of the head (the midbrain). This branch rejoins the external branch of the Du channel at the forehead.²

¹ Serious injury along *other* channels that flow from the head to the feet can result in backwards-flowing Qi being shunted from these channels into the Stomach channel. As long as this backwards pattern is foisted off on the Stomach channel, the Stomach channel may also be induced to run backwards, or at least back and forth, as it accommodates the incoming backwards Qi flow from injury on *other* channels. If the Stomach Qi flow is disrupted enough, and flows back and forth or even backwards, it can trigger a short circuit into the Gallbladder channel that help a person rest while doing his initial healing – even though the injury was not actually on the Stomach channel.

² A note to acupuncture adepts: thinking of the Du channel in terms of its named points, one may start to think that the *main* branch of the Du is the branch that runs over the top of the head. The old texts say that the *main* branch of the Du runs from the base of the spine up to the nape of the neck and then flows into the brain.

From Yin Tang, the Du flows down the midline of the face to the upper lip. From here, the Du runs internally through the gastrointestinal tract, emerging at the anus and flowing back up the spine, making an unending loop of current.

At the base of the skull, a branch diverts from the main flow of the Du channel. This branch, sometimes called the “external path” of the Du channel, passes up the back of the head, over the top of the head, and then descends down the midline of the face to Yin Tang. At Yin Tang, this external branch of current rejoins the main current of the Du channel.

As an aside, the word “Du” is usually translated into English as “Governor,” in the sense that the Governor is subordinate only to the King. The King of the body is, of course, the heart. The heart is more crucial than the brain: a person can be “brain dead” but still alive. When King Heart dies, life ends.

A *branch* of the Du diverges (the old texts do not say where, but it is logical to assume that the branch diverges at the nape of the neck), running up to the vertex, over the forehead, and down the bridge of the nose. This latter branch of the channel is the one with the named points. (Continued on next page.)

The old texts do not specify where the main branch, the internal branch, emerges. This may be because the amount of Qi in the internal branch varies over the course of the day. When the energy in the main branch predominantly emerges at Yin Tang, one is conscious, clear, and calm. When one is sleeping, the Qi of this branch is barely active at Yin Tang; more of the Du channel Qi remains in the deeper portions of the brain. When a person is sleeping, the great Du “loop” is somewhat inhibited, and gastrointestinal function slows dramatically. And in cases where the Qi in the brain becomes deranged, *not* emerging at Yin Tang during waking hours, a person has less clarity and calmness. For example, in schizophrenia, excess neural activity is observed in the hippocampus, and less than normal activity is seen in the frontal lobe.

Modern English texts sometimes say that the Du channel flows into the brain and a branch flows up to the vertex. This phrasing suggests that the second branch, the external branch, is flowing out of the middle of the brain stem, up to the top of the head, and thence into the named points. One must admit to the potential existence of a branch that emerges in the midbrain and goes to the vertex. However, *significant* amounts of Qi only flow in this pattern, making this the main branch of the Du, when one is an advanced spiritual adept, capable of carrying the (Continued on next page.)

waking consciousness away from its normal home in the frontal lobe, up to the higher point at the top of the head. Usually, the Qi flow pattern of the *main* branch of the Du in healthy, *conscious* individuals is the one that flows through the brain stem and frontal lobe and emerges at Yin Tang.

The acupuncturist who is uncertain as to the “correct” flow patterns of the main branch of the Du channel might want to sit quietly for several minutes and watch where the Qi in his own head tends to flow. He may also want to sit quietly and see what happens as he sends Qi through various pathways in his head, including backwards.

Certainly, even the most pedantic acupuncturist knows that the calming, mind-focusing benefit of needling Yin Tang does *not* come from increasing the flow of Qi through the *scalp* over the top of the head. The benefit of needling Yin Tang comes from enhancing the flow of Qi through the midbrain: increasing the flow from the brain stem through to the frontal lobe.

Possibly for reasons relating to modern Chinese political, anti-spiritual policy, Yin Tang is considered an “extra point,” and is not even placed on the Du channel. Incredibly, this point named “meeting hall” is not considered, in the modern, politically correct texts, to meet with any other points! Future research on acupuncture channels will show that the channels are not short chunks of Qi, beginning and ending in the middle of random bits of flesh, as modern interpretations of the classics would have us believe. The channels are completely integrated, each arising from and flowing into one another.

“Tang” means meeting hall. Yin Tang is the crucial meeting hall for the Yin (front side) part of the body; all of the Primary Yang channels on the Yin (front side) of the body either end or begin there. Yin Tang is where the body’s formative soul energy, which enters at the back of the neck (its Yang aspect), is mirrored. This mirror, at Yin Tang, at the third eye, is only a *reflection* of the light of the soul. Thus, it is “Yin,” which means “away from the light.” Yin Tang is also the place where the mighty Du, the channel that governs all others, meets with the Yang channels on the Yin side of the body: a “Yin meeting place” indeed!

Update: In 2007, an international group decided that Yin Tang *is* once again on the Du channel!

Review: the influence of the Gallbladder channel on the mighty Du

This next section is redundant, but it is crucial that a person with Parkinson's understands that dopamine inhibition is not necessarily pathological. Therefore, bear with me as I review the manner in which the Gallbladder channel influences the Du.

As noted already, the head portion of the Gallbladder channel runs parallel to and *in the opposite direction* of the Du channel. Because the Gallbladder channel runs in the opposite direction of the Du channel, a surge in Gallbladder channel Qi serves to diminishes the force of the Qi in the Du channel.

This relationship between opposing currents is a basic principle of physics: when electric current "A" runs counterflow to adjacent current "B," both currents modify each other.

As noted earlier, when the Gallbladder channel increases in amperage, it *decreases* the Qi flow in the Du channel. When the Gallbladder channel has an increase in current, the flow in the Du channel is diminished and consciousness is diminished.

When the Gallbladder channel flow decreases again to its normal level, the Du channel is potentially able to resume its role in regulating alert consciousness and parasympathetic (non-emergency, non-fearful) motor functioning.

The Gallbladder channel thus serves as a switch to decrease the powerful Du channel currents when needed.

Although sleep is a complex phenomenon and in humans can be influenced by thoughts emotions, diet, and myriad factors, and the physiological factors that induce sleep can be overridden by adrenaline, the increase in Qi in the Gallbladder channel may be one of the strongest triggers for the sleep process.¹

An aside: the path of the Gallbladder channel on the head

While the following details might be over the top and absolutely unnecessary for the lay reader, acupuncturists and researchers can use the following reminders to see how the head portion of the Gallbladder channel runs in the "opposite" direction of the Du channel:

The path of Qi from GB-14 through GB-20 runs parallel to the Du channel but in the opposite direction. The path of Qi from GB-8 to GB-12 also runs in the opposite direction of the Du channel.

¹ The following question arises for some acupuncturists: Why don't the currents of the (Urinary) Bladder channel, during its maximum flow period at 3:00-5:00 p.m., create the same type of inhibitory effect on the Du channel? After all, they too run parallel and in the opposite direction of the Du. The answer is, they do. Many people do experience a mild slump in the late afternoon. In some cultures, this time of day, when the Bladder channel is mildly inhibiting the Du channel, is nap time.

However, with physical activity, a person can override the mild inhibition of the Du that occurs during the hours when the Bladder channels runs at higher amperage. As the Bladder channel courses downward and parallel to the spine (BL-11 through 31, and BL-41 through 53), two simultaneous effects are induced: (1) the downward flow of the Bladder channel inhibits the flow of the Du channel; and (2) activity in the body increases the electrical activity of the nerves which feed into the spinal column, inducing an excitatory effect on the upward current flow in the Du channel. The result: the excitatory effect can offset the inhibitory effect. A nice balance is thus achieved if a person is active. Many people will attest to the need to activate a little bit of adrenaline to override the body's natural desire for a short nap during this time when dopamine release is mildly inhibited due to Du inhibition via the increase in the (urinary) Bladder channel.

Perpetual excess in the Gall bladder channel in PD

In healthy people, the Gallbladder channel only produces a “stop dopamine” signal during the surge of Gallbladder current that precedes sleep initiation. In PDers, this signal is sent throughout the day and night.

In Parkinson's, because of the short circuiting of Stomach channel Qi into the Gallbladder channel at the forehead on the side of the body that sustained the foot injury, a constant level of excess current flows in the Gallbladder channel on that side of the body. Therefore, the sleep initiation/stop dopamine *release* signal is broadcast to the midbrain twenty-four hours a day – on one side of the brain. Over decades, this dopamine inhibition on one side of the head causes the partial substantia-cell dormancy that eventually, over decades, puts the brain's dopamine *production* system into partial hibernation.

Asymmetry in the channels of the head

In Parkinson's disease, the Qi of the Gallbladder channel is usually excessive on only one side. (As an aside, we have seen PDers with injuries on both feet. These PDers have excess Gallbladder channel Qi on both sides. Also, they tend to not have tremor symptoms.)

This excess on one side of the head somewhat effects the Du channel. At the same time, on the uninjured side of the body, the healthy Gallbladder current is exerting its own correct effect on the Du channel. With one Gallbladder channel coursing normally (at correct Qi level) and the other coursing excessively, the net effect is one of a *partial* decrease in dopamine release and dopamine-based consciousness. Thus, the overall inhibition of the Du channel is not severe enough to induce sleep. However, the partial inhibition of dopamine allows a person to maintain a state of pre-sleep muscle relaxation and diminution in overall dopamine levels. Meanwhile, if a person is unable to relax or let his guard down, the presence of an ongoing injury tilts the body towards the sympathetic system and away from the parasympathetic system. This means that the partial dopamine inhibition can be overridden with adrenaline.

Adrenaline and dopamine levels in the body are related: when one increases, the other decreases. When adrenaline is the dominant neurotransmitter, dopamine levels are reduced. A person with an unhealed injury who is adrenaline-dominant, unable to relax and enjoy the healing calm of his injury-induced dopamine-inhibition, will actually experience a *second* source of dopamine-inhibition: his elevated adrenaline levels. In *many* PDers, dopamine levels are also reduced because of the adrenaline dominance that occurs when a person can't let down his guard or dissociates from his ability to feel pain.

Overriding the Parkinson's pre-sleep condition

In Parkinson's disease, the levels of dopamine are reduced all day long because of the Stomach channel Qi being shunted into the Gallbladder channel.

The effect of slightly excessive Gallbladder Qi coursing through one side of the head (while all the other body channels except the Stomach channel continue to flow in their correct pattern) merely triggers the first step in the complex pre-sleep process. It also creates a state in which there is a physical/muscular sense of pre-sleep weariness all day long, together with inhibition of curiosity, imagination, and playfulness.

Even so, mental alertness can be maintained through exertion of adrenaline-based (fear-based) will-power and/or the intentional creation of a sense of urgency, intensity, or even false emergency. Any of these can force the body to release higher levels of adrenaline, which then serves as a substitute for the decreased dopamine. In a healthy person, both dopamine and

adrenaline are in use throughout the day for various tasks. In the PDer, the decline in dopamine availability is supplanted with adrenaline.

This shift over to reliance on adrenaline is often assisted through exertion of negativity-based will power, guilt- or fear-driven determination, or even the cultivation of adrenaline-releasing, wariness-based thoughts such as “If I don’t do the job, it won’t get done” or “If I want it done right, I have to do it myself.”

This attitudinal posturing can create thoughts that resemble those that are set in motion by excess levels of dissociation. There is no biological difference between a purely mental dissociation-induced adrenaline increase and a healthy, temporary increase in adrenaline that enables a person to keep going even when injured is hard to determine: after all, the ability to keep going when injured is activated by switching over to a mental state that is dissociated from the injury.

After an individual has lived for decades with an unhealed injury, it can be hard to determine how much of an individual’s tilt towards adrenaline-based living is a reasonable response to the fact of dopamine inhibition and how much is emotionally pathological and preventing the body from being able to know about and heal the injury. In the end, the question may be moot. It doesn’t matter if one can figure out exactly what behavior started which compensating behaviors. The great thing is to get rid of the dissociation and heal the foot injury.

The emotional aspect of the adrenaline dominance problem will be discussed further in chapters on dissociation. This chapter is primarily addressing the Gallbladder’s *electrical* inhibition of dopamine.

LONG TERM CHANGES FROM QI FLOW CHANGES ON THE HEAD

Decrease in dopamine-producing cells

Possibly because of the myth that Parkinson’s disease is caused by a decrease in dopamine production, most PDer’s in my experience have been terribly preoccupied with what their dopamine “is doing.” Therefore, although the decrease in midbrain dopamine-producing cells is a side effect of the overall Parkinson’s picture, I will address that first even though it is not actually the most important change to take place as a result of the Qi flow changes in the head and face.

Dopamine, like all body chemistry, is produced on an “as needed” basis. In Parkinson’s, the signal of “need” for dopamine is perpetually reduced because Du channel Qi flow, which triggers the signal to release dopamine, is constantly running at a decreased level. The Du channel Qi flow is constantly running at a decreased level due to a constantly higher level of amperage in the Gallbladder channel. The Gallbladder is constantly running higher because the Stomach channel Qi is constantly flowing into the Gallbladder channel from ST-8. Stomach channel Qi is constantly flowing into the overflow point at ST-8 because an unhealed injury on the foot it causing Stomach channel Qi to flow backwards. The Stomach channel Qi is flowing backwards because it cannot get past the foot injury.

So, the Du channel is inhibited via the foot injury: a perfectly normal consequence of injury, and one that is supposed to induce dopamine inhibition and “healing rest.”

However, if the foot never heals, dopamine inhibition becomes constant. Following the “use it or lose it” principle of physiological efficiency, this lack of dopamine usage results in gradual reversion to neutrality (re-undifferentiation) of dopamine-producing cells. In the decades following the injury, because of minimal use of midbrain dopamine, the same “use it or lose it”

principle may result in a reduction in dopamine receptors, reuptake enzymes, and all parts of the dopamine system chemistries.

Changes in motor, sensory, and proprioception functions along the effected channels

Electrical current running backwards in the Stomach channel, starting at the center of the foot and going all the way up to the jaw, sends incorrect signals to all the tissues in its zone of influence. A channel's zone of influence covers all the cells that receive electromagnetically signals from the channel. In some areas, this zone is a fraction of an inch across. In some areas, the channel is influencing cells over a distance of several inches. The influence extends from the skin into the areas deep under the skin, influencing bone growth and organ function.

Changes set in motion where Qi runs backwards

In places where Qi is running incorrectly or backwards, the aberrant Qi causes cellular alterations, muscle rigidity, and organ malfunction or decreased function, all along the reversed path of the channel. In Parkinson's disease, these changes are especially pronounced on the side of the body that has the foot injury. Symptoms usually develop first on this side of the body.

Also, nerve contact declines between the brain and the body areas that are under the influence of backwards Qi. This decline involves nerves that regulate proprioception, sensitivity, and motor function. We know this because of the sensations that occur when these nerves return to health. The chapters on recovery symptoms describe the painful and unexpected recovery symptoms that helped us to realize the enormous amount of nerve dormancy that occurs in Parkinson's.

Although the changes in cellular function, muscle rigidity, and nerve function may be worse on the side with the foot injury, the problems may eventually become bilateral due to the mutual influence of paired electrical currents. The basic physics phenomenon of the mutual influence of paired electrical currents will be discussed in the next chapter.

Changes set in motion where Qi ceases to flow

The disarray in the portion of the Rebelliously running Stomach channel that traverses the neck and lower back corner of the jaw prevents the normal crossing over of the Large Intestine channel, at the neck and back corner of the jaw, onto the face. The subsequent absence of Large Intestine channel energy over the face, plus the subsequent shortage of energy in the face portion of the Stomach channel (which should, in a healthy person, derive from the terminus of the Large Intestine channel at Yin Tang), serves to create a pocket of current-free, energy-empty flesh in the front of the face. This "Qi free" zone runs from Yin Tang, down either side of the nose, across the cheeks, and down to the jaw.

An area without Qi performs differently from an area with backwards flowing Qi. As noted above, where Qi flows *backwards*, rigidity can result. Where Qi is insufficient, limpness or numbness can result. In Parkinson's, the lack of channel Qi in the portions of the face that *should* be traversed by the Large Intestine and Stomach channels but which are empty in PDers causes the slackness in the muscles of the face, the decreased ability to form a real smile (one that uses the muscles of the cheek), the sagging lower eyelid, sinus problems including sleep apnea, lack of control of the salivary glands, seborrhea alongside the nose, poor circulation in the face, and intermittent pain in the lower back teeth on the side of the body that has the foot injury – a pain for which dentists can find "no reason," a pain that is often experienced by people with

Parkinson's. (One patient said that her lower molar on that side had "no root at all." Her dentist had never seen that before.)

As noted earlier, similar changes from lack of Stomach channel Qi in the toes causes a decrease in circulation and proprioception.

Brain disconnect

Body areas that are influenced by *either* Rebellious Qi or insufficient Qi over a long period of time will have a decrease in the functionality of the nerves that connect these body areas to the brain. Many PDers experienced brief periods of spastic, dyskinetic movement and unfamiliar sensory awareness and proprioception in "returning" body parts during their recovery from Parkinson's. Changes in nerve function occurred during recovery in areas that had been traversed by Rebellious Qi *and* in areas that were deficient in Qi. The recovery symptoms varied, depending on the nature of the Qi problem: areas that had been numb from insufficient Qi became highly sensitive. Areas that had been rigid because of Rebellious Qi became limp. In recovering PDers, these changing conditions, and other changing conditions too numerous to mention in this chapter, painted a clear picture of where Qi had been either absent or Rebellious.

All of these recovery symptoms, which occurred exactly along the lines of the affected channels, suggested that some degree of nerve dormancy *had* been present in those areas which had been long influenced by incorrect Qi flow, whether insufficient *or* Rebellious.

Electrical flow change in the Large Intestine channel

The Large Intestine channel, blocked at the top of the neck where it is supposed to cross the jaw, eventually starts running backwards and discharging out the tip of the index finger. Cell alterations, muscle atrophy and rigidity, and motor, sensory and proprioceptive nerve degradation all along the path of the channel, especially on the side first affected, appear along the course of the Large Intestine channel. When the Large Intestine channel Qi is Rebellious, it seems to cause more atrophy than we see in the Rebellious portions of the Stomach channel. The bicep muscle is nearly gone in most people with Parkinson's disease. Other arm muscles fill in for the bicep, a process called "splinting."

Another muscle that atrophies is the muscle on the back of the hand, the muscle that bridges the distance between the thumb and the index finger (the first dorsal interosseous muscle). This is the muscle against which many writers rest the shaft of their pens. This muscle usually becomes almost completely atrophied in Parkinson's disease. The brain loses a sense of connection with this atrophied area. When this muscle is no longer under conscious control, it passively vibrates in time with the internal tremor. As it vibrates, it moves the somewhat numbed index finger. This muscle's atrophy and its subsequent attunement with the internal tremor cause the "pill rolling" tremor of Parkinson's disease.

This subject is moving us towards the lengthy topic of tremor and this chapter is already growing overlong. The next chapter will continue with the subject of the changes in channel flow that occur in Parkinson's and how they create the symptoms that are known as Parkinson's disease.



“Two wrongs don’t make a right.”

-An old saw

CHAPTER NINE

THE DEVELOPMENT OF PARKINSON’S: CHANGES IN CHANNEL FLOW, PART II

TREMOR

Two major factors are involved in the *earliest* presentation of tremor: the displacement of one brain hemisphere relative to the other, which drives the constant, internal tremor, and atrophy, which determines the *location* of tremor. As Parkinson’s progresses, a slew of other factors appear – many of them mental or emotional, and some of them complications of the electrical and channel changes.

The internal tremor that we have reason to believe is caused by brain hemisphere displacement is the more important factor, and will be discussed first.

Brain hemisphere displacement: driving the internal tremor

The previous chapter mentioned the head-spinning shift that some PDers recalled, a shift that happened long before the appearance of Parkinson’s disease. During recovery, many PDers experienced a reversal of that spinning sensation, after which their ability to tremor utterly, permanently ceased. Based on case histories of PDers who have recovered, we hypothesize the following:

The constant flow of excess Qi in the Gallbladder channel on the foot-injury side of the body causes the brain hemisphere on that side to be slightly askew from its normal orientation relative to the other brain hemisphere. Not that it matters, but our understanding of Qi leads us to suspect that the crown (as opposed to the base) of the tilted hemisphere will have become tipped slightly backwards (towards the back, away from the face), relative to the other hemisphere. This tilt can be explained by the constant excessive current in the Gallbladder channel across the sides of the upper part of the brain, which would tend to push the upper part of the brain towards the back of the head.

At any rate, when the Qi at ST-8 shunts violently into the Gallbladder channel, causing a momentary sensation that was “dizzying”, “spinning,” or “like static on the side of the head,” to mention just a few descriptions, a few PDers have said, in retrospect, that this may have been when they first began to feel a faint, very mild sense of internal agitation. This feeling eventually causes what some PDers refer to as their “internal tremor.”

Then again, many of our patients had no sense that they had any internal tremor until after their visible tremor started up. Some didn’t ever realize that they were trembling internally until the tremor finally ceased; the ensuing internal stillness was utterly unexpected. For example, in the case of young Tim in chapter one, he had no idea that he was trembling internally until his trembling stopped. Only when it stopped did he realize that he had been shaking internally, probably for years.

And yet, sometimes the internal tremor is clearly obvious decades before the Parkinson’s appears: one PDer related to me that her grandmother had often mentioned how she had always

been such a calm baby until the accident during her baptism. During the baptism, the priest's hand slipped and he nearly dropped her. As her head flew towards the floor, he grabbed her by her foot. Her foot bent completely backwards as her body was jerked out of its downward fall by the priest's iron grip on her foot. As she started to scream, the priest dunked her in the water. When she came up out of the water, she was no longer screaming: she was silent.

The grandmother often recounted, "After that day, you were always agitated, always shaky. You couldn't sit still." As an aside, this patient had foot pains and problems by the time she was in her early twenties, and early Parkinson's symptoms before she was thirty, even though she never recalled injuring her foot. Her foot had displaced bones in the center and Rebellious Qi in the Stomach channel. Possibly because her initial scream of pain had been punished with a plunge into the cold font, she had never been able to address and recover from her baptismal foot injury. Although the patient did not recognize that she had an internal tremor until it stopped, she realized in retrospect that her grandmother had picked up on the her internal shakiness.

Some PDers were so used to the constant internal tremor in their head that, when it stopped, they momentarily assumed that they had died.

One PDer described the recovery brain shift in the following way: "a vibration moved through my head, going from left to right. When it was vibrating at the center, I assumed I was going to die, but I decided I was going to be OK with that, and just surrendered to it. But when it was over, I wasn't dead, and the tremor was gone forever."

Other descriptions were similar. Based on these descriptions, and the fact that the tremor comes to an absolute halt afterwards, we suspect that an electrical or material displacement has occurred inside the head in PDers and that this displacement is the source of the electrical chaos that some PDers call their "internal tremor." Western MDs, using equipment to measure the excessive electrical activity in PDers' brains, refer to the location of the internal tremor as the "firestorm."

No doubt, this internal tremor is a perfectly healthy signal: one that an injured person is supposed to heed. A person with healthy sensitivity to his own pain whose head is creating an electrical firestorm will notice that he doesn't feel quite right, and go lie down. He will also notice that there's something wrong with his foot, or notice whatever injury or stress is causing the brain shift. He might ask for help. He might focus his attention on the injury or the stress, and sit with it, massage it, or do whatever it takes until the problem starts to heal or feel better.

Only a very few of our patients with Parkinson's had ever been able to relate to the idea of paying attention to pain and asking for comfort to help them feel safe while processing physical pain. Instead, most PDers deal with pain by dissociating from it; essentially, they pretend that it doesn't even exist. We might assume that, when the internal tremoring started, they might not have been able to feel it. Even if they *had* felt it, they might not have realized that that they should heed its message.

An emotionally healthy person who notices that he has become shaky inside will take some time to figure out what the problem is. A person who has dissociated from his ability to cognize physical pains or injuries may not be able to notice the internal shaking.

A later chapter on re-association gives examples that show how healthy re-association with pain that had been temporarily "put on hold" until a safe time, is perfectly normal and *automatic*. The re-association occurs as soon as the person feels safe and relaxes. However, re-

association with painful events and their subsequent healthy processing and emotional neutralization *cannot* occur if a person has determined that he is *not* going to feel pain.

The idea that an internal tremor may be a healthy, normal signal that reminds a person to relax and deal with whatever caused him pain, and which is still causing him pain, should give comfort to a person with Parkinson's disease: the tremor is not a part of some dopamine pathology.

Further proof that the tremor is not particularly related to tremor comes from the fact that, in many PDers, antiparkinson's medications do not bring relief from tremor even if they enable the PDer to initiate movement. Although emotionally based amplified tremor can be reduced with antiparkinson's medications, the basic internal tremor and the resting tremor are very often not helped by the drugs. (A definition of "amplified tremor" will be provided later in this chapter.)

We can presume from this that the underlying tremor problem has a different origin from the dopamine inhibition and movement inhibition problem. As mentioned already, we propose that the problem that drives the internal tremor is a slight shift in position in one hemisphere of the brain – a shift that is first initiated when Stomach channel Qi first surges into the Gallbladder channel. It is sustained because the Stomach channel continues to short circuit into the Gallbladder channel. We propose that this is a somewhat "correct" occurrence. The body is able to rectify this shift as soon as the foot injury – and/or the emotional injury that is causing a person to dissociate from his foot injury – has been addressed. We have seen that some PDers' foot injuries heal but the tremor continues. In some of these patients, the tremor only ceased when they consciously let go of their emotional rigidity. In other words, the brain might be first induced because of Qi shift related to foot injury. But once that brain holding pattern is established, it may stay in place until the PDer is willing to surrender from his posture of rigid self-control.

Atrophy: determining the location of early stage tremor

Atrophy on the Large Intestine channel

The first presentation of visible tremor is very often on the hand, in the index finger. The decrease in brain-to-muscle neural connections in the first dorsal interosseous muscle (the muscle that bridges the span between the index finger and the thumb) leads to lack of energy and poor motor response in the index finger.

As an aside, early tremor sometimes first appears in other parts of the hand if other channels besides the Stomach channel are running backwards. Also, unhealed injuries in the hand, arm, or shoulder can cause variations on the pill-rolling (index finger) tremor. For example, a not uncommon variation is one in which the middle finger or third and fourth fingers extend straight out and vibrate. When I see this tremor pattern, I usually look for an unhealed injury on the Gallbladder channel, in addition to looking for an unhealed injury on the foot.

Getting back to the decrease in the brain-to-muscle connection: it is caused by the disarray in the Large Intestine channel. As mentioned earlier, this disarray is set in motion when the Large Intestine channel Qi, because of Rebellious Stomach channel Qi, cannot make it past the cross over point on the jaw near ST-6. If the Large Intestine channel Qi is not able to flow up the arm in its correct path, it rebounds down the arm and is discharged, as static, out the

fingertips. Of course, some of the Large Intestine channel's Qi may also shunt into other nearby channels, or create other variations on the correct pattern.

The area on the Large Intestine channel that is near the index finger (acupoint LI-4) is main point where energy flows *into* this channel. If the Large Intestine channel is flowing Rebelliously, energy cannot flow into the Large Intestine channel at the index finger from its source (the Lung channel).

Because of Rebellious Qi in the Large Intestine channel, flow *into* the channel does not occur. Lung channel Qi flows out the fingertips or into other nearby channels instead of flowing into the Large Intestine channel at the point where the thumb meets the index finger (LI-4). When Qi no longer flows through the LI-4 area, the muscles in the area of LI-4 no longer receive any electrical signals from channel Qi. The muscles in this area eventually atrophy.

In a healthy person, the first dorsal interosseous muscle bulges up (on the back of the hand) when a person presses his thumb against the side of his hand, against the side of the 2nd metacarpal bone. In many people with Parkinson's, this muscle area is either a saggy indentation or makes only a feeble bulge when pressed against the side of the hand. In PDers, the skin of this area is very often heavily wrinkled and thin. Because of a lack of Qi, this area has atrophied.

The area between the index finger and the thumb, in the absence of growth-directing electrical Qi signals, can be one of the first areas where the muscle atrophy, loss of Qi, and loss of brain-to-muscle connection become apparent – in the form of tremor. After this area becomes somewhat lifeless, it becomes susceptible to the relentless rhythm of the internal tremor. The index finger, when not being *consciously* moved by the splinting support of the nearby muscles that are still healthy, begins to move in time with the only brain signal that it can get: the signal being given off by the electromagnetic wave of the internal tremor. When the muscle atrophies, the subconscious connection between the brain and the muscle is simultaneously diminished. Therefore, when the brain is not making a conscious effort to move the index finger with an adrenaline override, the nerves to the index finger will be influenced by the brain waves and electrical pulses generated by the internal tremor.

Eventually, as other areas atrophy and lose conscious connections with the brain's motor area, they too begin to tremor. These tremors are called "resting tremors."

This introduction to the two factors in early stage tremor, brain shift and atrophy, will be augmented in later chapters.

Variations on resting tremor

As the symptoms of Parkinson's worsen and the tremor becomes more constant, another factor comes into play: the mental/emotional state. Tremor is highly susceptible to negative emotional states, social stress, and negative thoughts, including self-conscious or self-aware thoughts. All these influences can increase adrenaline levels and therefore decrease dopamine levels. Fear of tremor itself can even contribute to an increase in adrenaline and therefore, an increase in the intensity of tremor.

After a frightening event, animals have a natural inclination towards shakiness. The reason for this will be discussed later. For now, just consider that a person who already has an internal tremor can *amplify* this tremor when he adds a fear-based shakiness to the system.

Because the predominant force behind amplified tremor is emotional, and is very likely related to a long-delayed shock response, a further explanation of anxiety-heightened tremor will be addressed in the chapters that address the emotional aspects of Parkinson's. This chapter is

focusing primarily on those aspects of Parkinson's symptoms, including tremor and certain forms of amplified tremor, that are related to channel disturbances.

Amplified tremor

In amplified tremor, whole limbs may shake. Muscles of the upper arms and hips may fire off in rapid tremor that usually moves at about the same tempo of the resting tremor. The amplified tremor affects a greater number of body parts than the resting tremor. Based on thousands of reports from patients, it seems as if this larger set of tremor-like movements that occur during stress or while eating may actually be caused by *adrenaline* hitting the muscles that no longer have a good connection with the motor neurons in the brain.

To understand the physiology of the larger tremor movements, we need to go back to the discussion of the degeneration in the Large Intestine channel. We've already noted that the muscles in the vicinity of the index finger are not under good brain control. The muscles that underlie the Large Intestine channel farther up the arm are also degenerating or dormant, including in the region of the biceps. We know this because we were able to see, during PDer's recoveries, that the degeneration of muscle and dormancy or nerves in these areas spontaneously, often painfully, regrew and came "back to life." These changes occurred in the muscles along the Large Intestine channel *and* in the proprioceptive and motor nerve connections between the brain and those muscles.

In ordinary circumstances, the muscles further up the arm are less prone to *resting* tremor, although we have seen a few cases in which the remains of the biceps do perform resting tremor.¹

The weighty muscles on either side of the Large Intestine ("LI") channel that are not so much under the influence of the LI channel are not as atrophied, if they are atrophied at all. These muscles help to keep the dormant muscles of the LI channel somewhat under control when a person is "resting" his limbs. Therefore, the larger body parts do not manifest resting tremor even though atrophy is present. However, when increased anxiety sends spurts of adrenaline throughout the body, this adrenaline seems to affect the degenerating muscles of the Large Intestine channel – the muscles that are no longer under conscious control.

Again, the muscles that have been under the influence of Rebellious Qi for decades are no longer under good brain control. In times of stress, when spurts of adrenaline hit these muscles, they may brainlessly fire off with rapid flexions and extensions moving in time with the internal tremor. This can lead to the whole arm or whole leg amplified tremor that is seen during times of stress in advanced Parkinson's. When the large tremor calms down, which is to say, when the adrenaline decreases, the PDer may revert back to his milder, "resting" tremor.

In advanced Parkinson's, when a PDer is in a nearly constant state of anxiety, the flow of adrenaline may be almost constant, and the amplified type of tremoring may be constant as well.

¹ Throughout this book, I could be writing, "however, we have seen people with variations on this symptom." Please understand that I am describing generalities and explaining the pathologies most often seen in people with "classic" Parkinson's. The truth is, almost no one exactly matches the description of "classic" Parkinson's. Because Parkinson's is set in motion by individuals' unique injuries and by unique, mentally created dissociations, no two people with Parkinson's have the exact same symptoms. From here on out, if my description does not exactly match the symptoms of any given PDer, please understand that my real focus is on the principles that allow Parkinson's to develop. If some PDer has symptoms that are slightly different from "classic," he can nevertheless understand the principles behind his pathologies. The medical principles I'm presenting apply to all PDer's, and to all humans.

Amplified tremor while eating

As Parkinson's worsens, an amplification of tremor can occur when a PDer tries to eat. The amplification seems to be caused by the fear that is triggered when the body *tries* to shift into Stomach-dominant parasympathetic mode – the mode in a person should eat, and also the mode in which pain can be felt – and is unable or unwilling to do so. As Parkinson's worsens, PDers become less and less able to access parasympathetic mode.

The internal conflict that arises when a person's body tries to activate his parasympathetic system while simultaneously trying to dissociate from his ability to use the parasympathetic system (the system with which a person is able to feel pain in his body), generates a stress of its own. This stress, in turn increases the flow adrenaline.

In healthy people, eating, in addition to triggering a shift into parasympathetic mode, also causes a concomitant increase in Qi flow in the Stomach channel from the various divergent channels that support the stomach. In a person with Parkinson's, any increase in Stomach channel Qi will only make the Rebellious Qi run more strongly, *worsening* all the symptoms of Parkinson's.

Finally, the dopamine shut down that occurs following an injury is supposed to inhibit appetite. Wholesome appetite is a dopamine-driven function. Following a severe injury or illness, appetite is supposed to be suppressed until the injury or healing is far enough along that channel Qi is once again running correctly and the stomach is once again fully functional. In other words, an emotionally person with an unhealed injury will want to let his body rest and work on healing his injury instead of wolfing down food. As soon as correct Qi flow is restored, hunger will return. So when a PDer tries to eat, he is doing so in spite of *every* electrical system in his body telling him not to eat. Many PDers recall always having a “cast iron stomach” or else “never feeling hunger.” Both of these are indications that normal sensitivity in the stomach itself was inhibited.

However, since most PDers have become numbed to the subtle physical clues of their bodies and have dissociated from the unhealed injury, they 1) don't realize that they have an unhealed injury to fix before they should think about eating; and 2) they are in sympathetic (adrenaline) mode and eating is best done in parasympathetic mode: they need to calm down, slow the heart rate, and really *feel* their stomach. Most PDers can't do this; plus 3) the *stress* inflicted on their system when they eat even though they are in sympathetic mode causes a surge in adrenaline. This adrenaline surge causes those muscles that are not longer under good brain control to move brainlessly, in time with their internal tremor.

Whew. While the MD or curious reader may think that this seems a bit too complicated, many PDers whose Parkinson's has advanced to the point of “increased tremor while eating” have nodded in agreement at the above description and said something along the lines of, “Yes. That is *exactly* what it feels like is happening. And if I get upset about it, it just makes the tremor that much bigger. In fact, sometimes I get the amplified tremor before I even *start* eating. I just know that, as soon as I start to eat, I will tremor. That thought stresses me, and I start the *eating* tremor even before I start eating.”

The subject of tremor will come up again in later chapters.

LOSS OF COORDINATION

Left-Right integration

In a healthy person, when the *right*-side Large Intestine (arm) channel surges slightly, as it does when it triggers the muscle activity in an arm swing, it is followed, a split second later, by corresponding surge in the *left*-sided Stomach (foot) channel. This is due to the Large Intestine channel crossing over to the *opposite side* of the face before it flows into Yin Tang and becomes the Stomach channel. Thus, the current that creates a *right*-side arm swing will, a split second later, create a *left*-side leg stride. And vice versa. These surges of current drive the left-right coordination of arm swing with leg swing.

In a healthy person, when the arm swings *using the bicep*, the surge in LI channel Qi results in a surge in Stomach channel Qi in the opposite side of the body. This surge provides the timing for the movement in the anteriolateral part of the leg (the Stomach channel traverses the leg muscles that are used to move forward and to move to the side). As a result, when one side of the body has an arm swing, the leg on the opposite side of the body is stimulated to move forward a split second later.

This crossover of Qi on the face, in which right-side LI channel Qi flows to the left side of the face before becoming left-side Stomach channel Qi, and vice versa, also helps to regulate the internal left-right brain integration of motor coordination and other balanced aspects of the brain-hemispheres. With a walking or running gait, the right arm should move a split second after the left leg and vice versa, i.e., the normal "crosswalking" movement. *While* walking, other aspects of the left-right brain integration benefit from the right and left alternating surges in Qi. The benefit to the whole body and brain from regular walking – if one has normal Qi patterns – cannot be overstated.

In Parkinson's, since the Qi flow in the Large Intestine channel gets stymied at the jaw, the channel Qi cannot flow to the opposite side of the face. The driving force behind the left-right arm swing integration is unable to manifest. Instead, in PD, the ability to integrate left-right movement gradually deteriorates, especially the integration of arm swinging with the leg stride.

Many PDers who insist that they *can* still swing their arms if they think about it might want to observe themselves "swinging their arms." They will be able to see that they aren't actually using their biceps. They are forcing other arm muscles into play to replicate the normal arm swing that is performed with the biceps. Very often, the arm swing of the PDer is activated by *pushing* the arm forward instead of pulling the arm forward. PDers can do this by using muscles that are on either side of the atrophied biceps, or muscles at the back of the arm and the underside of the arm. Because this is a very unnatural form of arm swing, it can only be maintained as long as a person mentally focuses on performing it.

Balance

In mid-stage and advanced Parkinson's, loss of coordination and balance may become a problem. Most of the balance problem actually comes from the movement inhibition: most subtle balancing movements are lightening fast – a healthy person never even knows that he is making them. When movement imaging ceases to exist, so that a person has to *think* about the thousand subtle balancing movements, there is no way that he can make these correcting movements quickly enough. Instead he falls over or crashes into a wall before he can even begin to *think* of which muscles he must activate to modify his trajectory.

As movement initiation becomes inhibited, the problem only worsens. A person with advanced Parkinson's will have both inhibited movement initiation (slowness) *and* decreasing ability to figure out what he needs to do to maintain his balance. He must "figure out" how to balance himself because of his inability to use the normal method of balancing, a method that involves proprioception and mental imagine. Proprioceptive nerves become increasingly dormant as Parkinson's progresses. PDer's, because they are increasingly locked into sympathetic mode, are increasingly unable to activate the movement imaging part of the brain – a brain function that is activated with dopamine.

Festinating gait

A festinating gait is one in which the PDer has been walking, so that his head and torso have forward momentum, but suddenly, the legs stop taking normal length strides. Instead, the feet revert to small, shuffling steps. This occurs when adrenaline levels start to diminish so that the PDer is unable to focus, with adrenaline, on several things at once. When the PDer, who is in sympathetic mode and unable to release dopamine, is unable to maintain his focus on keeping his legs moving – usually because his thoughts have drifted, however briefly, to some other focus – his forward steps abruptly become quite small. However, his torso is still moving forward. As gravity starts to pull downward on the forward-leaning, still moving torso, the PDer realizes, too late, that he is going to fall on his face. He tries to activate movement in his feet. Momentarily lacking enough adrenaline to "step out," he merely shuffles. As his small, shuffling steps try to keep up with his descending torso, his overall movement gives the impression that he is accelerating both forward and towards the ground. Most PDer's end their festinating by coming to a halt against a wall or a piece of furniture. If they are outside with nothing to crash into, they fall to the ground when their torso is too far forward from their center of gravity. The same movement inhibition that prevents long strides may also prevent the PDer from being able to put his arms out to break the fall. Sometimes, PDer's fall face first with no obvious physical movements occurring that might have helped break the fall.

Careful self-observation will teach the PDer that he tends to festinate when his becomes distracted by any thought other than "move the feet and keep moving the feet." Of course, as movement initiation becomes increasingly difficult, a PDer might not be able to move his feet even if he focuses on them. But in the early stages of Parkinson's, festinating gait is most likely to occur when the PDer becomes distracted from his focus on keeping his body moving.

Stuck foot

Some PDer's have difficulty taking a "first step" when they want to initiate walking. They say that their feet are "stuck" to the floor. Part of this stuck feeling occurs because the Kidney channel is running incorrectly. The Kidney channel originates on the sole of the foot and is supposed to flow up the leg. In some PDer's, when the Stomach channel becomes distorted at the site of the foot injury, some of the Qi flows into the nearby Kidney channel. This increase in Qi in the Kidney channel can cause an obstruction to the flow of the Kidney channel on the sole of the foot. Some PDer's can feel the electromagnetic pull that connects the foot to the floor when the Kidney channel becomes obstructed. This PD symptom is referred to as "foot sticking." This magnetic connection to the floor, combined with the movement initiation inhibition that is inherent when the body is getting a go-to-sleep signal in the brain, can make it very difficult to lift the foot off the floor to initiate walking. As adrenaline levels decline, the PDer has a

decreasing ability to override the body's movement inhibition and electromagnetic pull on the sole of the foot.

Adding to the above one-two punch, a person's Qi system is designed to create an electrical static point on the sole of the foot when a person lapses into automatic dissociation. In the event of severe blood loss or near-death injury, when a person becomes rigid and possibly comatose, the Qi flow in the channels diverts deeply into the interior. Qi flow in the Stomach channel ceases, and Qi flow in the center of the foot moves back and forth instead of flowing into the Kidney channel. This Qi flow pattern causes the toes to tighten. Much more about this will be explained in the upcoming chapter on automatic dissociation.

The Parkinson's gait

The phrase, "Parkinson's gait" can refer to festinating gait, foot sticking, or the tendency, in advanced Parkinson's, to be unable to take a step over a threshold or over changes in flooring patterns.

The latter form of stride inhibition can occur when a PDer must walk over a transition in flooring. The transition might be from carpet to linoleum or from one pattern of parquet to another. This occurs in late stage Parkinson's, and is triggered in a deep fear center of the brain that inhibits movement when the terrain is uncertain. This deep animal fear is that same one that makes painted stripes on the road serve as effective cattle crossings. In advancing Parkinson's, a person can no longer muster the adrenaline to override even these most primitive, animal fears. Very often, PDers use some sort of walking stick to tap the floor on the other side of the threshold or the "different" flooring onto which they must step. When assured by the firm response of the walking stick, they are then able to step into the next room or onto the next type of flooring – if they can muster enough adrenaline to move their feet.

Several of my PD patients have incorrectly referred to their dragging foot or uneven gait as a "Parkinson's gait." This is not correct. What they have is a dragging foot or an uneven gait – traits that are not necessarily related to Parkinson's. When a doctor refers to a Parkinson's gait, he should be referring to either festinating gait, foot sticking, or the halting way in which a person with advanced Parkinson's finds himself unable to cross a threshold or a transition between flooring types.

BILATERAL SYMPTOMS

Although most symptoms of Parkinson's disease begin on the same side of the body that has an unhealed foot injury, the symptoms eventually become somewhat bilateral. This can be explained by basic, high school level electricity principles.

When similar electrical currents run parallel and close enough to each other, they influence each other. For example, in the following diagram, with currents of three types, A, B and C, a change in the top current A will cause a resonant change in the bottom current A. Any change in one of the B-type currents will cause a resonant change in the other, parallel B-type current. This phenomenon can be easily seen in parallel electrical currents in a physics lab.

A _____
B _____
C _____

B _____
A _____

Fig. 5.7
Parallel currents

This resonance of similar, parallel, electrical current is also demonstrated in the Qi flow of living organisms.

Most of the channels in humans are bilaterally symmetrical and changes in one channel can often be felt in the paired channel on the opposite side of the body. This influence that electrical currents exert on each other causes the Qi irregularities from a one-side-only injury to eventually manifest on the other side. The current on the healthy side can exert a healthy, somewhat stabilizing effect on an opposite side current that is running erratically.

In PDers, despite this harmonizing effect of similar, parallel electrical currents, the Qi in the Stomach channel is palpably *more* disrupted on the side where symptoms first appeared. The Qi flow in the Stomach channel on the uninjured side of the body may feel merely deficient or feel as if it alternates rapidly between running backwards and running correctly. Sometimes, a slight stimulation of the *healthier* side via acupuncture or needling with acupuncture *may* cause the Qi to run, briefly, in the correct direction on the healthy side, and only on the healthy side.

A similar stimulation of the injured side can increase the amperage of *backwards* flowing current. It may also cause the Qi of the healthy side to run more vigorously in a backwards direction.¹

The appearance of bilateral symptoms occurs as a result of the electrical tendency for resonance in parallel circuits. The Qi disruption, though remaining worse on the injured side, becomes, over time, somewhat bilateral. The symptoms, correspondingly, also become somewhat bilateral over time, while usually remaining somewhat worse on the side that has the injury.

HYPOTHESES SUMMARY

In summary, the physical disarray in the unhealed injury sets in motion electrical changes that eventually translate into changes in the electrical patterns that traverse the brain. One of these brain alterations, the constant increase in Qi in the Gallbladder channel, serves to electrically put the brain in go-to-sleep mode: a mode that inhibits dopamine release. In the early years, this inhibition can be overridden with adrenaline. When adrenaline levels start to lag, the dopamine inhibition becomes apparent as impaired movement initiation, poor balance and rigidity.

¹ Acupuncturists often treat a person by needling the *uninjured* side of the body. If a body part is too sore or sensitive to be needled, the acupuncturist can access the problem area by needling into the same vicinity as the problem area on the *uninjured* side. Because of the influence of parallel electrical currents, the benefit can nearly as strong as if the injured area had been needled directly. People with Parkinson's should *not* be needled on either side of the body; until the injury has healed, the stimulation will always serve to increase energy in an already backwards system. However, people with Parkinson's may receive needling along the midline of the body, in the Ren and Du channels. However, even this type of needling can stimulate the processes at work in the body. Because the body of a PDer is *correctly* inhibiting dopamine release and motor function due to injury, regular needling even of these channels prior to removal of the injury can cause acceleration of the development of Parkinson's symptoms.

The electrical changes in the channels also set in motion inhibition of muscle and nerve function in those areas of the leg, torso, arm, and face that are under the influence of those aberrant channels. The left-right coordination, including arm swing and stride, that is normally driven by currents crossing from left side to right side, ceases when currents are no longer able to follow their correct paths.

The changes in the currents that traverse the side of the brain is the most likely cause for the brain shift that seems to be at the root of the internal tremor.

The symptoms that have been described up until now in this summary are perfectly normal symptoms of severe injury. These symptoms can help a person sleep or stay relatively motionless while an injury is healing. The brain shift, and the internal tremor that it causes, is a symptoms that can be helpful in reminding a person that he is injured.

However, in Parkinson's disease, these symptoms are not heeded. The PDer uses adrenaline to override his body's many signs and signals that something is amiss. The aberrant Qi flow becomes semi-permanent.

After decades, the muscles that have degenerated from lack of any Qi flow are at the mercy of the internal tremor: the atrophied muscles vibrate in time with the internal tremor: resting tremor.

The muscles and nerves in the areas of the arms, legs, face, and torso that are in the field of influence of Rebellious Qi become rigid and dormant, respectively.

Eventually, because of the brain's extreme plasticity (extreme ability to change) and its "use it or lose it" prioritizing of cell creation and maintenance, anti-dopamine changes occur to reflect the minimal use of dopamine. The slow, steady conversion of dopamine-producing cells into dormant, neutral (re-undifferentiated) cells, and the simultaneous decline in the functionality of dopamine receptors and other dopamine-related chemistries eventually become significant enough that they can be detected (in autopsy). These dopamine-related changes reflect a long-term decline in the use of dopamine but are not the original cause of idiopathic Parkinson's disease.

The original cause of Parkinson's is two-fold. The one of the causes is a perfectly ordinary foot injury. The second part is dissociation from pain to the extent that the injury cannot heal. This combination leads to *permanence* of a perfectly logical variation on the normal channel pattern – a variation that is only supposed to be activated for a short time. This variation should only preside until the injury heals enough for normal Qi flow to resume. In Parkinson's, the injury never heals because of dissociation.



“All happy families are happy in the same way. All unhappy families are unhappy in their own way

Anna Karenina, by Leo Tolstoy

CHAPTER TEN

INTRODUCTION TO DISSOCIATION

The first chapter included passing references to “dissociation” and “mental/emotional blockages.” In order to recover from Parkinson’s, some – though not all – PDers need to consciously rid themselves of their pain-related dissociations. Therefore, the next few chapters will go much deeper into these subjects. This introductory chapter will touch on a wide spectrum of dissociation-related subjects ranging from definitions to our statistical methods.

WHY ARE THE BLOCKAGES BOTH “MENTAL” AND “EMOTIONAL”

“Mental/emotional blockages” are so called because they are *mentally* executed dissociation responses to painful events. These responses are locked in place by the *emotion* of fear. The blockages can technically be referred to as “dissociations.” However, within the field of medicine, the word “dissociation” refers to two very different types of events: one is a conscious or subconscious decision, the other is an automatic, reflexive response. In addition to having a foot injury, PDers have one or the other *or both* of these types of dissociation.

GATHERING STATISTICS: NO TWO PDERS ARE THE SAME

As you may have guessed from the case studies in chapter two, no two of our patients with Parkinson’s had the *exact* same kind of dissociation from the foot injury. Some of them were like Gus, who had merely dissociated from his memory of his war-time foot injury, along with all the other events of that bloody day. Others were more like the frequently abused Lynne, who, at an early age, even before her foot injury, had dissociated from her ability to feel any physical or emotional pain.

There was an enormous range in the degree and breadth of PDers’ dissociation from pain: some people dissociated only if they anticipated severe pain; others dissociated if there was a risk of feeling *any* physical or emotional stimulation. Some people had taught themselves to dissociate from the ability to feel *if* they were in the presence of anyone or anything that possessed qualities that in any way resembled qualities of the person or thing that had first terrorized them; still others had convinced themselves that a heartless dissociation from all physical and emotional sensations was the route to spiritually superiority. Every PDer had created his own rules as to which activities or relationships were potentially pain inducing and which activities or relationships were emotionally “safe.” Because most PDers’ *dissociations* came to the fore or retreated back into their mental dens depending on mood, emotion, and external circumstances, their Parkinson’s symptoms also ebbed and flowed, depending on mood, emotion, and external circumstance. Possibly the strongest examples of these day to day, and even hour to hour, changes in most PDers symptoms are the “safe” activities.

Example of “no two the same”: variations in safe activities

Many case studies are available in the Parkinson’s literature describing PDer’s with one or more “safe” activities. When PDer’s do these safe activities, which might be anything ranging from painting to playing the violin to doing the crossword puzzle, they can initiate movement perfectly *normally*, even if, the rest of the time, their Parkinson’s symptoms are so highly advanced that they are in a wheelchair and/or otherwise unable to care for themselves.

Even our small project had a few PD patients who had safe activities. During these periods of normal movement initiation, they still manifested their purely *physiological* PD impediments such as poor foot circulation and facial numbness. However, like any other person with a few purely *physical* impediments such as a missing leg, they were able to *initiate* and *execute* most movements with normal fluidity and speed. However, as soon as the “safe” activity ended, they immediately reverted back to rigidity and poverty of movement.

Even though these cases, in which a person with very *advanced* PD can still move normally during a safe activity, are somewhat rare, *every* person with idiopathic Parkinson’s disease will confess, if pressed, that his ability to initiate movement or the size of his tremor varies from one day to the next, and even one minute to the next – and that these variations seem to be based on habit, mood, and circumstances: *not* pure physiology.

Even though a majority of our patients consider themselves to be very logical and analytically minded, they have rarely stopped to consider the implications of these movement variations. As one PDer put it, “I’m really mad at my family. They keep pointing out that I can move easily when we’re doing things that I want to do but I can barely move when we’re doing things I don’t want to do. They say there’s a psychological component.”

I replied, “But evidently they’re right, aren’t they?”

She snapped back, “Yes, but I don’t like it when they point it out. It hurts my *pride*.”

“But maybe there’s something psychological that can be rooted out so that you can move normally *all* the time.”

“I don’t want to discuss mental problems. Parkinson’s is a *real* illness.”

(The patient quoted above did talk with her young, well-informed neurologist the next week. She reported back to me that she’s asked him if Parkinson’s had an emotional component. He’d said yes. I asked if she was now willing to work on some of the mental issues. She said no.)

Generating statistics

Because one of the dissociations PDer’s use is *mentally* induced, as demonstrated by the existence of “safe” activities, PDer’s movement and tremor variations are as infinite as the human capacity for thought. Because of the extreme range of variations in our patients, I have been unable to use the statistical conventions so loved by medical researchers. Instead, throughout this book, the words “a few” “some,” “many,” and “most” are used to describe the numbers of people we saw with specific Parkinson’s symptoms or specific recovery symptoms.

Needing a thousand patients

In 1999, a PDer with a degree in medical statistics was frustrated with my lack of precise numbers. He traveled to the United States from his home in Germany to teach me how to create a meaningful assessment of my research. For two weeks, he observed patients at the free clinic, observed the Parkinson’s Treatment Team’s private patients, and was treated himself. At the end of two weeks he announced, “No two of your dozens of patients are anything like each other. They are all going through changes, but no two of them are going through the same sets of

changes. There is no way that you can create any *meaningful* statistics about your program. Your patients all appear to be recovering, but you will probably need to treat more than a thousand people before you can have anything approaching meaningful numbers.”

His words resonated with a warning I had received earlier. When I first started the Little Project, Dr. Fred Jones, our advisor who used to teach medical research, told me, “Parkinson’s is thought of as incurable. Therefore, every single case of a person who recovers will be considered anecdotal, and probably deemed a case of misdiagnosis. You will need to have at least a thousand recoveries before the medical community even considers looking at your work.”

Dr. Jones went on to point out that, in the field of psychology, the single-case study was becoming increasingly acceptable. In recognition of the fact that no two people are psychologically alike, researchers in psychology have always been able to publish and present hypotheses based on what happened with *one* patient. These single-patient reports were increasingly acceptable to researchers in the more “scientific” field of general medicine. Therefore, he encouraged me to write up our findings using examples from single-case studies, while foregoing the statistical side of things until we’d had more than one thousand patients. Because we did eventually find a psychological component to Parkinson’s, Dr. Jones’ advice was particularly appropriate, if not prescient.

He gently encouraged me by adding, “The way I see it, you only have two choices. Either you can keep treating people, and someday folks will say, “There used to be a woman who could cure Parkinson’s disease, but she’s dead now.” Or, you can write up everything you do and everything you know, and try to figure out a way to *explain* what you’re seeing and thinking and what you’re doing so that other people can understand what you’re doing, replicate what you’re doing, and even build on it. And maybe in twenty to a hundred years, the medical community will accept that there is a cure for Parkinson’s disease.”

When he put it like that, I had no choice. With Fred’s admonition in mind, these chapters on dissociation explain what we saw and what we’re now thinking about the psychological component to Parkinson’s disease.

DISSOCIATION FROM THE HEART

I’ve already mentioned that one type of dissociation is a mental decision. This is the type of dissociation that allows a person to mentally not know about his foot or things that happen to his foot, as if his foot does not exist, or he can’t actually feel his foot, or his foot injury hadn’t happened, or if it happened, it didn’t hurt. These are all somewhat normal dissociations.

We slowly figured out, and were eventually able to prove, that a person of great will power and mental focus can perform a different type of dissociation, one that is *highly* specific, in which he dissociates, *not* from the thought or memory of an event, activity, or body part, but from his heart’s *ability* to physically resonate with or register the electromagnetic waves and electrical signals that are created by pain. This type of dissociation might also be described as dissociating from one’s ability to feel pain. Selectively dissociating from the actual physical heart *and* selectively dissociating from pain are actually one and the same thing.

If, in the above paragraph, I had merely written, “Some people dissociate from their heart,” my words might be taken metaphorically; a reader might assume that I was trying to say that some people dissociate from their inner goodness or some such thing. That would be inaccurate. The reason I described this type of dissociation by saying it applies to “the heart’s ability to physically resonate with waves and signals” is because the reader needs to appreciate

that, when I say “heart,” I am talking about the physical organ of the heart and the role it plays in responding to pain signals that come from the brain.

We discovered that this fairly rare type of dissociation, if cultivated and practiced vigorously, can trigger a modified version of an automatic, reflexive neurological state that ordinarily is only activated following a severe loss of blood, severely punctured skin, or a trauma that leaves a person at the edge of death. This well-studied neurological state causes rigidity, difficulty in initiating movement, cold skin and extremities, a shut down of dopamine and a minimizing of adrenaline, and numbness in the heart nerves that communicate sensory responses to the brain. If a person survives the trauma, he may tremor while coming back out of this neurological state. In other words, a body’s condition while in, or while coming out, of this neurological state resembles the conditions that slowly develop in Parkinson’s disease.

Horribly, terribly, confusingly (from a writer’s standpoint), this neurological condition that *usually* occurs reflexively, or you might say “*automatically*,” a condition that in animals and most humans has *nothing* to do with mentally choosing to dissociate from an unpleasant event or memory from normal consciousness, is also called “dissociation.”

To make things easier for the reader and myself, I have named this neurological condition “*automatic* dissociation.”

Now, with this new vocabulary, I repeat: some people can, with one quick mental dissociation from the heart, turn off both the heart’s ability to resonate with sensations and the heart’s ability to transmit information about that resonance to the brain. This dissociation from the heart prevents a person from being able to feel physical pain from injury or the physical pains associated with emotional distress. This *self-induced* numbness to one’s own pain, triggered by dissociation from the heart, can lead to symptoms identical to those of *automatic* dissociation which, in turn, are nearly identical to the symptoms of Parkinson’s disease.¹

Putting it another way, if a person merely dissociated from the *event* of his foot injury, a more common type of dissociation and one that might only prevent him from being able to remember his injury, he might not develop Parkinson’s disease because his body would still be able to know that he had a foot. He might have a limp and a sore foot, even if he couldn’t remember *why* he had a sore foot. His foot would be able to heal even though he didn’t remember the injury event because his foot would still send pain signals to his brain and heart. But if he doesn’t *feel* the foot injury because he’s dissociated from his ability to feel physical pain – a neat trick that requires turning the heart off – he might *never* be able to even know that he has a foot injury. Therefore, he cannot heal from it.

When we finally learned enough to start asking the right questions, we discovered that this terribly strange decision, a decision to rise above pain by the austere method of walling the heart off, killing the heart or rendering the heart unresponsive, or even mentally removing one’s *physical* heart, had been consciously made by many – but not all – of our patients with Parkinson’s.²

¹ Researchers in the relatively new field of neurocardiology have discovered that nerves in the heart, and the electromagnetic heart waves, similar to but much more powerful than brain waves, play an enormous role in sensory perception. The heart, it turns out, is just as much a sensory radio as it is a pump for the blood. More about the findings in this new field that relate directly to Parkinson’s is presented in chapter xxx.

² A few of my PD patients had seen other practitioners of Asian medicine before they came to see me. In two cases, the acupuncturists contacted me, with the patients’ permission, to tell me confidentially that they feared a heart problem was the underlying problem, and *not* Parkinson’s disease (which they assumed was a brain disorder).

By dissociating from their hearts for decades, they had unwittingly set in motion the PD-like symptoms of automatic dissociation: symptoms that exactly resemble those of Parkinson's disease. How true it is, that "truths suppressed lead disconcertingly to a host of errors."¹

Dissociation from the heart, with no foot injury: psychogenic parkinsonism

Before I can actually launch into the discussion of the various types of dissociations that PDers often have, I must discuss a tangentially related illness: psychogenic parkinsonism. This condition, only recently named "psychogenic parkinsonism," sheds light on the role of the mind in creating symptoms of Parkinson's. People are usually diagnosed as having *had* psychogenic parkinsonism only after they recover from it. Psychogenic parkinsonism is diagnosed in people who recover from Parkinson's disease, people who presumably had idiopathic Parkinson's disease.

If a person manifests increasing rigidity, poverty of movement, postural instability and/or tremor, and is therefore diagnosed with Parkinson's, and *if* this person's Parkinson's goes away, his diagnosis will be *retroactively* changed to psychogenic parkinsonism. In many cases of psychogenic parkinsonism, the patient can recall a traumatic event that coincided with the onset of symptoms, *or* (more importantly) an emotional release that coincided with the cessation of symptoms. Therefore, the current assumption is that some emotional shock causes psychogenic parkinsonism to develop; when the patient recovers from the emotional shock, the parkinsonism can go away.

When I first started the Little Project, Parkinson's disease was supposed to be incurable. At that time, doctors still slapped a label of "neurotic" on any person who had symptoms of Parkinson's disease but who eventually recovered. The assumption was that these people had only pretended to have symptoms of Parkinson's for hysterical reasons of their own. Some of my first patients to recover were told, when their symptoms went away, that they were neurotic, or that they had pretended to have Parkinson's disease.

At about the same time that PET and then SPECT scans became available, the diagnosis for people who spontaneously recovered from Parkinson's was changed from "neurotic" or "pretended to have had Parkinson's" to "psychogenic parkinsonism." I have to wonder if this change came about because people whose parkinsonism turned out to be "psychogenic" had brain scans, during their period of parkinsonism, that showed the *same* pattern of diminished dopamine receptor activity that is sometimes seen in the brain scans of people with idiopathic Parkinson's disease. In other words, even if the Parkinson's symptoms cleared up when the patient ceased being emotionally inhibited, the patient *had* been through a period during which dopamine receptor activity had been *measurably* reduced. If there was a measurable cause, then these people had *not* been pretending – they really had experienced a problem that causes symptoms indistinguishable from the symptoms of Parkinson's disease.

However, since these patients had recovered, and since the Current Wisdom was still clinging to the dead dopamine-cell theory and the idea that Parkinson's was inherently incurable, it was wrongly assumed that psychogenic parkinsonism and idiopathic Parkinson's disease could

By using tongue and pulse diagnosis, they had both detected deficient levels of Qi in the hearts of their PD patients, even though, from a mechanical standpoint, the person's heart muscle was working perfectly normally and was perfectly strong. I had to tell these acupuncturists that modern heart research, and our findings as well, showed that heart nerve dormancy was actually a symptom that nearly all PDers have in common – even though every PDer's overall *collection* of symptoms is unique.

¹ Paramahansa Yogananda, in his *Autobiography of a Yogi*.

not have anything in common. The strong emotional component for the former was proposed at that time, and remains the medical community's hypothesized basis for psychogenic parkinsonism.

The Little Project had two patients with psychogenic parkinsonism. Their symptoms and personal stories contributed to our understanding of the dissociation processes at work in people with idiopathic Parkinson's disease. One of these patients, who had no evidence of foot injury and who, when her mother had died, more than fifty years earlier, had made a *conscious* decision to *not* have a heart that could feel emotional pain, had a PET scan that showed diminished dopamine receptor activity. Her symptoms of parkinsonism appeared, full-blown, the day after she was diagnosed with osteoporosis (the illness that she most dreaded). However, she only had symptoms of parkinsonism when she walked or was eating: when she might be in danger of breaking a hip or when she might be in danger of the poor nutrition that she presumed was the cause of osteoporosis. At all other times, she could move perfectly normally. For example, when she was sitting down, she could gesture with her arms while speaking, cross her legs without thinking about it, talk rapidly, and move perfectly normally. She had no stiffness anywhere in her body if she was not in danger of falling down or in danger of eating anything "wrong."

Her neurological had given her a diagnosis of Parkinson's disease. She wanted me to agree with his diagnosis of idiopathic Parkinson's so that I could "fix her." Based on her ability to move normally except when she was worried about breaking her hip or eating wrong food, I could not. Also, she had none of the Qi flow aberrations that I had seen in hundreds of people with Parkinson's disease. She then saw another doctor, who confirmed that she did *not* have idiopathic Parkinson's disease, but seemed to have some sort of mentally-induced immobility during certain activities. Her original neurologist maintained that she had Parkinson's, and pointed at the brain scan to prove it.

At every subsequent visit, when she demanded I diagnose her with idiopathic Parkinson's, I mentioned one small, very specific, "classic" symptom of idiopathic PD that she did *not* have. At the next visit, she would conveniently have the exact symptom that I had mentioned. I would point out another symptom that she did not have. At the following visit, she would have developed the next symptom. This went on for two years. She had no foot injury, and clearly had mind-induced parkinsonism. We learned a lot from her that crossed over to our research on people with idiopathic PD.

The other patient with psychogenic parkinsonism had, at age four, after losing four elderly aunts and uncles to heart attack over the period of less than a year, decided that the only smart thing to do was to not have a heart. After that, she was unable to cry at sad stories and events and unable to be shaken by startling circumstances. She was not worried by these attributes, but other children often accused her of being heartless. She often wondered what led the other children to suspect that she had secretly removed her heart. Her first parkinsonism symptom to appear, when she was seventy years old, was stiffness in her arm, and tremor. She had no history of foot injury. She also said that she knew that she needed to stop pretending she had no heart, but that she was terribly afraid to resume having a heart because of the bottled up pains that she just knew she would feel if she did. Her case, and the preceding case, both helped us learn to ask the questions that finally led us to discover the selective heart dissociation in people with Parkinson's disease.

We'd known since 1998 that everyone we'd seen with Parkinson's disease had an unhealed foot injury and aberrant Qi in the Stomach channel. Over the next ten years, assisted by

our wonderful patients, and even the two patients with psychogenic parkinsonism, we figured out the mental angle that was keeping the foot injury in place.

As an important aside, please note that when people with psychogenic parkinsonism recover, they do *not* experience the changes in blood circulation in the face or foot, the stinging or tingling in long-numbered toes and face, the recovery dyskinesia or any of the other purely physical recovery symptoms that are described in chapters xxx through xxx. Oppositely, our patients with *idiopathic* Parkinson's disease, all of whom had unhealed foot injuries, *did* experience some of these physical changes when their feet start to heal.

Foot injury and the various dissociations: two distinct problems

People with idiopathic Parkinson's disease have a foot injury *and* some collection of dissociations. *All* of our patients with idiopathic Parkinson's disease had dissociated to some degree from their unhealed foot injury. *Some* had dissociated from other body parts, as well. *Some* of them had *also* dissociated from their hearts' ability to recognize vibrations that are triggered by various sensory events such as pain. The latter PDers had this heart dissociation in amounts ranging from "a little, now and then, depending on circumstances," all the way to "I do not have a physical heart, therefore I *never* feel physical or emotional pain."

Based on what we've seen and heard, we hypothesize that people with psychogenic parkinsonism have dissociated from their hearts, but do *not* have an unhealed foot injury. In other words, all our PD patients except the people with psychogenic parkinsonism had at least two *major* problems: 1) an unhealed foot injury *and* 2) one or more types of dissociation.

We came to suspect that, because people with psychogenic parkinsonism have only one of the *major* problems instead of two (they don't have the foot injury problem), they can "spontaneously" recover if they come to terms with their heart dissociation that was induced by a fear of pain: they are not locked into Parkinson's by an unhealed foot injury.

Now that I have introduced the idea that there are two *major* components, a foot injury and one or many dissociations, I can begin to discuss the rates of recovery for people with Parkinson's disease. I introduce this "rate of recovery" idea here because the varying tempos of recovery demonstrate the importance of the dissociations.

Recovery from both Parkinson's problems: the foot injury *and* the collection of dissociations

Easy recovery

Some of our patients recovered fully, within a few weeks or months, after the foot injury started to heal. A selective dissociation had evidently been in place, preventing them from being able to acknowledge or fully heal the foot injury. As we brought their attention to the foot, by using very gentle Yin Tui Na, the foot injury healed, sensory feeling improved in the foot, circulation in the foot improved along with better temperature regulation, and mental awareness of the foot improved. These patients had all had an unhealed foot injury and a dissociation: a mild dissociation from their foot injury.

Recovery with some emotional shifts

Other recovered PDers, as their foot injuries responded to treatment, *automatically* underwent changes in their attitudes towards heart feeling and feeling safe. For example, when

their foot injury began to heal, some of them suddenly realized that it was OK to cry in front of other people. Others were *able* to cry for the first time since early childhood. Some, when the foot injuries began to heal, found themselves undergoing emotional maturation with regard to long-held fears or susceptibility to moods. Many made carefree remarks such as “I know I was late for my appointment today, but what the heck; it’s not like anyone’s going to die.” These remarks, which reflected a feeling of “I’m safe *even if I make social mistakes*,” were stunningly uncharacteristic of their previous personalities, personalities that had been remarkable for their projection of responsibility and “correctness.” Others who were obviously healing from the foot injury nevertheless needed to put in a bit of conscious work to overcome some hesitancy when it came to being aware of and expressing the new feelings emanating from their hearts – including the feeling of having a foot.

These patients had each had an unhealed foot injury and dissociations (plural). Their dissociations included a mild dissociation from their foot injury *plus* mild to moderate levels of dissociation from their hearts.

Recovery with some tough emotional challenges – usually slow

Still other patients with idiopathic Parkinson’s disease manifested symptoms typical of recovery from Parkinson’s when their foot injury began to heal, but then started experiencing intermittent bouts of moderate to severe parkinsonism when confronted with specific situations, activities, or people. Very often, the fear induced by these *lapses* into parkinsonism caused these people to panic and/or rapidly become more wary, and less and less able to ever feel safe. As their ability to feel safe diminished and their emotional wariness rapidly increased, sometimes overnight, their fear of being emotionally susceptible and their fear of not ever recovering from Parkinson’s disease seemed to burgeon, as well. Along with these increases came an increase in rigidity, tremor, and other symptoms of Parkinson’s. These increases came about far faster than the expected development trajectory of Parkinson’s.

These people were only able to bring about a *permanent* recovery from Parkinson’s symptoms by teaching themselves how to feel safe *and* relearning how to feel physical and emotional pain and pleasure. (As many PDers finally came to realize, when you make yourself impervious to the sensations of pain, you risk making yourself impervious to sensations of pleasure, as well.)

Based on their eventual complete recoveries and their collections of recovery symptoms, we were able to make the case that, even though these people *recovered* from their foot-injury induced idiopathic Parkinson’s disease and had the usual physical symptoms of recovery, they also continued to have psychogenic parkinsonism. Only when they did the necessary work to heal emotionally, as well as physically, did they *completely* recover from Parkinson’s.

These patients had each had an unhealed foot injury and dissociations (plural). Their dissociations included dissociation from their foot injury *plus* a *high* degree of dissociation from their hearts.

Review of recovery from both problems

When we consider all the variables that contribute to the eventual manifestations of the conglomeration of symptoms that doctors lump together and call “Parkinson’s disease,” it is no wonder that no two PDers have the exact same collection of symptoms: no two people with Parkinson’s disease *could* have the exact same combination of physical and mental/emotional *causes* for their symptoms.

And yet, we were able to construct two generalities:

1) All our patients with idiopathic Parkinson's disease had an unhealed foot injury. Energy in their leg(s) was palpably running backwards, *up* the anteriolateral side of the leg, instead of making it past the injury site and proceeding gaily to the toes. This backwards-running electrical pattern, which matches the electrical disarray that occurs during severe injury, causes dopamine-inhibiting shifts in the electrical signals in the brain. These electrical signals, over time, cause the physiological symptoms of Parkinson's disease.

2) All patients manifested some degree of mental dissociation. These dissociations could be placed on a spectrum ranging from mild dissociation, having only to do with the foot injury, to extreme dissociation, in which a person had dissociated from his physical heart's ability to resonate with sensory stimuli and convey that resonance to the brain. This latter dissociation triggered symptoms of *automatic* dissociation. Automatic dissociation causes symptoms that exactly resemble those of Parkinson's disease.¹

After we digested the above information, we finally realized this: if a PDer had both a foot injury *and* symptoms of automatic dissociation (from dissociating from his heart), he essentially had two kinds of Parkinson's disease occurring simultaneously. He would have to recover from both if he wanted to completely recover.

I know I am being redundant here, but I need to drive this home: the symptoms of Parkinson's disease can be triggered in either of two ways: severe or long-term *unhealed* injury *or* dissociation from the sensory resonance of the physical heart.

Just over twenty of our more than two hundred patients with symptoms of Parkinson's disease recovered after receiving foot treatments and doing *no* emotional work, so we can assume that they *only* had the foot injury and foot-specific dissociation. Two of our patients had psychogenic parkinsonism and *only* had dissociation from the heart and no foot injury. Most of our patients had both an unhealed injury, dissociation from the foot, and some degree of

¹ These two methods, the foot injury and the mental dissociation from the heart, are not the only ways to produce symptoms of Parkinson's. *Short-term* symptoms identical to the symptoms of Parkinson's disease can be triggered in many ways. For example, a person who accidentally gets locked into a walk-in freezer for a bit too long will come out severely hunched over, shuffling, no arm swing but trembling, with poor balance and reflexes, cold skin and extremities, and an expressionless face. The symptoms go away when he warms back up. A person who for several hours has been running a high fever or suffering severe pain from intestinal troubles may struggle out of bed in order to shuffle slowly into the bathroom, with his body hunched over, no arm swing, no facial expression. If, on top of all that, he's got the hot and cold running chills, he may be trembling. The symptoms go away as soon as the fever drops or the pain goes away. A person can also be temporarily rigid, slow moving, and/or trembling following a severe emotional shock. For some reason, most people with Parkinson's never stop to think that these short-term symptoms that we've all seen or even experienced might just have the same biological root as the symptoms of Parkinson's: dopamine inhibition. Because these are short-term events with an obvious cause, they aren't mysterious or even worrisome. And yet, when these symptoms occur over the long-haul, doctors are mystified and wonder what calamitous events must be taking place. But the cause is just the same for the long-term situation as it is in the short-term situation: dopamine inhibition. The difference is that, in Parkinson's disease, the cause is unremembered because it has usually been in place for a long time *and* the person is mentally holding on to trouble that started the dopamine inhibition in the first place. The symptoms of Parkinson's disease appear when the PDer can no longer summon up the adrenaline to *mask* the symptoms. That's when his perfectly natural dopamine-inhibition starts to become obvious. As for the PDer's decrease in the number of dopamine-producing cells in his midbrain, that is the result of the body's very efficient "use it or lose it" policy. Happily, the body also has an "increase the use and you'll increase the juice" policy.

dissociation from the heart. In this latter group, some of their heart dissociations were mild and intermittent. Some of them had constructed heart dissociations as severe as the heart dissociations in our two patients with psychogenic parkinsonism, in which the patients had decided, astonishingly, to not *have* a physical heart.

TWO TYPES OF “DISSOCIATION RESPONSE”

Up until now, I have touched ever-so-lightly on the subject of there being two types of dissociation: 1) dissociation from awareness of body parts, including awareness of the heart, and 2) automatic dissociation. In order to make more sense of the upcoming discussion of both these types, I need to expand on the fact that “dissociation response” means two different things to two different groups of scientists.¹

Animal behaviorists and some doctors use the term “dissociation” to refer to a group of short-term physiological changes that occur following severe trauma. These changes might even be referred to as “pre-death” changes. As mentioned earlier, these changes include rigidity, difficulty in initiating movement, cold skin and extremities, a shut down of dopamine and a *minimizing* of adrenaline, and numbness in the heart nerves that communicate sensory-resonance responses to the brain. I refer to this process as *automatic* dissociation.

If an animal in a state of automatic dissociation manages to pull through, these changes ebb: the animal reverts to normal physiology.

Psychologists, psychiatrists, and some other doctors use the term “dissociation” to refer to the process of mental compartmentalization or separation that allows certain memories or thoughts to be kept apart from normal consciousness.

Some amount of psychological dissociation is perfectly healthy. For example, if, while reading a book, a person “tunes out” a ringing phone or doesn’t realize how much time has passed, he has “dissociated” from the phone or his awareness of time. The degree to which a person can dissociate is directly related to the degree to which he can maintain focus while “tuning out” distractions. The ability to dissociate is considered innate: we are born with it. But some people can dissociate from distractions better than others. In this book, I will refer to this psychological phenomenon as *selective* dissociation.

I repeat: in order to differentiate between these two very different types, I made up the term “*automatic* dissociation” to refer to physiological shifts that occur automatically following severe trauma or loss of blood. I made up the term “*selective* dissociation” to refer to the

¹ Dissociation is a bothersome word! To chemists, dissociation refers to a specific type of chemical breakup. To sociologists, dissociation refers to a person no longer identifying himself as part of some group. To some spiritualists, dissociation describes the attitude a person should have towards his body in the sense that he does not *identify* himself as his body, but identifies himself as his soul. The spiritual man uses his body and feels his body, and takes reasonably good care of his temporary body. He uses the body as a mechanism for interfacing with and feeling *deeply* the Divine presence in everything via sensory experiences: the wholesome sights, sounds, smells, tastes, and touch sensations of the world. But he is dissociated from these experiences inasmuch as they are not actually happening to *him*, to his soul. His soul is able to enjoy them, via his body, in the way that a person enjoys a movie.

Dissociation has too many meanings. But in the world of medicine, dissociation *is* the correct word to describe *both* of the dissociative processes that can be occurring in people with Parkinson’s.

psychological process of keeping certain thoughts, memories, or awareness separated out from normal consciousness.



“ ‘Playing possum’ is not a strategem but a swoon; ‘the animal can no more stop the involuntary action than a sensitive plant can withhold the folding of its leaves.’ ”

The World of the Opossum, by James F. Keefe

CHAPTER ELEVEN

AUTOMATIC DISSOCIATION

What I refer to as “automatic” dissociation is a recognized set of physiological shifts that can occur in response to severe physical trauma, puncturing of the skin, or a high level of blood loss. When automatic dissociation sets in, the injured person or animal experiences an extreme decrease in heart and breathing rate. His blood moves towards the deep interior of the body, away from the arm and leg muscles and the digestive system. The extremities and skin may even become cold. Midbrain dopamine release is inhibited, preventing easy movement, hunger, or “seeking” behaviors such as curiosity. Adrenaline release is *minimized*: adrenaline levels will be just sufficient to maintain *minimal* breathing and heartbeat. At the same time, endorphins (opiate-like chemicals) are released into the injury area(s). The endorphins block the perception of pain. If a very high level of endorphins is released, the endorphins can get into the general blood supply, whence they can trickle into the brain, causing sedation or even loss of consciousness. If a human experiences some degree of automatic dissociation but does *not* lose consciousness, he may undergo an *alteration* in consciousness: he may feel as if he is outside his body, *observing* his body rather than *feeling* as if he is inside of it.

This altered consciousness is called “depersonalization.” Depersonalization can cause “a sense of detachment from the self. Patients sometimes describe depersonalization as feeling like a robot or watching themselves from the outside. Depersonalization disorder may also involve feelings of numbness or loss of emotional ‘aliveness.’”¹

Depersonalization can, in some cases, make a person feel apart, or different, from others, just as he feels apart from himself. If a person is unable to resonate with his own sensations and his own physiology, he will also be unable to resonate with the underlying “human resonance” of himself or others. Lacking awareness of this resonance, he will tend to *evaluate* others critically, based on their variations in actions and words – their differences. If he could feel his heart, he would be able to wordlessly *resonate* with mutually similar, vibratory heart patterns and characteristically human electromagnetic fields – and thus appreciate others as being basically similar to himself.²

¹ *Health A to Z*; from website www.healthatoz.com, date accessed 1-14-8, derived from *Gale Encyclopedia of Medicine*; essay author Frey RJ PhD; Dec, 2002.

² Curiously, most of our PD patients have been insistent that they are different from all the cases I have written up. To our eyes, and in the eyes of their spouses, they often conform quite nicely with many of the case studies and with the generalities that I make about people with Parkinson’s disease. However, many PDers grasp at small differences, or aggrandize their bits of uniqueness to such an extent that we were, at first, baffled. It was only when we learned that depersonalization *makes* a person view his relationship with others analytically, even critically, by examining *differences* instead of being able to enjoy and resonate with *commonalities*, that we began to understand. What had seemed like compulsive nit-picking was actually part of the depersonalization that occurs with dissociation.

During an automatic dissociation response, the heart's role in sensory interpretation is temporarily shut down: the heart nerves that tell the brain *which* neurotransmitters to release and *how much of each* become temporarily inhibited. In other words, the nerve communication between the heart and brain that ordinarily determines the heart rate and the breath volume, and all other sensory-resonance heart-brain communications, are temporarily suspended. In this respect as well, the short-term physiological shifts of the automatic dissociation response resemble the long-term heart-nerve changes that are *only* seen in people with Parkinson's disease.

In this neurological mode, the chest moves only as much as is needed for breathing: the chest area is unable to expand with pleasure or contract with displeasure or fear in response to sensations in the body or in the environment.

Like Parkinson's disease, automatic dissociation symptoms can range from mild to severe. The symptoms of *mild* automatic dissociation resemble the symptoms of *early- to mid-stage* Parkinson's disease. The symptoms of *severe* automatic dissociation create a condition in which a living animal appears to be dead because he's encased in a rigid, seemingly dead body – very like some people with *late-stage* Parkinson's disease.

Is automatic dissociation a variation of the sympathetic nervous system response?

A sympathetic response (as opposed to a parasympathetic response) swings the body over to an adrenaline-dominant, dopamine-reduced condition. Because automatic dissociation also causes an adrenaline-dominant, dopamine-inhibited state, it might be classified as a variation on the sympathetic nervous system response. On the other hand, enough differences exist that the automatic dissociation response very possibly should be in its own category. Even so, it can be helpful to compare the automatic dissociation response with the sympathetic response.

When most people hear “sympathetic” or “adrenaline-type” response, they think “fight or flight.” This is gross oversimplification. The adrenaline-dominant response actually can take many forms, including the relatively rare “fight,” or “flight,” the more common “freeze,” and, the most common of all, “wariness.” These responses to danger or challenge feature varying amounts of increase in adrenaline, a corresponding *decrease* in dopamine, and a corresponding *increase* in heart rate, bronchial dilation, alertness, and blood supply to the muscles of the arms and legs.

As noted earlier, the automatic dissociation response – even though it is a type of adrenaline-dominant response inasmuch as adrenaline levels are higher than dopamine levels – causes a *decrease* in heart rate, breathing, alertness, and blood supply to the muscles of the arms and legs. This is because, in automatic dissociation, adrenaline is released at very *low* levels, not high levels.¹

¹ Actually, the sympathetic/parasympathetic classifications need an overhaul. These two modes only define the body's behaviors during times of alertness. When sleeping, a different mode kicks in – a mode similar to the automatic dissociation mode. More than a century ago, doctors decided that anytime a person wasn't in sympathetic mode, he must automatically be in parasympathetic mode. How simple. However, parasympathetic mode is usually understood to be the mode in which a person is relaxed and his heart and brain are oriented towards the vagus nerve – the nerve that activates the stomach, intestines, and all the organs that run in tandem with the gastro-intestinal tract. In the 1960s, when I was in high school, we were taught that we could think of parasympathetic mode as the “cud-chewing mode.”

During sleep, as during automatic dissociation, the vagus nerve is somewhat inhibited (we don't have bowel movements in our sleep, nor do we digest food well when we are sleeping), consciousness is altered, heart

Dissociation in prey animals

In nature, dissociation usually occurs when a predator's teeth or claws have punctured or severely cut the skin of his prey. The dissociation response is beneficial to the prey animal in the following ways: because heart rate is slowed and blood is shunted to the interior of the body, bleeding from the torn flesh is minimized. More importantly, the endorphin-induced sedation, the severely slowed heart rate, and the slow breathing of the prey animal can cause it to become cold and rigid. To a casual observer, the animal will look as if he is dead.

The supreme benefit of the dissociation response is this: if the predator was catching the prey for sport instead of for food, he may very likely lose interest in his seemingly dead prey. He may drop his prey and go in search of livelier fun elsewhere. Several minutes to an hour later, the seemingly dead animal will return to an alert state and make his getaway.

Many of us have seen a cat drop a mouse when the mouse lapses into a deathlike rigidity. The cat may make a few swats at the mouse to see if he can stimulate the mouse to movement. However, if the mouse remains cold and motionless, the cat often loses interest and moves on.¹

rate and breathing slow down, and blood flow is shifted away from the muscles of the arms and legs – a combination of conditions that does not match the parasympathetic *or* sympathetic modes. The outdated classification system leaves us no way to easily refer to the physiological shifts that take place during sleep – or during automatic dissociation. If we use the existing system, we are reduced to describing all physiological responses in terms of “happy or hungry” *or* “fight or flight.” Simple, but incorrect.

¹ Children who are regularly abused sometimes teach themselves to consciously activate an automatic-type dissociation response when the abuse is about to begin, for the same reason: an enraged or sadistic attacker sometimes redirects his energy or his energy becomes diminished when his “prey” is unresponsive.

When abused children intentionally dissociate from their ability to feel *anything*, opiate-like chemicals (endorphins) are released and numbness pervades the body. Then, if the child bothers to observe the abuse from his depersonalized perspective *outside* of his body, it seems as if the abuse is happening to his numbed physical form – but not to *him*.

As noted earlier, in humans, dissociation from the heart can allow a person to feel no pain and to perceive himself as apart from the body that is being hurt, as if he is watching the event from outside his actual body. While not *all* PDers have taught themselves, via dissociation, to maintain this type of outsider's perspective, many of our PD patients did maintain this outside-the-body perspective almost continuously.

As an illustrative aside, some people who have learned, often during abuse, the pain-numbing benefits of the dissociation response may later perform self-mutilations such as cutting or burning to trigger the pain-deadening rush of opioid-like chemicals, endorphins, that naturally occur when the skin is perforated. Children who frequently engage in this type of dissociative response can develop significant brain changes – presumably from regularly flooding their brains with endorphins (or, more exactly, the *dopamine* that is released in *response* to endorphins). In brain SPECT scans, the brain changes – patchy areas of decreased levels of cellular activity in the *frontal* lobe (not the midbrain) – that occur in children who frequently dissociate resemble the brain changes seen in people with frequent use of powerful dopamine-enhancing drugs such as methamphetamine. It is presumed that these children's brain changes are due to frequent periods of high levels of endorphin-triggered dopamine that is released during their dissociation responses.

Sometimes, during an extreme dissociative response, even if the dissociative response was self-induced via superficial slashing that does *not* cause much blood loss, *if* the endorphins are released at high enough levels, a person can have a response similar to a heroin *overdose*: coma and even stopping of the heart. To rapidly reverse the life-threatening heart stoppage or coma, the hospital's emergency room can treat a person in this condition by administering the anti-opiate drug noxalone. For more information on this subject, see: *The Boy Who Was Raised As A Dog And Other Stories From A Child Psychiatrist's Notebook*, by Bruce Perry, MD, PhD, Basic Books, New York, 2006, p. 189.

How does this relate to Parkinson's disease? Understanding that mental control over dopamine release *and* dopamine inhibition is an innate ability and not a reflection of pathological genetics or illness can help the PDer who had been emotionally sandbagged by the idea that Parkinson's occurs when an unknown “something” kills off dopamine cells for “no reason,” or as many PDers have bitterly described it: “My body is betraying me.” PDers

Dopamine inhibition and turning dopamine back on

Following automatic dissociation, the “safe feeling” must occur in order for dopamine release in the substantia area of the midbrain to resume.¹ We’ve been able to determine that, in humans, the “safe feeling” can best be described as the physical sensation of expansion that occurs in the chest when the sensory cues for danger have ceased. Perception of the safe feeling leads directly to the release of substantia (midbrain) dopamine. Release of substantia dopamine initiates “seeking behaviors:” curiosity, interest in food, playfulness, uninhibited self-expression. Indulging in these behaviors leads to the release of yet more dopamine in the ventral striatum, which induces still more positive, safe feelings, thus activating a positive feedback loop.²

Seeking behaviors need to be inhibited during times of 1) potential danger, 2) trauma-induced dissociation, and 3) during times of initial healing from severe injury. Therefore, because *dopamine* is the neurotransmitter that initiates seeking behaviors, dopamine release is *inhibited* during 1) a strong sympathetic (danger) system response, 2) an automatic dissociation response, and 3) the initial phase of shock and healing that follows a severe injury.

may be uplifted by learning that altered dopamine-cell function can be set in motion by something curable: either an unhealed foot injury or a long-term, fear-based mindset; a *dopamine-inhibiting*, automatic-dissociation mindset.

¹ *In Search of Memory: The Emergence of a New Science*, by Erik R. Kandel, Nobel prize winner; p. 350. Research linking the safe feeling and the release of midbrain dopamine used mice that had received Pavlovian training so that they “felt safe” at the sound of a specific tone. When they heard this tone, a surge of activity occurred in the substantia: they subsequently moved more freely, and over a larger area. Prior to this research, no one knew exactly what the stimulus was for the initial surge of activity in the substantia that then led to seeking behaviors. Chapter xxx goes into great details on this highly significant research. The safe feeling has nothing to do with logic or thoughts: mice do not consider whether or not they might reasonably deem themselves to be in a safe situation. In order to help PDerS who were locked into mindsets that couldn’t imagine feeling safe, we had to figure out the *biological* events that occur in a mouse when he is “safe”: the events that, through Pavlovian training, are activated automatically when the mouse hears the “safe” sound. Because most of our PD patients had no idea what was even meant by the phrase “feel safe,” part of our research was directed at figuring out how to *describe* the physical processes and sensations that occur when a healthy person feels safe, so that the PDer could learn to produce them.

² Research and general news articles supporting the finding that dopamine is the “reward” for indulging in seeking behaviors – behaviors that can be performed after the initial release of dopamine – are easy to find. While writing this up, I looked for an additional citation to support this relatively new finding, and within minutes I had grabbed one off the Internet, titled “Brain Scientists Discover Why Adventure Feels Good.” This article referenced a study by Wittman and Daw, of the Wellcome Trust Centre for Neuroimaging at University College in London, that had been published in the online journal *Neuron*. The study drew the conclusion that “Seeking new and unfamiliar experiences is a fundamental behavioral tendency in humans and animals [because of the reward of additional dopamine that is released when doing something new or unfamiliar].” (Continued on next page.)

For our purposes, we might add a correcting modifier: “*If a person feels safe*, then seeking new and unfamiliar experiences is a fundamental tendency, etc.” Oppositely, one of the manifestations of the harm avoidance that is a prime component of the Parkinson’s personality is the increasing reluctance to engage in new or unfamiliar experiences.

The next article I pulled up, “What’s in a Smile? Maternal Brain Responses to Infant Facial Cues”; Strathearn, Li, Fonagy, Montague; *Pediatrics*; Vol. 122. No. 1, July 2008, pp. 40-51, stated that new research shows that parents’ midbrains release dopamine when their babies smile at them. The reward of dopamine release makes new parents become “addicted” to making the child smile. This article suggested that parental bonding and wanting to keep the child happy is driven by the dopamine reward. In terms of animal behavior, trying to stimulate a baby’s ever-new smile is a type of “seeking behavior.”

These examples of dopamine-inhibiting conditions demonstrate how *inhibition* of movement and seeking behaviors via inhibition of dopamine *can* be a correct, naturally occurring event.

Relating this information back to Parkinson's disease, PDers sometimes exhibit all three of the above dopamine-inhibiting conditions: 1) a perpetual sense of wariness or an inability to feel safe, 2) varying degrees of automatic dissociation brought on by selective dissociation from the heart's ability to resonate with physical or emotional pain, and 3) an unhealed injury that, because of its location at the terminus of the Stomach channel, causes the type of electrical disarray that is usually activated by *severe* injury.¹

Turning dopamine back on: feeling safe

Most of the PD patients that we worked with almost never felt safe. They had taught themselves to be perpetually wary. Many PDers were proud of their constant wariness, considering it to be a sign of high intellect. Some of our PD patients felt negatively towards the very idea of "feeling safe." Some insisted that only an idiot could ever feel safe.

To help our patients who got stuck in partial recovery who were, for the most part, deeply confused by the concept of "feeling safe," we also tried to define "feeling safe" more technically: we told them that the feeling was "a subtle sensation of physical expansion in the chest, like the feeling of chest expansion that occurs when you perceive something of great beauty or grace." Many patients claimed to have no memory of ever having experienced a sensation of expansion in the chest in response to relaxation, or to something beautiful or stirring, or to feeling safe, or in response to anything. Others thought that they *might* have felt something along those lines, years or decades earlier, but they had no idea how to recapture the feeling. Many had no interest in trying to feel safe, even if it meant a release of dopamine. One PDer expressed it this way: "I'd be lying if I made myself *feel* safe when the truth is, no one is actually safe."

When we discussed the importance of feeling safe with PDers, many of them protested, saying something along the lines of: "The whole purpose of life is to protect yourself from strong emotions. If a person feels safe, he might let his guard down."

Then again, a few insisted that they always felt perfectly safe. They admitted that their tremor and other symptoms were worse in certain situations, but the worsening of symptoms had nothing to do with being *unsafe*. I asked them *why* their symptoms worsened in certain situations if fear and/or not being safe weren't involved. Their reasons ranged from "Social stress" to "I have no idea." They often added statements such as "Worry isn't related to fear or safety," and "Social stress has nothing to do with fear." Some of these people refused to admit that worry or stress had *any* relationship with fear or not feeling safe. Many felt very strongly on this point, explaining to me that they *did* feel safe; that they had built their entire life around the idea of keeping themselves safe. A few went so far as to say that *everything* they did was an intentional effort to stay safe. I tried to suggest that a person who actually felt safe wouldn't have to make a constant effort to feel safe, and that a person's constant efforts to make everything safe might be

¹ An aside: I do realize that this chapter is about automatic dissociation, and not about injury. I included the fact that injury can also cause dopamine inhibition because the same benefits of dopamine inhibition apply in the case of injury. I am redundantly including the fact of dopamine inhibition during injury in this section on automatic dissociation because it helps make the larger point: dopamine inhibition can be a perfectly natural, beneficial response to certain situations. For the same reason, I have introduced the idea that dopamine may be inhibited when danger looms. An editor was concerned that, by introducing the subjects of injury and danger into this already meandering chapter, I might confuse the reader. This footnote is an attempt to clarify.

considered *proof* that he didn't yet feel safe. These patients were usually unable to comprehend the point I was making.

But a large majority of PDers, after reading the earlier editions of this book, volunteered that they did tend towards constant wariness, overthinking, and/or fear of letting their guard down.

It wasn't until 2008 that we realized that many PDers did not understand the literal meanings of words and phrases such as "feeling" or "physical sensation of expansion in the chest," let alone "feeling safe." They had spent so much of their lives in a state of altered consciousness, in which they perceived themselves as being outside their own body, observing their body but not feeling it, or feeling only those strong signals of pain that made it past their mental blockages, that they only accepted metaphoric meanings for literal phrases such as "feel your heart," "open your heart," and for other sensation-related words and phrases. Many of them truly thought that "feeling safe" meant "thinking about how to protect oneself from risk or criticism." They had no idea that feeling safe could include an actual feeling, a physical sensation, a relaxation or expansion of the chest.

Actually, a few PDers' symptoms instantly worsened for up to two minutes when I slipped any form of the word "feeling" into a sentence, even in an innocuous usage such as "I *feel* like the weather should be nice by the afternoon."

Many of our patients had selectively dissociated from their ability to feel, period, and thus had dissociated from their ability to *feel safe*. Because they could not feel safe, they could not activate the surge of midbrain dopamine that should kick in when trauma, injury or immanent danger is *over*. They remained locked into wariness – an attitude that triggers the sympathetic nervous system and inhibits the parasympathetic system: a neurological posture that *also* increases adrenaline and inhibits dopamine! Yet another dopamine-inhibiting behavior!

But I am getting ahead of myself. This section was merely to make the point that dopamine inhibition can be a perfectly normal function *and* the inhibition ends when a person feels safe. In a physically and emotionally healthy person, mentally induced dopamine inhibition can be an appropriate, healthy *short-term* event in response to a fleeting fear, danger, or trauma. We saw that, in many people with Parkinson's disease, it had become a lifestyle.

What dopamine actually does, and how this relates to automatic dissociation

Because many of my patients with Parkinson's are fascinated with the subject of their so-called dopamine insufficiency, are somewhat well read on the subject of dopamine, and drill me with all sorts of questions about dopamine, this section will explain a little more about other roles of this major neurotransmitter. To fully appreciate the automatic dissociation response, during which dopamine in the substantia is inhibited, it can be helpful to know what dopamine does when it's not being inhibited.

Dopamine is a neurotransmitter that doctors have, at one time or another, defined as being the neurotransmitter of sleep, waking, relaxation, alertness, pleasure, addiction, pain relief, muscle movement, joy, parasympathetic system-based imagining of movement that *leads* to motor initiation, and now, most recently, "seeking behaviors." Some of these ideas were just plain wrong. Some of them were only partly wrong. Also, many doctors are ignorant of the fact that dopamine behaves differently in different parts of the body, being a stimulator of nerve transmission in some areas and an inhibitor of nerve transmission in other areas. Many are also

ignorant of the proven fact that thoughts, as well as chemistry, can release dopamine and turn on or off the different types of dopamine *receptors*.

Today, most school children correctly learn in health class that excess dopamine is related to the addiction process. From what I can tell, many newspaper science writers have learned that dopamine is the neurotransmitter of happiness: a *mood* chemical. But they also repeat the cant that the death of dopamine-building cells causes idiopathic Parkinson's disease, which was found to be *not* the case at the turn of the twenty-first century, nearly a decade ago. And from what I've learned from patients and various other sources, most MDs who were educated prior to 1995 still think that midbrain dopamine initiates sleep and muscle relaxation – two of the proposed dopamine roles that have turned out to be completely wrong. So, purely for those who want more information about dopamine, and its *necessary* inhibition during times of danger, automatic dissociation, or severe injury, here is the latest.

Dopamine in different places

In and around the spine, dopamine *inhibits* the transmission of nerve signals. In the midbrain, dopamine *stimulates* neural signals. (A nerve inside the brain is called a “neuron.”)

Dopamine in the substantia, in the deep inner core of the brain, known as the midbrain, activates seeking behaviors *if* the animal is awake and *if* the animal is in the parasympathetic (joyful and/or content) mode. Seeking behaviors include the uninhibited, self-expressive *movement* that PDer's cannot perform (except during safe activities). The neurotransmitter that actually activates muscles is acetylcholine; dopamine allows activity in the *imaging* area of the brain, which, if approved (because one is safe to self-express), then *leads* to the initiation of motor function.

Dopamine's role in feeling good

Dopamine in the midbrain also contributes to a feeling of well-being. In this capacity, dopamine is exquisitely regulated so that a person never has too much. If a person felt too good, he might not bother to inhale or he might jump off a cliff just for fun. (People have been known to do both these things while under the influence of dopamine-enhancing drugs.) Dopamine-enhancing drugs include methamphetamine, cocaine, opiates, alcohol, and nicotine, as well as the dopamine-enhancing products produced by the pharmaceutical industry. Because of the real risk of sudden, carefree, *willing* death if dopamine levels are ever too high in the consciousness and the fear-inhibiting parts of the brain, the body will instantly institute a decrease in the brain's dopamine receptivity if a person, even briefly, experiences an excessive level of dopamine in these areas. After an event in which dopamine levels are deemed excessive, the brain may wait for up to ten weeks before it will once again release or be receptive to enough dopamine to provide for a minimal level of the feeling of “well-being.” The process by which the brain reduces dopamine receptor activity or dopamine release in response to even a momentary excess of dopamine is called “addiction.”¹

During an automatic dissociation response, the body inhibits the release of the *midbrain* dopamine that is used to initiate seeking behaviors. *Imagination* and/or *visualization* of motor

¹ More details on the addiction process are available in *Medications of Parkinson's or Once Upon A Pill*, available for free download at www.pdrecovery.org.

initiation are among the midbrain-initiated processes that are inhibited during automatic dissociation.

An aside: inhibition of visualization during automatic dissociation

Inhibition of the *thoughts* of movement and the mental *images* of movement that precede parasympathetic motor function is important during automatic dissociation. For example, when a mother bear is contemplating taking another swat at your seemingly lifeless body, or when the enemy soldiers are giving “just in case” bayonet jabs to any almost dead soldiers on the field who are still moving in the slightest, you don’t want to be able to even *think* about moving, or even *visualize* movement.

As an aside, we were to discover that most of our patients who got stuck in partial recovery from Parkinson’s disease were either *unable* to visualize themselves moving, or they felt uneasy when doing so. Only while they were consciously forcing themselves to work at it could they imagine themselves moving. If they were in my office, and if I was standing right there, encouraging them to practice the hated imagining exercise, and if they managed to visualize themselves moving, then they could move effortlessly. They *loved* the feeling of “weightless” limbs and effortless movement. However, this pleasure and ease of movement was never enough of an inducement for them to work at learning to *maintain* the highly unpleasant process of forcing themselves to visualize movement. They didn’t like to visualize themselves moving: it didn’t feel *safe*.

Dopamine’s role in pain relief and altered consciousness

Getting back to the subject of what dopamine actually does, consider its role in preventing pain. During automatic dissociation due to severe injury, *location-specific* opioid-like endorphins (molecules similar to morphine or the other opiates) are released in the vicinity of the injury. Endorphins then cause *localized* (point of injury) dopamine release. (Opiates also work by causing dopamine release.) The endorphins cause the release of pain-relieving dopamine in very specific, pain-numbing, parts of the body (usually at pre- and post-synaptic spinal nerve junctions). At very high levels of endorphin release, endorphins and dopamine can both overflow the localized pain management areas and get into the bloodstream.

Endorphins in the bloodstream can get into the brain. But dopamine in the bloodstream cannot pass through the blood brain barrier: dopamine cannot pass into the brain from the bloodstream. This is a safety mechanism that serves to prevent excess dopamine appearing in the midbrain.

When endorphins can get into the brain, they flow to the areas where endorphins have receptors. Endorphin receptors are found in brain areas that influence mood and certain aspects of consciousness, but not in the *substantia*. When the endorphins “hook up” at their receptors, they cause the release of dopamine in those specific areas.

Endorphins in the brain cause dopamine release in the areas that regulate mood and consciousness. In the *substantia*, no endorphins get in and no dopamine release will be triggered. Thus an injured animal can have a dopamine release in the brain that causes heavy sedation or coma. *And* he will have the pain-blocking benefits of dopamine on the spinal nerves. *But* he will not have the dopamine-induced hunger, curiosity, or imagining of muscle function that leads to movement. The *substantia*-area dopamine, which triggers seeking behaviors, is only released

when an animal feels safe – a signal that comes from the heart after the brain stops sending signals of danger.

The high level of dopamine release in the consciousness area of the brain is not without risk: as footnoted earlier, brain scans of abused children who use dissociation to deal with frequent assault show that the frequent instances of unnaturally high levels of dopamine in the consciousness-focusing frontal lobe of the brain set in motion brain changes that are similar to the changes seen in the brains of addicts who frequently use strong dopamine-enhancing drugs such as methamphetamine. However, as a once-in-a-great-while method for escaping from imminent death, the automatic dissociation mechanism has definite benefits.

In times of near-death injury, a person who has automatically dissociated can have the benefit of localized dopamine-based pain relief and even the ability to appear as if dead, while *inhibiting* the potentially dangerous mid-brain dopamine-based behaviors such as hunger, curiosity, or self-expression. In other words, during trauma, dopamine is still being used, but its use is limited to certain highly specific areas and functions: local pain relief and alteration of consciousness in the brain. During these traumatic times, dopamine release and receptor activity for *other* dopamine-based functions such as imaging movement, hunger, and curiosity are inhibited.¹

Three situations that can trigger dopamine inhibition

During automatic dissociation, dopamine inhibition keeps the severely injured animal from making any incautious moves. Dopamine inhibition can also be beneficial during times of severe danger, as in the example of frequently abused children: a person who appears lifeless may be less likely to excite the interest of a predator.

¹ The mechanism for pain relief from opium-derived drugs and from internally produced endorphins is this: the endorphins latch onto endorphin-specific receptors on the nerves that carry pain messages from the body to the brain. When these receptors are activated (hooked up to an opiate or an endorphin), the release of GABA is inhibited. GABA is a regulatory neurotransmitter. When GABA is inhibited, dopamine release in the area of the endorphin receptors becomes uninhibited. This means that, when the endorphin receptors are activated by either endorphins or opiates, dopamine floods the area, hooking up to the nerves. When dopamine attaches to the nerves that carry pain signals to the brain, the pain signal is blocked.

This pain blocking is automatic and occurs when endorphins activate the area. “Runner’s high,” the rush of well being that floods the body of a person who overexerts himself in sports, is thought to be set in motion by the release of endorphins. Sometimes this flood of good feeling is called an “endorphin high.” Whatever it’s called, the sense of well-being is actually caused by the flood of dopamine that results from the surge of endorphin activity. In nature, the mild sedation of consciousness induced by intense exertion, the “runner’s high,” is probably the mechanism that tells the animal that he has run enough and he can now *stop* running. We can hypothesize that, when he’s run long enough, the animal feels calmed by the endorphin “high”: he feels *safe*. Unless danger is still imminent, in which case the animal’s adrenaline will override the endorphin-induced feelings, the “runner’s high” will cause him to relax, to stop running. Animals cannot “decide” to stop running, nor do they use logic: they rely on innate mechanisms to determine whether situations are “safe” or “not safe,” or if enough running has been done. The “runner’s high” may serve animals by telling them that it is now time to stop running.

Interestingly, prior to manifesting obvious symptoms of Parkinson’s, some PDers have learned that, by overexerting themselves into an endorphin rush and its accompanying safe feeling, they can obtain a moderate level of easy motor function for a short while: but the easy movement only lasts until the endorphin rush wears off. This method of staging a false metabolic surge in order to stimulate endorphins in order to stimulate a short burst of safe feeling followed by a short burst of midbrain dopamine is not actually a healthy mechanism. Over the long run, it may reinforce the habit of relying on overexertion in order to move: just the opposite of learning how to feel safe regardless of circumstances.

Also, during the initial healing stages of a severe injury, midbrain dopamine is electrically inhibited so that the injured person will sleep a lot, without much movement, and will not even notice sensations such as hunger. This electrical inhibition of midbrain dopamine allows the body to focus its energy on mending and rebuilding.

Again, although this section was primarily about automatic dissociation, I want to drive home the larger point that midbrain dopamine-release inhibition is a *healthy*, automatic body function during times of danger, trauma, or severe injury.¹



¹ The following is just an interesting aside. There are many types of dopamine receptors, and depending on mood and levels of alertness, these dopamine receptors activate various mental and physical behaviors. These types of receptors are named, cleverly, D1, D2, D3, D4, etc. I find it even more interesting that, according to cutting edge research, *thoughts* play a role in determining which dopamine receptors will be active at any given moment. For example, if a person *thinks* about getting up off the sofa, dopamine receptors in the activity-imaging area are opened up and ready for dopamine. As “getting up off the sofa” is imaged, neurons in this section activate the muscles necessary to perform the imaged activity. (The word “image” is *not* a misspelling of the word “imagine.” A person *imagines*, and his brain imaging area processes the *imagining*.)

Pathologies of receptor activity can occur. These errors allow dopamine to hook up to the wrong receptors at the wrong times. Sleep-walking is an example of what happens if motor-function-imaging dopamine receptors are receptive during sleep – a time when these receptors are supposed to be off and dopamine release is supposed to be minimal – if the person happens to dream about or imagine walking.

Although the idea that thoughts can determine chemical function may seem like science fiction and many neurologists are blissfully unaware that this concept has been proved, scientific research over the last decade increasingly backs up this new (and ancient) idea. In Dr. Candace Pert’s *Molecules of Emotion*, she explains some turn of the twenty-first century research that proved neurotransmitters and other chemicals throughout the body change shape and change function in response to thoughts. This shouldn’t actually be so surprising; *electrical* forces create the bonds that determine the *configurations* of atoms in the smaller molecules in the body. Brain waves and the much stronger waves generated by the heart create electromagnetic fields that must necessarily exert influence over these electrically determined, easy to “flip” molecular configurations.

Another good book on the subject of thoughts directing brain changes is *Train Your Mind, Change Your Brain*, by Sharon Begley. This book describes brain scan-using studies that revealed that focused thinking modified or changed brain wave patterns and neural connections, and that brain structures and zones changed or grew larger or smaller in response to repetition of focused thoughts. Other books, including *The Brain That Changes Itself*, by Norman Doidge, M.D., and *The New Brain*, by Richard Restak, M.D., are included in the suggested reading bibliography in the appendix.

“Canst thou not minister to a mind diseas’d, Pluck from the memory a rooted sorrow, Raze out the written troubles of the brain, and with some sweet oblivious antidote cleanse the stuff’d bosom of that perilous stuff Which weighs upon the heart?”

- Shakespeare’s Macbeth

CHAPTER TWELVE

DISSOCIATION RESPONSES: PART 2

SELECTIVE DISSOCIATION

Selective dissociation is the compartmentalization or separation of certain thoughts or memories away from normal consciousness.

A mild amount of selective dissociation is perfectly healthy. A high level of dissociation can even be thought of desirable in some instances. Consider the “absent-minded professor” type. He is able stay highly focused on his research while selectively dissociating from events such as hunger, the passage of time, and social events or calamities. He may even be highly respected because of his extreme ability to dissociate from any distractions that interfere with his research.¹

¹ Intelligence and the ability to dissociate may have a linked genetic component. PDer’s tend to be highly intelligent *and* have a high capability for dissociating. This suggests that the long-sought genetic link to Parkinson’s disease, if found, may prove to be a genetic factor related to intelligence and dissociative ability, rather than a gene that triggers cellular pathology. If this is the case, the gene is not causing Parkinson’s, per se. Genes that impart an elevated ability to make a choice towards dissociation may be more common in people with Parkinson’s than in the general public. But the gene itself would not *cause* a pathological pattern. The PDer’s *choice* to *misuse* this ability may contribute to his eventual manifestation of Parkinson’s.

If *choosing* what turns out to be a pathological level of dissociation is a part of the Parkinson’s syndrome, we can start to understand why most identical twin studies show that PD is not genetic, even while epidemiological studies show that Parkinson’s tends to run in families. Parkinson’s is not strictly genetic, but the ability to dissociate may be. If this is the case, family values and cultural influences that honor the “stiff upper lip” may reward the person who dissociates from his emotions. A genetic tilt towards high mental capability and its corollary, a high ability to dissociate, *combined* with cultural or family praise for “sucking up the pain” or cultural or family disdain for showing emotion, *may* set a person on the path of dissociating early and often.

Also, to the extent that genetics may play a role in the degree to which a person uses more visual memory or more verbal memory, genetics may also help determine the way that a person processes trauma. People who have primarily visual-based (or possibly any sensory-based) memory function may have a hard time compartmentalizing, mentally organizing, and de-traumatizing horrific events. Oppositely, people who have primarily *word*-based memory function are able to make up justifications, modify facts, and otherwise organize traumatic experiences in such a way as to diminish the impact. Post-Traumatic Stress Disorder (PTSD) occurs primarily in people who use visual-based (or other sensory-based) memory. Traumatized people who primarily use word-based memory are much more able to process their traumatic experiences and organize them into tolerable “memories” that are more compartmentalized and justified, or at least explicable. They tend to not develop PTSD.

Dr. Temple Grandin, in her book *Animals in Translation* (Simon and Schuster, NY, 2005) refers on page 194 to the work of Dr. Ruth Lanius at University of Western Ontario. Dr. Lanius “did brain scans of people with PTSD as a result of sexual abuse, assault, or car crashes and people who had suffered the same experiences without developing PTSD. The main difference she found between the two groups was that one group remembered their trauma visually and the other remembered it verbally, as a verbal narrative. Their [brain] scans backed this up. When people with PTSD remembered the trauma, visual areas of their brains lit up (along with other areas), and when people without PTSD remembered their traumas, verbal areas lit up.” (Continued on next page.)

However, pathological levels of selective dissociation can result in disorders ranging from selective amnesia, at the mild end, to multiple personality disorder (which has actually been renamed “dissociative identity disorder”), at the extreme end. In some cases, if the selective dissociation is somewhat severe, it can cause depersonalization.

(An historical aside: the two “dissociative” conditions that I need to differentiate by the use of the adjectives “automatic” and “selective” were, by chance and at different times, both named “dissociation” in recognition of the depersonalization that can be characteristic of these two otherwise very different events.)

An example of selective dissociation: Numb from the waist down

The following case study involved in one of my patients who had completely recovered from Parkinson’s disease. Five years after recovering, she called me from out of state with a new problem: she was numb from the waist down. Because of our successful work together on her Parkinson’s, she hoped I could help her with this new numbness. She couldn’t get to Santa Cruz any time soon, so we decided to try to fix the situation over the phone.

The numbness had started one week earlier. I asked a lot of nousey questions, and the following story came out. A week earlier, she had decided to enter into a romantic relationship with a man. She had never been with a man; she had been a lesbian. She had broken up with her long-term partner more than a year earlier. Now, in her mid-forties, she had fallen in love with a man. She was considering entering into what she hoped might be a satisfying sexual relationship. But the day after she kissed him for the first time, when she woke up in the morning, she was numb from the waist down.

I asked her specifically about any negative associations she had with regard to male sexuality. She finally remembered one seemingly mild, almost cute, situation. When she was nearly four years old, her mother had discovered her lifting her skirt and “showing my privates to the little neighborhood boys. My mother grabbed me and brought me into the house. She told me that I had made the Holy Mother cry. I was devastated at the time. But now I think it’s funny.”

We agreed that this situation *might* be at the root of her sudden numbness. Since she might have selectively dissociated from the physical pain of being devastated, she might benefit from re-associating with it. I used a re-associating technique, one that is too difficult for most people who still have Parkinson’s, but since she’d recovered from Parkinson’s already I thought she might be able to do it.

Over the phone, I led this woman through a variation on the “soak the problem in the heart” exercise developed by the Heartmath Institute. She imagined how she had *physically* felt at the time she was so devastated. Next, she imagined that her physical heart was floating inside her chest, floating in a sea of supportive waves of energy that were being made by her heart. When she could imagine the feeling of energy waves expanding in her chest, she imagined that the physical pains she’d felt because she’d made the Holy Mother cry were floating in the same

(Continued from previous page.) Most PDers in our study have been highly word-based. We suspect that the ability to selectively dissociate may also be related to the process by which word-based people can more easily “classify,” or “make sense of” painful experiences and thus avoid the more *obvious* symptoms of post-traumatic stress.

waves of heart energy. (This exercise is included in chapter xxx of the Treatment Technique section of this book.)

I talked her through this exercise on the phone, and after about ten minutes of feeling her childhood pain floating in the heart waves in her chest, while simultaneously feeling the waves of heart energy, the pain diminished. As the pain diminished, she felt sensation returning to her abdomen, her groin, and then her legs. The numbness never returned. When she started the exercise, she was surprised at how much *physical* pain she felt at first when she allowed herself to recreate the “You made the Holy Mother cry” episode. Her stomach was knotted and her abdomen was tight.

I wish to make clear that the point of the exercise is not to wallow in pain, but to let the dissociated pain become re-associated into normal consciousness so that the person can accept that the pain did occur, neutralize the pain, which is to say, *heal* it, and move on. *Consciously* remembered, correctly processed events cannot harm the body nearly as much as events that terrorize the body from their secret hidey-hole in the subconscious. Unaddressed pains are nearly always more terrifying than pains that are looked square in the eye and recognized for what they are: fleeting pains. The old adage comes to mind, “Look fear in the face and it will soon cease to trouble you.”

This example demonstrates how the *pain* from which a person has selectively dissociated can lay buried, even while the word-based, conscious mind has created a “laughable” memory of the situation. It also shows how a purely mental dissociation can, years later, have a sudden, very powerful effect on the physiology.

I included this example primarily to make the point that the mind can exercise a fantastic, even bizarre control over the body. Also, that the symptoms that erupt from selective dissociations can come and go: they are not necessarily constant. Pathological symptoms, that is to say, manifestations of subconscious fears from which a person has selectively dissociated, can be triggered or worsened by thoughts, moods, and environment – just like the motor initiation and tremor symptoms of Parkinson’s.

Frequent dissociation

A person who has learned to dissociate easily and often, as many PDers do, may realize, at some point during recovery, that his first line of defense against anything he doesn’t like has been to dissociate from the emotional or physical *sensations* associated with it. As will be explained in more detail in chapter xxx, “sensations” refers to any of the five sensations of sight, smell, taste, hearing, and touch.

Some PDers have realized, during recovery, that they are almost *constantly* selectively dissociating from the physical sensations of non-emergency, physical *or* emotional, events. They sometimes realize that, with worsening Parkinson’s, they have increasingly dissociated from *anything* that interferes with their non-stop stream of mental anxieties. For example, many PDers who *want* to be less anxious have told me that they used to enjoy listening to music, but now they ignore music or don’t like to even have it around: music is too distracting; it distracts them from their mental focus. When I ask what they are usually focused on, they say that they’re

usually focused on their anxieties. In other words, they avoid or dissociate from the very things that would allow them to *stop* being anxious, even though they claim to dislike the anxiety.¹

A few case studies

Depersonalization

After we realized that dissociation might be playing a role, I conducted the following exercise with all my PD patients who were stuck in partial recovery.

The patient was always lying down on the treatment table, relaxing. I said to him, “Imagine your entire body as being filled with light, beautiful, perfect light.”

After the PDer assured me that he was successfully doing this, I asked, “Where is that body of light?”

I did this exercise with healthy people, as well. When I asked the healthy people, “Where is that body of light,” they were puzzled. They always replied something like, “It’s inside me. Isn’t that what you asked me to imagine?”

The PDers who were stuck in partial recover *never* said that. The replies from those PDers ranged from “It’s sitting over there on your office sofa,” to “It’s floating in space about two feet above my body,” all the way to “It’s in Vermont, playing on the sea short, and it’s ten years old.”

Just in case they had misunderstood the question, which I carefully phrased exactly the same way with each subject, I asked them if they could please let their body made of light into their physical bodies. The PDers who were stuck in partial recovery had responses ranging from the fairly common “I can get the head to be in my head but, from the neck down, the rest of the body is floating outside my physical self,” all the way to “No! That would be disgusting! I refuse to even try.”

After much struggle, some PDers were able to force themselves to imagine, briefly, that their “body of light” was inside their own body. However, many of them noticed that one limb – very often but not always the leg with the foot injury – could not be forced into the physical body. Sometimes the imagined *left* leg (if the injury was on the left foot) might be crossed over the right leg, even though the real legs were stretched out straight. Sometimes the imagined right leg (if the injury was on the right foot) might be alongside the physical leg, separated by only a few inches.

Even if partially recovered PDers were able to force themselves to imagine that their own physical bodies could be filled with beautiful light, they were unable to imagine that their bodies could be filled with energy and sensory awareness in *conjunction* with that light. Even if they

¹ When I first meet a PDer, he often insists that he never dissociates. Very often, it is the spouse that points out examples of the PDer’s nearly constant dissociations or long-term emotional non-responsiveness. At this point, the PDer may well reply that he isn’t doing it consciously, so therefore he is not responsible for his actions and can’t be expected to change.

Along the same lines, many PDers in our experience have been proud of their high level of “pain tolerance.” We now suspect that their ability to minimize pain signals is due to their high ability to dissociate from pain. Our patients have usually assumed that their high level of pain tolerance was due to superior nerves or superior will power. They are therefore amazed during recovery when, ceasing to dissociate from their own body in order that foot healing may begin, they find themselves highly susceptible to pain and emotionalism in response to every little ache or twinge. During recovery, many PDers have had to learn how to feel the new onslaught of sensations without becoming emotionally attached or terrified of them: they must learn to recognize that a sensation, per se, is merely a sensation and not necessarily a personal threat that is best countered by becoming “heartless.”

could force themselves to “picture” it, they could not force themselves to “feel” what it was like when they imagined light and energy and vitality inside the physical form.

Healthy people feel a sense of warmth, vitality, or something when they allow themselves to take the time to “see” or imagine their bodies filled with radiance. Partially-recovered PDers were utterly unable to feel anything in response to this exercise.

This type of selective dissociation is a variation on depersonalization, and *all* my PD patients that were stuck in partial recovery had this symptom.

Creation of a non-functional body part

Other manifestations of dissociation in some of the same PDers were more complex than simple depersonalization.

One patient was absolutely unable to move his arms, even though his legs were once again functional. His arms were clamped so tightly to the sides of his body that he could no longer work. His hands tremored violently. A nurse came to his house three times a week to bath his armpits. The nurse needed to use all her strength to create a wide enough slit between his arm and his torso so that she could get a washcloth up to his armpit.

After doing Tui Na and various acupuncture treatments on his arms, neck, head, and spine, I asked him to close his eyes and imagine his body full of light. After he said his body was full of light, I asked him where his arms were. He said that they were sticking straight up in the air, poking out of his shoulders. They were withered and rigid, like sticks from leafless trees in the dead of winter.

I pointed out that his arms weren’t actually sticking out of his shoulders, and would he please try to imagine that his arms were inside his physical arms. He made a half-hearted attempt, and was unable to get his “mental arms” to go inside his body.

As an aside, I *was* able to get him to move his whole body, including his arms, by having him pretend that he was a five year-old child pretending to be an airplane. He tried for over an hour, insisting that he couldn’t do it. At some point he became furious with me. He screamed, “What do you want me to do? Actually put my arms out to the side and pretend I can fly?” He gave an exaggerated demonstration of a plane banking steeply, and as he did so, his arms lifted gracefully out to the sides. His tremor stopped. He was able to pretend to be an airplane – arms and all. He was so excited that he followed the airplane maneuver with some folk dance routines that he’d done as a young man. For over an hour, he was able to move his arms normally – and did not tremor. After it was over, he told me he’d only been able to do it because I had been there. He just *knew* he would never be able to do it again. And he never did. His mental image of his own arms remained, until his death four years later at age fifty-four, an image of dried, rigid sticks, poking straight up out of his shoulders.

In this case, he had not merely dissociated from his arms or depersonalized his sense of where his arms were. He had created the idea that his actual arms were dead and sticking straight up out of his shoulders. Based on the extraordinary degree of rigidity that he manifested in his arms, I might guess that what he was doing was much more destructive to health than merely imagining his arms were OK, but outside of his body. He was not the only partially-recovered PDer who had created mental images of diseased or decaying body parts.

Situation-specific dissociation

One patient, visiting from Germany, had two weeks of treatment in Santa Cruz. During this time, she became able to move perfectly normally. Her health practitioner had been doing Tui Na on her injured foot for many months and they had both noticed many recovery symptoms. All we did in Santa Cruz was a little additional Tui Na on her leg *and* encouraged her to let go of her negative attitude.

On her last day, I congratulated her that she no longer had Parkinson's. She stared at me in amazement. "I still have Parkinson's," she said.

I asked her why on earth she thought she still had Parkinson's.

She replied, "I don't have it here, but when I get home, it will come right back. I know it will. I don't have it here, because here it's OK to walk around smiling, without a care in the world. But when I get back to Germany, I'm not going to walk around smiling, looking like a stupid American."

I asked her if she might be willing to smile even in Germany, since she felt so strongly that being able to smile had been the *temporary* cure for her Parkinson's. She said that she would not smile when she returned home. So I asked her, "You'd rather be unable to walk than have people think you're smiling too much?"

She replied, "I'll have to think about it."

When she got home, her Parkinson's reappeared.

At this point, she no longer had symptoms of idiopathic Parkinson's disease. What she had after this was psychogenic parkinsonism.

Event-specific dissociation

One patient with a PhD in nutrition never tremored or had any other symptoms of Parkinson's disease while she was eating desserts. I asked her to run a few experiments. She tried eating dessert first, dessert before the main dish, during the middle of the meal, and after the meal. She tried eating only dessert, and eating only the main dish. No matter when she ate it, she never tremored during dessert and she tremored violently during the main course.

I asked her if she had any idea why. She replied quickly, "Of course. It's because desserts don't matter."

When she was eating desserts, nothing mattered: she could be relaxed. But when she ate nutritious food, she was keenly aware of the nutritive value of the food, thanks to her PhD. As soon as she ate nutritious food, she would switch over to "thinking mode," and shut down her heart. As soon as her heart was shut down, she had all the symptoms of Parkinson's disease.

Dissociation from the heart

The intentional dissociations from the heart itself, and/or the diseased images of heart that PDers had created, and/or the situation-, event-, and mood- specific triggers that PDers used to *amplify* the heart shut-down, causing their PD symptoms to appear or to intensify, will be shared in later chapters that are devoted specifically to dissociation from the heart. The above examples were merely presented to illustrate what is meant by body part-, situation-, and event-specific dissociation.

SELECTIVE DISSOCIATION LEADING TO SYMPTOMS OF AUTOMATIC DISSOCIATION

At the beginning of the Little Project, we did not suspect the weird truth: that most PDers had dissociated from their hearts. Even though a not uncommon recovery statement was something along the lines of “Oh. I just remembered when I decided to not be able to feel pain,” we just assumed that they had only blocked out the memory of pain. Because many people *temporarily* block out pain signals and don’t develop Parkinson’s, we did not consider that these statements were hinting at a significant commonality in Parkinson’s.

Only after nearly a decade of research did we begin to suspect what was going on: a *consciously*-induced (and therefore selective) *automatic* dissociation. And it was being activated via *selective* dissociation from the heart’s ability to resonate with pain.

We spent a lot of time pondering the connection between dissociation from the heart and automatic dissociation. During automatic dissociation, dissociation from the heart is normal. But does the reverse also occur? Does dissociation from the heart trigger symptoms of automatic dissociation? We were able to prove that it does, although we still can only guess at the mechanism.

One of our guesses is that the shutdown in the heart is the very thing that triggers all automatic dissociations. Possibly, whether or not the shutdowns are caused intentionally *or* by severe injury or trauma, they all start with a heart disconnection. When severe trauma occurs, the body’s electromagnetic fields become highly destabilized. The heart, strongly resonant with electrical fields, may be the first organ to react. Possibly, it reacts by shutting down its ability to resonate with pain, setting in motion the symptoms of automatic dissociation. If this is the case, it makes sense that mentally *choosing* to shut down the heart’s ability to resonate with pain, intentionally inhibiting the heart to a moderate degree with regard to certain types of pain sensations, might cause some of the same processes that occur when the heart is utterly stunned via trauma.

The reader may be surprised that an electromagnetic shift in the heart could trigger so many reflexive changes in physiology. As you will read later, the heart’s electromagnetic fields drive more body processes than the brain’s much weaker fields. When a person is in parasympathetic mode, brain waves entrain with the much stronger heart waves: brain waves are subordinate to heart waves. Only when a person moves into an adrenaline-dominant state do the brain waves act independently from the heart waves. In a healthy person who is in parasympathetic mode, the heart waves “instruct” the brain waves, and thus determine an enormous amount of physiological behavior. Shutting down the heart, oppositely, forces a person to be brain-dominant, unable to *resonate* with, unable to *feel*, sensory input.

Partial recovery and selective dissociation from the heart

By 2005, we had strong evidence to suggest that many PDers, and in particular those PDers who became stuck in partial recovery, had selectively dissociated from their hearts. By 2007, we had begun to suspect that this rare type of dissociation was the trigger for those constantly fluctuating mood- and thought-based Parkinson’s symptoms (as opposed to constant, unchanging foot injury-induced symptoms) that looked so much like a mild to moderate version of automatic dissociation. We were able to run several experiments that proved that this triggering *can* occur. We were also able to *prove* that this triggering *was* active in those of our

partially-recovered PD patients who were still manifesting thought- or mood-based parkinsonism. Those proofs are included in chapter xxx.

In partially recovered PDers, this triggering of severe heart dissociation occurred either in intermittent lapses, increasingly, or almost constantly. It occurred even *after* their foot injuries healed and they had manifested various recovery symptoms related to the healing of aberrant channel flow.

While we were able to uncover some physiological muscles in the pericardium (around the heart) that can get “stuck,” thus literally holding the heart in a position appropriate for fear or anxiety, we also determined that mental attitude played an enormous role in determining whether or not partially-recovered PDers’ hearts were shut down at any given time.

As we discussed this finding with PDers, many of them wondered at why they might have a fear-based, or “negative” attitude.

Learning to fear

Many PDers often insist that they have consciously worked hard at *not* having a negative mindset, or at least not presenting it to the world. Or they only recall one or two negative experiences. They wonder why they have might have become susceptible to negativity, wariness, or increasing reliance on selective dissociation.

First off, the fact that they have *worked* at fighting off negativity suggests that possibly some negativity or wariness lurks in their subconscious. This negativity or fear may be snuggled in amongst the dissociated thoughts and memories that have been separated off from normal consciousness, but which are still sending out warnings.

For those who do not recall *many* negative or fear-inducing events and therefore doubt that there is *much* negativity or fear lurking in the brain, it might be helpful to consider how the brain handles fear-inducing events. New research on brain plasticity and *animal* behavior explains how one or two mild fears, if not processed correctly using our *human* capability to confront and neutralize our fears, can expand into a near infinitude of fears over time via the wordless, dumb animal, portion of the brain.¹

Learning through linkage: the corn dog event

Because of the way the animal brain builds associations and links, items that were *ever* associated with risk or danger, however peripherally, will have equal “Danger!” status in the brain with all other “Danger!” associations. For example, if a deer is jumped by a mountain lion but manages to escape, the deer will associate every sensory input during the event as something to fear. In this way, the deer will have learned from its mountain lion experience that early morning, high ledges, creek sides, long shadows, the smell of willow, the sound of a plane high overhead, and empty potato chip bags by the side of the trail are not only *all* potential mountain lion-creating risks, they all have *equal value* as potential risks. The deer *cannot* use logic to figure out which of the lion-associated visual, smell, touch, taste, or hearing perceptions were of

¹ When considering the “dumb animal” model for understanding how animals learn to fear, please do not include dogs in your model. Domesticated dogs are genetically different from wolves primarily in that they do not develop characteristics of mature wolves. All their lives, dogs remain more like wolf pups than like mature wolves. Dogs’ ability to learn about danger is also immature. A dog must be horribly abused before he will learn the wariness that wolves develop quickly. Dr. Temple Grandin’s book, *Animals in Translation*, goes into this subject in depth. Please use models such as deer, tigers, geese, or wildebeests when considering an animal model for this section on dumb-animal fear.

greater significance when it came to producing a mountain lion attack. The deer has to remember all of the associations. In the deer's mind, all of these sensory perceptions are remembered and linked to danger.

In humans, this animal brain is at work any time that the human fails to calmly consider a past trauma and process it in a way that sorts out the risk factors from the non-risk factors.

For example, a person who got sick at the boardwalk after eating a corn dog and then going on the Tilt-A-Whirl may find himself with a lifetime aversion to corn dogs.

Then, because of the fear-linking process in the animal part of his brain, this same person may find, ten years later, that he scorns all ocean-side amusement parks. Twenty years later, as the amusement park fear linkage continues to make more links in his brain, he may feel a virtuous superiority towards "the inferior type of person who could possibly enjoy going to 'those kinds' of places."

If this person had taken the time, later that day or during the week of The Corn Dog Event, to calmly recall the events of the fateful day, processing the sensations and embarrassment of being sick *and* also giving careful thought to the situation, he might have ended up with the wisdom that a full stomach and rapid motion don't mix well. He might also have a gentle tolerance or even a soft spot in his heart for kids of all ages who go to amusement parks – especially those who eat corn dogs.

As an aside, I cannot begin to guess how many PDers have told me that they could really relate to the "hating corn dogs and eventually hating everyone who goes to the amusement park" scenario. Though many had never eaten a corn dog, they could recognize that they increasingly tend to dismiss, disdain, or be wary of whole groups of people, despite their conscious efforts to be as loving and circumspect as possible.

As for taking the time to calmly process unpleasant events sometime after the fact, a process called re-association, chapter xxx of this book includes a description of the *physiological* steps involved. An emotionally healthy person *automatically*, without any training, performs these steps as soon as the trauma is behind him and he feels safe. We have found that most PDers are completely unfamiliar with the sensations and thoughts involved in this ordinarily automatic process, the process by which temporarily dissociated events are brought back into normal consciousness and de-traumatized.

Learning through linkage: from car crash to pleasure craft

As noted above, the animal brain "learns" about danger by making linkages. Another example of linkage is the person who is in a near-fatal car accident. The last thing he hears is the sound of a car horn blaring loudly. He recovers, but six months later, strolling past the downtown *pizza* parlor, a car horn blares loudly. He has a panic attack while standing outside the pizza shop. He assumes that he was startled by the loud car horn. But four months later, while vacationing at the ocean side, gazing at the *sailboats*, he walks past the boardwalk's pizza shop, and has a panic attack. This time, he has no idea what caused it. Several months later, in the depths of winter, he is looking dreamily through some Tropical Island Summer Vacation brochures. When he sees the picture of the sailboats, he has a panic attack. At this point, he may just assume that he has become the sort of person who panics easily. But these attacks were not random. His animal brain had been making linkages. He linked the car horn to danger and fear,

he linked fear and panic to pizza, pizza to sailboats, and voilà! He panicked at the picture of a pleasure boat.

People who are even subconsciously determined to selectively dissociate from the things that they fear or dislike do *not* process their fears or dislikes at a later, safer time, nor are they able to take advantage of the human capacity for reason. Worse, their fears can grow: their mentally formed *linkages* to and from dissociated negative experiences may cause them, over the years, to dread *anything* tangentially or even remotely linked to previous fear experiences. Their fears can thrive in the subconscious, busily doing what the animal brain is supposed to do: making linkages. At some point, without knowing why, the PDer who has dissociated from pains and fears may find himself wary or anxious about nearly everything. Or, as in the case of the woman who went numb from the waist down, a dissociated pain can erupt in bizarre, seemingly random manifestation. These manifestations might be somewhat characteristic of a partial or profound fear response, or a protection-from-pain response (numbness) even though the *prompt* for the response seems unrelated to anything fear-inducing.

Learning through habit

People who make a habit of selectively dissociating from anything that might potentially be painful may not even realize the extent to which they are constantly living in fear. It is reasonable to dissociate during a crisis. But if a person doesn't bother to take time, after the crisis is over, to experience his pains and transmute them into an emotionally neutral learning experience, he may find that, over time, his emotions increasingly move towards wariness and away from contentment.

For example, people with Parkinson's who use their tremor as an indicator of their mindset or emotions can usually see how their tremor is triggered more and more easily over time, by smaller and smaller worries. At some point, most PDers with tremor can activate or increase the amplitude of their tremor merely by remembering that they have a tremor, or by noticing that their tremor has temporarily become somewhat smaller or has stopped. The mere thought, "tremor," has become associated with other thoughts of fear, sometimes even a fear of worsening Parkinson's, and so the *thought* of tremor increases or activates the tremor.

This extremely brief section on brain linkages and fears was included to demonstrate for the PDer that dissociation can bring about highly illogical fears, fears that are impossible to "track to the source." PDers who get stuck in partial recovery are usually keen to "figure out" what the "thing" was that made them start dissociating in the first place. Oppositely, those PDers who have fully recovered have *not* worried about whatever it was that first made them dissociate.

Those who have managed to overcome partial recovery and join the ranks of the fully recovered have been those who said to themselves something like, "Heck with it. I don't know, and I can't know. So I'm going to stop being afraid: I'm going to *surrender* control of my safety over to the universe. From now on, the universe is going to have to keep me safe: *I* can't even begin to know how to keep myself safe." And then when they suddenly realize, deep inside, that the universe *has* always been taking care of them, they feel safe. And as soon as they feel safe, the heart kicks back into parasympathetic, the dopamine starts to flow. They start to feel what it's like to be inside their bodies. They can imagine movement, and they can execute that movement. It's very simple, really.

Ironically, after PDerers decide to just surrender and to not worry about which specific event(s) might have set their heart dissociation in motion, and after they have obviously recovered from Parkinson's disease, they very often remember, spontaneously and with mature understanding, an event that was unforgivable or terrifying at the time.

Also ironically, if I had to pick one word that our partially-recovered PD patients really hate, it would be the word "surrender."

Two more example

This example shows how the original cause of fear no longer matters. A partially-recovered PDer was having worsening tremor, especially when eating. As soon as she thought about eating, her tremor became violent. She explained that the tremor amplified because she knew it was going to get bigger.

Chris Ells asked her to focus her mind on a song while she was eating. As soon as she realized that she wasn't focused on the song, she had to put down her spoon and start over. (Brain researchers have found the area in the frontal lobe of the brain that forms a linkage between music and the physical heart, thus "opening up the heart" somewhat.)

It was extremely difficult for her to keep her mind on her song. And everytime she started to tremor, she realized that she'd stopped the music just before the tremor started. Finally, after forty-five infuriating minutes, she fixed her mind with intense concentration and was able to *stay* focused on mentally singing her song. She was also able to eat without any tremor.

When she reported this to Chris, she laughingly said, "It worked, damn you!"

This same person had increasing difficulty in taking a long stride, or even initiating a stride. After her treatment, Chris asked her to *not* think about how far she needed to walk to get to the waiting room. He asked her to only think about the movement that she needed to do in that moment. When she was finally able to understand what he meant, she realized that she could take graceful, long strides. As soon as she started to think "How much farther?" she would start to freeze up.

These two examples show that the PDer was *not* trembling or freezing up because of some deep, dark, long-buried fear. In the first example, her amplified tremor was being caused by her fear of amplified tremor. In the second example, her inability to walk was triggered by her fear that she wouldn't be able to walk far enough. In both cases, a very immediate fear was the one that was causing her to slip into heart shut down mode, which in turn made her tremor or unable to move.

The real problem for her was *not* the original thing(s) that triggered a heart shut-down. The real problem was that she had increasingly taught herself and allowed herself to use the heart shut-down for every possible difficulty. She had gotten to the point where the thought of difficulty caused her to shut down. And when she shut down, her dreaded difficulty came true, thus strengthening her conviction that she was doomed. Her strengthened conviction made it even more necessary for her to shut down.

In PDer who get stuck in partial recovery, the vicious cycle of self-fulfilling need to shut down because of the pain of not being able to function makes the PDer increasingly less functional.

Those people who fully recover decided physically hold their hearts in the open position. They do *not* dig deep into their subconscious and find the original event that caused them to shut down the heart.¹

OVERRIDING AUTOMATIC DISSOCIATION

Getting back to the theory, it's time to discuss why the various types of dissociation don't render the PDer absolutely comatose.

The adrenaline override

A person whose movement inhibition is caused by a severe injury can always override the inhibition by using adrenaline. We see this all the time in nature. For example, when a hungry lion appears on the scene, a deer or a human can run on a broken leg: adrenaline overrides injury-induced movement inhibition.

Likewise, by using adrenaline, a person can learn to override any automatic dissociation that has been *selectively* induced.

¹ The ability to shut down the heart to avoid feeling pain or pleasure may well have a genetic component. Some people believe that genetics are merely physical manifestations of a person's mental tendencies, "selected" prior to birth because those genetics are the best "fit" for the individual's vibrations of consciousness. It makes sense that a person who, in a prior life, developed the mental control necessary for dissociation, would fit best in a body that is genetically suited for high intelligence. Many PDers have told me that they suspect that they were born with the ability to dissociate.

This recalls to mind how, during the dark ages, many well-meaning but misguided religions emphasized the idea of dissociating from pain. Museum pieces such as whips for self-flagellation, and clothing imbedded with shards of metal, are reminders of the dark ages mentality that assumed "higher realms" could only be known by "killing the feelings in the body."

My first group of twelve volunteers had a disproportionate number of professional religious: a retired Lutheran minister, a retired Presbyterian minister, a Catholic nun, a life-time Baptist missionary. The group also included a highly devout Korean Methodist whose father had founded the Methodist church in Korea, a devout student of the Book of Miracles, and two yogis. There is a powerful spiritual streak in nearly all of my PD patients, whether or not they are members of any specific church. Ironically, PDers are often perceived as "cold," because of their reliance on mind instead of heart.

I have to wonder if, in lifetimes past, many PDers have tried to attain to higher realms by teaching themselves to become numb. In answer to these PDers, I quote from Paramahansa Yogananda's translation and interpretation of *The Bhagavad Gita*: "The scriptures and [saints and sages] do instruct the devotee [of God] *not* to destroy the actual senses, but to slay their bad habits. The devotee is not asked to blind his eyes, deafen his ears, nor to paralyze his senses of smell, taste, and touch. He is directed only to dislodge the enemies of optical, auditory, olfactory, gustatory, and tactual *attachments* [preferences; ego-based likes and dislikes – which includes pain: sensation that is disliked] which keep the soul imprisoned, forgetful of its omnipresent kingdom."

At any rate, many PDers have told me that they think that they were born with an innate ability to dissociate from the heart, and that they used this ability in their youth to give themselves a sharper mental focus and to stay impervious to pain. Many also admit that they had terribly painful childhoods, and the ability to dissociate came in handy. One has to wonder if these people didn't choose the "terrible" childhoods into which they were born. By choosing these childhoods, their omniscient souls knew that they would have an opportunity to either indulge in their chosen game of dissociation or to make enormous spiritual progress in learning the correct way to deal with pain. Either way, the Parkinson's presents the logical conclusion to the "trick" of turning off the heart. A person can respond to Parkinson's with more of the same heart shut down. But Parkinson's also can be the logical, if unpleasant, conclusive force that compels a person to finally master the *conscious* ability to open the heart. When the PDer learns to intentionally open his heart despite all circumstances, he starts on the road of truly mastering his heart and overcoming his mind-based attachment to his ego-based dislike of pain or pleasure. This is not to say that a person should learn to *like* pain. A person must learn to perceive pain via wisdom and love. Wisdom and love can neutralize the emotionalism of pain, thus allowing pain to be perceived as what it actually is: vibrations of energy.

Early in their teen years or their early twenties, many of our patients learned how to create almost constant surges of adrenaline in order to override their budding tendency toward movement inhibition: what one PDer called “the push-pull of Parkinson’s.” They learned to activate adrenaline release by staying terribly busy, creating “false emergency” reasons for completing mundane tasks, or actually participating in dangerous activities such as extreme sports.

Adrenaline, the neurotransmitter of fear, pushes the buttons that override movement inhibitions, whether the inhibitions were induced by injury or automatic dissociation. Even a person with advanced Parkinson’s can initiate movement perfectly normally during a *true* emergency.

But after a lifetime of creating false emergencies, or as Aesop would say, “crying wolf,” the PDer becomes too calm or too tired to activate much internal sense of excitement over his false emergencies. Like the villagers in Aesop’s story, the PDer no longer responds to his false emergencies. The PDer’s level of mentally-induced adrenaline release starts to drop. But by this time, he has become too habitually wary to feel safe. So he finds himself in an awkward neurological position: he is wary but he can’t muster the adrenaline to back up the wariness. And/or his movement inhibitions are getting worse, but he can’t muster the increasing amount of adrenaline needed to override the increasing movement inhibitions. At this point, his injury-based movement initiation problems and/or symptoms of automatic dissociation become visible. We then call these symptoms “Parkinson’s disease.”

Parkinson’s disease does not appear when a person’s dopamine levels are too low. The PDer may not have even *been* using much midbrain dopamine for decades.

Parkinson’s symptoms appear when the PDer is no longer able to conjure in his mind a strong enough adrenaline-releasing crisis to override his steadily increasing movement inhibitions. The symptoms also show up when the PDers tendency for chronic wariness is greater than his ability to summon up the adrenaline necessary to support that wariness. Wariness *with* adrenaline makes a person poised, ready to respond. Wariness *without* sufficient adrenaline makes a person anxious, shaky, and vulnerable.¹

Again: we hypothesize that, when the PDer’s *movement inhibition* (which might be physical *or* mental) is greater than his ability to induce an adrenaline-releasing override, he will begin to manifest the symptoms of movement inhibition, rigidity, postural instability, and/or tremor. Also, when his *habitual wariness* and/or *fear* (which are purely mental) is unable to trigger a tremor-overriding amount of adrenaline, he will begin to manifest the symptoms of movement inhibition, rigidity, postural instability, and/or tremor.

At this point, he will *start* showing overt signs and symptoms of Parkinson’s disease. However, he didn’t “just get” Parkinson’s. He’s been slowing developing it for decades, and actively hiding it by using either the low levels of adrenaline that are available to a person in automatic dissociation, or the high levels of adrenaline that are available when a person lives in fear or creates an “emergency” out of everything.

¹ Peter Levine has written an excellent book, *Waking the Tiger*, which discusses the psychological problems that arise in people who, in response to fear-inducing situations, get stuck in the wariness stage and are unable to make the transition into adrenaline-based action. Some PDers, in their early years, had the opposite responses: they responded to *everything* with action. When the Parkinson’s appears, they may find themselves stuck in wariness, but not because they *cannot* let themselves emotionally transition into adrenaline-based action. They can’t raise the necessary adrenaline because the things they are wary of (nearly everything) aren’t really worthy of the *level* of fear that is needed to cause an adrenaline surge big enough to override their movement inhibition.

A few of our PD patients have protested vigorously that they have no fear, and therefore, have not been using adrenaline. We have pointed out that fear of being criticized or judged, fear of being late, fear of making a factual or social mistake, and a thousand other “little” fears are the more typical fears of the PDer. These fears, though small, tip the neurotransmitter balance in favor of adrenaline.

SUMMARY

The basis for our hypotheses

When I first started working with PDers, I was surprised at how many of them had a stoic attitude towards their bodies – one that was combined, in many cases, with a powerful aversion to therapeutic touch.

Over the course of several years, we discovered other emotional similarities in partially recovered PDers, such as an inability to visualize anything with a positive connotation, inability to daydream in a positive manner, or inability to imagine that their own bodies, especially the damaged areas, could be filled with light. The most stunning similarities in PDers who became stuck in partial recovery had to do with their diseased, deformed, caged, or otherwise abnormal mental images of their own hearts – if they were even able to conjure one.

A few partially-recovered PDers, after learning about our hypothesized heart dissociation problem, insisted that they were different, they could imagine having a perfect heart. If they claimed that they had a perfect heart, I only had to ask them to imagine that they were looking at a dear friend or someone they admired deeply. Then, I asked the PDer to imagine what *that* person’s heart looked like. *Always*, the friends’ hearts had qualities of radiance and beauty that the PDer could *not* imagine coming from his own heart.

Also, the strange remarks made by some PDers during recovery, remarks like “Oh. I remember when I started pretending that I couldn’t feel pain,” seemed to fit with the “It didn’t hurt at the time” phrases that young Tim and so many others had used when telling me about their injuries.

It took nearly nine years before we finally connected all the dots and could prove, in experimental fashion, that many PDers have, or had at some point during their childhood, intentionally employed a selective dissociation response to their heart’s ability to feel physical and emotional pain.

In addition to selectively dissociating from the *ability* to feel pain – a condition in which *all* neurotransmitter activity is diminished – many, though not all, of our PDer patients had lived life in a manner that suggested they’d consciously worked at staying in sympathetic (adrenaline-dominant) mode. When these PDers wanted to think about things or get something done, they always tended to think and move with adrenaline, not dopamine. But although they were adrenaline-dominant, their adrenaline *levels* were very often at the low end of the spectrum: they used highly self-controlled types of low-level-adrenaline behaviors. Their behaviors were not so much “fight or flight” as “perpetually wary or alert.” When using the sympathetic-dominant mode, a word-based person experiences his life primarily by *thinking* about it, rather than *feeling* it.

Some of these PDers had no memory of ever having engaged the parasympathetic system; the system in which *dopamine* is dominant and adrenaline is subordinate: the mode in

which a person experiences life through perceiving sensory input and *guiding* the mind via the heart's ability to resonate with sensory experiences and/or the heart's *intuitive* resonance.

Many PDerers agreed that they had experienced life predominantly via thinking. Many of them also had no idea what we meant by the word "feeling" or could not understand how a person's life *could* be experienced via sensory feelings, and via the feelings of the physical heart, in particular.

"Feel?"

One PDerer said that her girlfriend once asked her why, when they went to concerts, she spent her time looking at everyone and assessing what everyone was doing. "Why don't you ever just sit back and enjoy the music?" When the PDerer started being able to *feel* music, during recovery, she told me, "Until now, I'd never had any idea what my friend was talking about."

Another quick anecdote also demonstrates the difference between "feeling" experiences and thinking about them. A PDerer's wife told me about his reaction, or lack of, when they went to Florence to see Michelangelo's magnificent statue of David. As she and many others turned the corner and suddenly found themselves facing David, they were visibly stunned, in a chest-expanding, breath taking sort of way, or even moved to tears. As their hearts resonated with the statue, their chests expanded with a *feeling*, something impossible to describe. The PDerer was physically unaffected by the statue. He was duly impressed at the excellent lifelike accuracy of the lines, made estimates of the size of the body parts, the size ratios of the body parts, and constantly nattered away with questions about the sculpting techniques.

Years later, sitting in my office, venting, the wife stated that her husband had never *really* experienced the statue or, as far as she could tell, any other feelings over the course of their long life together. When she said this to me, the husband was clearly surprised. He didn't understand how she could say that he hadn't experienced the statue: he obviously had seen the statue. What the wife meant, of course, is that he hadn't *felt* the statue, or very much else in their married life together. When she tried to explain to him what she meant, he had no idea what she was talking about.

I have more than a hundred similar anecdotes.

The emotional flatness that frustrates so many PDerers' spouses is difficult to complain about: very often, PDerers, due to their extreme dread of social mistakes or of making trouble, are *perfect* spouses in terms of functionality. The world often perceives PDerers as hard-working, morally upright, generous and kind to others, responsible, non-violent, clever and, did I mention, hard-working? In the eyes of the world, the spouses of many PDerers have *nothing* to complain of. And yet, the growing emotional flatness of the PDerer and his inability to *feel* his own experiences can be very hard to live with or relate to.

The remarks of PDerers, their spouses, and even their children, help us figure out where to look for the underlying cause of Parkinson's disease. Of course, one could make the case that dopamine insufficiency, not dopamine inhibition, is the cause of the emotional flatness. Then again, when we were able to help them heal the foot injuries and taught them how to feel safe, the emotional flatness went away. Sometimes, recovered PDerers felt as if an entirely new world opened out for them, a world of wordless, sensory and proprioceptive self-awareness that they had never suspected was there.

PDerers entering our program usually wanted to regain their physical competence; they wanted to be their old selves. When they recovered, they consistently said that they didn't care so

much about physical competence: they were thrilled to be able to really experience life for the first time in a long, long while – if ever. They were adamant that they *never* wanted to go back to being their old way, ever again.

Those PDers who recovered, and ceased dissociating from their hearts, often said that they'd been given the opportunity of living two lives in one lifetime. In the first life, they had enjoyed mental keenness and had been impervious to pain. In the second life, they learned about joy. They always preferred the second “life.”

In ending this chapter on selective dissociation, I want to point out that some psychologists and psychiatrists feel that the condition is curable. In the Little Project, we found that the selective dissociation used by PDers is, in fact curable. But it takes work.



“I keep six honest serving-men (They taught me all I knew); Their names are What and Why and When And How and Where and Who.”

The Elephant Child, by Rudyard Kipling

CHAPTER THIRTEEN

DISSOCIATION QUESTIONS THAT ARISE

This chapter will consider some of the questions that often arise from a discussion of dissociation and the role of the mind in Parkinson’s disease.

Can a person actually cause physical changes by imagining his heart is gone?

It was all well and good to *hypothesize* that a person can selectively dissociate from his own heart, and in so doing, create symptoms of automatic dissociation. But we wondered if this hypothesis could be confirmed. So, in experiments using PDers, recovered PDers, and people with no knowledge of Parkinson’s disease, we were able to prove that pretending to wall off the heart can inhibit sensory perception and reduce a person’s ability to engage the parasympathetic system. We learned that merely pretending to wall off the heart can cause a reduction in heart rate and breathing rate, and can inhibit the ability to feel the expansion in the chest that a person feels when he feels safe. In other words, pretending to utterly wall off the heart *does* activate modified symptoms of automatic dissociation.

Details of the various experiments that we ran are included in Part three of this book, “Related research xxx.”

Can one override the pain of physical injury with dopamine?

In order to understand safe times and the placebo effect, it was necessary to hypothesize that a dopamine surge can override the movement inhibition that occurs following a severe injury.

It is easy to understand that adrenaline can override immobility. We can see examples of that in nature. But we also see examples of a dopamine override. Imagine a person with a very recent, very painful, badly sprained ankle, separated shoulder, and neck injury. Picture him limping about, feeling sorry for himself, and moving more slowly than normal. Now imagine how his movement ability changes when he is informed that he just won three millions dollars in the lottery. He can spin around, throw his arms in the air, talk a mile a minute and dance on his one good foot. This is a dopamine override. He is still injured, but the injury does not hurt, nor does it inhibit his ability to move at a normal pace.

Although a majority of PDers could relate to the idea of using adrenaline to override symptoms, a number of PDers had used dopamine-increasing methods. Some told us that, in the years before they were diagnosed, when they noticed that they were feeling stiff, clumsy, depressed or “just not right,” they would force themselves to sing songs or play a musical instrument. Even if they didn’t feel like making music, they had learned, over the years, that they could always banish the stiffening up or the feeling of wanting to curl up in a ball and stay there for a few years.

Some PDers used both: they forced themselves to enjoy music or other dopamine-releasing activities some of the time and created adrenaline-releasing “mental emergencies” at other times. Again, no two PDers are the same.

A chemical override

A person who is immobilized with painful injury can move somewhat normally if he takes a wallop of dopamine-enhancing drugs such as methamphetamine or opiates. A person who is immobilized with Parkinson's can move somewhat normally if he takes dopamine-enhancing drugs such as methamphetamine, cocaine, or the pharmaceutical dopamine-enhancing drugs.

A huge surge of dopamine can override the immobility of pain, injury, fear, or automatic dissociation. But the surge of dopamine doesn't get rid of the underlying problem; when the surge wears off, the immobility resumes.

A safe activity override: how it works

As noted in the beginning of this chapter, some PDers can initiate movement perfectly normally even if they have an unhealed foot injury, *if* they are engaged in an activity that they have subconsciously decided is safe. This phenomenon is well known and documented in many PD research reports. In these cases, the rare surge of dopamine that occurs from feeling safe is able to override the brain-induced immobility from injury and the physical damage and pain at the injury site. This proves 1) PDers do not have a dopamine deficiency, but in most circumstance they cannot *release* dopamine, and 2) feeling safe is what allows for the release of dopamine.

Of course, during these safe activities, the safe feeling automatically displaces the "not safe" feeling of heart inhibition that causes automatic dissociation. When a person feels safe, not only can he *chemically* override the brain-induced immobility from injury and the physical damage and pain at the injury site via dopamine release, he can also override his selective dissociation from his heart. When he overrides the selective dissociation from his heart, his automatic dissociation temporarily ceases, as well.

Of course, as soon as the safe activity ends or the mind is diverted back to the "reality" of some negative thought, the dopamine ceases to flow, the heart dissociation resumes, the symptoms of automatic dissociation resume, and injury-induced movement inhibition resumes.

Maintaining a "safe mindset" that allows for constant dopamine release is almost impossible if a person has a serious injury that needs to be addressed. As soon as a drug-induced or safe activity-induced dopamine surge is over, the dopamine inhibition held in place by the unhealed injury will once again come to the fore. Just because the mind had a temporary surge of dopamine is no reason to expect the injury to heal or the mindset to change. During a safe activity, the foot injury does not heal, nor does the mind re-associate with the foot injury. Again: as soon as the safe activity is over, the symptoms of Parkinson's resume.

But the larger point is this: when PDers are doing their "safe" activities, they never run out of dopamine: they have *plenty* of dopamine. They only stop being able to move easily only when their *minds* remind them that they aren't safe, after all.

What about placebo research?

In the same way, placebo research also proves that most PDers, even those with advanced Parkinson's disease, *do* actually have sufficient levels of dopamine to move perfectly normally. Research findings about the placebo effect on PDers make this glaringly clear. A collection of placebo studies conducted using people with Parkinson's is presented in chapter xxx. Following

administration of a placebo, PDers very often can move normally for a while. They feel safe because they think that something positive is going to happen.

PDers' hours, days, and even a *year* in the case of one placebo study, of "normal" movement, when under the influence of placebos or while doing safe activities, *prove* that a PDer's problem is not caused by dopamine insufficiency. The movement inhibition of Parkinson's is *not* a problem of "not enough dopamine." The real problem is the inhibition of dopamine *release*. When that inhibition is lifted, dopamine flows freely.

Can we prove that dopamine cells are merely dormant?

We have hypothesized that dopamine-producing cells will emerge from their dormancy and resume their role as dopamine-producers if they are correctly stimulated. This assumption comes from the evidence of recovering PDers.

Yes, all PDers can release dopamine in sufficient quantities when they feel safe. However, some of our patients who recovered from Parkinson's did have, over time, a demonstrable *increase* in their amount of dopamine production and reserves.

Some patients, in the early stages of recovery, *if* they did a *significantly* greater amount of activity than usual, would actually *run out* of dopamine. The sensation was bizarre: their brains were calling for a release of dopamine, they were in parasympathetic mode, and yet, they had no ability to do anything, even think. They felt as if they were sleeping while awake, as if someone had turned off their power switch. The experience usually began with a feeling as if the body was becoming extremely heavy, weighing a thousand pounds. Within a few seconds, they would be immobilized.

We named this experience "a crash." These crashes were exactly like the OFF periods that PDers have when their antiparkinson's medications wear off *except* that the nearly-recovered PDers felt absolutely *relaxed* and calm when they became heavy and utterly motionless: as if their bodies were sleeping while the mind was awake. These crashes never lasted more than ten to twenty minutes – the time it takes for the brain to re-circulate dopamine back into dopamine-storage vesicles for re-use. After about ten to twenty minutes of sitting, motionless, on a bus stop bench or some convenient sitting place, feeling about the density of lead, or at least cement, the recovering PDer would suddenly feel a warmth and lightness pervade his body as if he was waking up. Instantly, he would be able to resume healthy, effortless movement.

Just like with the Parkinson's drugs, there was no "slow, medium, and fast" movement," as if dopamine supplies were running low or high. It was an all or nothing situation. Either there was enough dopamine to allow for perfectly normal, effortless movement, or there wasn't. If there wasn't, the PDer could not move, period (unless he ramped up the adrenaline).

The following example will explain what is meant in the above by *significantly* greater amount of activity: if, while recovering, the PDer customarily took a vigorous one-hour walk every day and then came home and rested for a bit, he would not crash. But if, one day, feeling extra good, he went on a vigorous *two*-hour walk, he might experience a crash partway through the second hour.

People who did have crashes usually had them in a predictable manner. For example, if the crash happened after an hour and a half of walking, it might happen after an hour and a half of walking every day for many days in a row. But after a few or many crashes at this exact level of work out, the crashes would cease: the recovering PDer could then walk vigorously for his

two full hours. If, after this, the recovering PDer added yet another hour onto his walk, he might have a crash during the third hour. This might happen once, or maybe twice, but he would even more quickly develop enough reserves to get through hour number three without crashing.

It seemed as if these PDers were developing greater dopamine-reserves. In the early stage of recovery, it almost seemed as if they developed larger stores of dopamine on an as-needed basis. But at some point, there were no more crashes, ever. At this point, it seemed as if dopamine-producing cells must have been continuing to develop *whether or not* they were compelled by crashes.

Within a matter of months after full recovery, recovered PDers stopped having crashes: their dopamine reserve levels had increased to provide *whatever* level of dopamine was needed.

These crashes only occurred in a few of the recovered PDers. *Most* of our recovered PDers never experienced *any* crashes. As soon as they were using dopamine – and the feeling of movement with dopamine was unmistakably different from their PD style of movement, which used adrenaline – their brains were able to provide as much dopamine as was called for.

But the PDers who had crashes when depleting their dopamine reserves were more exciting. They showed us that dopamine reserves *could* be increased. This increase suggested an increase in the number of dopamine-producing cells, or at least an increase in the amount of dopamine that was being produced.

Use it or lose it

Because we saw that increased use of dopamine caused increased dopamine levels, we are in a strong position to argue the reverse: failure to use dopamine will lead to decrease in dopamine production. No pathology needs to be presumed. No germ, gene, or environmental toxin needs to be found. In otherwise healthy people who develop idiopathic Parkinson's disease, the amount of dopamine production, like everything else in the body, is determined on a use-it-or-lose-it basis.

The increase in dopamine reserves, as evidenced by the decreasing number of crashes during recovery helped convince us that PDers did *have* dopamine production capability but they just didn't use it. The placebo studies and the evidence of safe activities just added that much more proof to our hypotheses. The dead dopamine-cell theory cannot make sense of any of the well-known dopamine-override events such as placebo-induced dopamine release and safe activity dopamine release.

But a hypothesis that dopamine release is being mentally *inhibited*, but can be overridden with a change in mental attitude, allows us to make sense out of the evidence.

Is dopamine inhibited because of injury or because of dissociation?

All the PDers in our experience had an unhealed injury causing backwards Qi flow, so they *did have* the mid-brain dopamine inhibition that is supposed to occur with a severe injury. They could not make full use of their substantia-area dopamine producing cells, and very possibly had not have been using them for *decades*.

Also, *many* of the PDers, because they had dissociated from their ability to feel physical and/or emotional pain and were therefore manifesting symptoms of automatic dissociation, *did have* mid-brain dopamine inhibition. They could not make full use of their substantia-area dopamine-producing cells, and very possibly had not been using them for decades.

Whichever the cause, they had not been making regular use of their dopamine reserves.

The body is efficient and gets rid of things that aren't used or needed: in PDers, it seemed as if this lack of use resulted in mid-brain dopamine-producing cells becoming re-undifferentiated. Researchers report evidence of that in the autopsy studies.

When certain brain cells are not used, they revert back to a type of non-specialized cell that resembles an embryonic cell.¹ These non-specialized cells may contain some residue of their dismantled dopamine-building structures and other residues that are *also* sometimes seen in sick or non-functional cells, such as lewy bodies. But these midbrain re-undifferentiated cells are not sick or dead: they just aren't being used.²

WRAPPING UP THE CHAPTERS ON DISSOCIATION

First, there is a time and a place for healthy inhibition of dopamine. Dopamine inhibition is a naturally occurring process. People with Parkinson's have gotten *stuck* in an electrical and/or neurological mode that inhibits dopamine. The electrical inhibition is set in motion by an injury. The neurological inhibition is set in motion by selective dissociation. In order to recover, PDers need to recover from their injury and they need to stop dissociating.

Next, and I will repeat this over and over and over in this book, not *everyone* with Parkinson's disease has selective dissociation from his heart or dissociation from feeling physical or emotional pain. Some PDers recovered from Parkinson's easily, as soon as their foot injuries healed. However, a majority of our PD patients manifested signs of mood- or situation-based parkinsonism after their foot injuries were gone, even after they had experienced many of the unpredicted, counterintuitive, and even bizarre symptoms of physical (as opposed to mental and emotional) recovery.

These *partially-recovered* PDers were unable to visualize light in their bodies, unable to visualize themselves having a functional heart, and unable to imagine themselves moving. They usually did not understand the meaning of the phrase "feel safe." They often thought that "feel

¹ The substantia is supposed to be full of dark-colored cells. I read a research report back in 2002 that stated that the light-colored brain cells in the substantia of people with Parkinson's had characteristics more like embryonic cells and less like differentiated cells. The cells were not dead. They had just become more "immature," less specialized. At the time, it merely confirmed what we'd already started to suspect. I was heartened by it, but didn't think to save it so that I could cite it. At the time, I assumed that much more research would occur in this field, and I could snag as many citations as I needed when the time came. However, a cursory search today on the Internet did not bring anything up. I do wish now that I had put a copy of this particular bit of research into my files. But even without this research that described these cells as reverting to a more embryonic type of cell, evidence abounds as to the fact that the brain cells in the striatum of people with idiopathic Parkinson's disease are *not* dead. The few people who have drug- or toxin-induced parkinsonism have dead brain cells. But the vast majority of people with symptoms of parkinsonism – the people with idiopathic Parkinson's disease – *do not* have dead brain cells.

² Researchers of Alzheimer's disease were disappointed recently when their new drug, which successfully prevents plaque formation in the brain, failed to make any improvement in the mental degeneration of people with Alzheimer's. Because research had shown plaque on the brain cells of people with Alzheimer's, it was fallaciously assumed that the plaque was the *cause* of Alzheimer's. The researchers assumed that, if they got rid of the plaque, the Alzheimer's would go away. But it turned out that the plaque was just cellular debris. The plaque was a side-effect of Alzheimers; Alzheimer's is not a side effect of plaque formation.

Many PD researchers have made the same mistake. They find a reduction in dopamine producing cells, or they find cellular debris where cells have reverted back to immaturity and jettisoned their "mature" dopamine-producing structures, creating lewy bodies and bits of cellular jetsam. But this debris is a side effect, it is not a cause.

safe” meant “be wary:” the exact opposite meaning. Some thought that “safety” meant “silence” or even “lifelessness.” They were unable to feel sensations of vibration and expansion in the chest in response to emotional events or beautiful or glorious sensory events. They often recalled having decided, decades earlier, that they were not going to feel physical or emotional pain.

Some *partially-recovered* PDers *were* able to fully recover. But in order to recover, they needed to be willing to risk feelings of physical and emotional pain. They needed to recognize sympathetic (fear-based) mindsets, including excessively cynical, judgmental (of themselves and others), or overly self-conscious attitudes, and needed to learn how to turn them off.

Many partially-recovered PDers needed to learn what was *meant* by the phrase, “feeling safe.” They also needed to learn how to create the safe feeling in the chest so that they could re-associate with the pain sensations that they’d put on hold for decades. They needed to learn the correct, mature techniques for addressing physical and emotional pain so that their old pains, when they re-associated, could be transmuted into non-threatening sensations and processed by the *conscious* mind.

Strangely enough, very often, as soon as a PDer learned to feel safe, usually via gratitude and surrendering control of his life to a higher power, all the other learning, attitude changing, and pain processing happened *automatically*. Sometimes, it seemed as if their attitudes and dissociations were just temporary poses, and the real personality was just waiting for the opportunity to feel safe so that it could re-emerge.

Then again, some PDers just needed to heal their foot injuries. Or if the foot was technically unhealable, they just needed to restore healthy energy flow through the foot and heal their attitude towards their feet.



“Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forward; and to pass from a walking to a running pace: the senses and intellects being uninjured.”

– the opening definition from *An Essay on the Shaking Palsy*, by James Parkinson, member of the Royal College of Surgeons, 1817.

CHAPTER FOURTEEN

THE WESTERN UNDERSTANDING OF PARKINSON'S DISEASE

This chapter will explain idiopathic Parkinson's from the current western perspective: symptom descriptions, diagnosing, current treatments, and where the western research might be heading.

It may become very important for a PDer who starts recovering to have the clearest possible understanding of the classic symptoms of Parkinson's. Only then can he tell the difference between Parkinson's symptoms and counterintuitive, often unpleasant symptoms that can occur during recovery.

Therefore, this chapter will start with a quick overview of the syndrome and then follow up with more detailed descriptions of PD symptoms. These descriptions are based on lectures, articles, various medical journals, Medscape's neurology weekly reports, books, the Internet and that medical standard, the Merck Manual. After that comes a section about diagnosing Parkinson's, then one on the current western treatments for Parkinson's disease: antiparkinson's drugs; brain implants; and stem cell research.

Please note – the symptoms listed throughout this chapter are those of unmedicated Parkinson's disease. Those patients who are taking antiparkinson's medication may exhibit a wide range of symptoms including ticcing, spasming, personality or mood change, and even psychotic behavior, all of which may be referred to by the patient – and even the uninformed doctor – as Parkinson's symptoms, but which are, in fact, short- and long-term effects of the medication.

OVERVIEW

In 1998, when I started my investigation, the following were accepted explanations of Parkinson's disease:

“Parkinson's disease (PD) is named for an Englishman, James Parkinson, who first wrote up a description of the syndrome. Parkinson's is the second most common neurological disorder in the world after Alzheimer's.”¹

¹ James Tetrad, MD, director of the Parkinson's Institute of Sunnyvale, California. From a lecture at the Santa Cruz Parkinson's Support Group, March 1998.

From the Merck Manual, I read the following: “[Parkinson’s disease] is an idiopathic (cause: unknown), slowly progressive, degenerative central nervous system disorder with four cardinal features: resting tremor, slowness and poverty of movement, muscular rigidity and postural instability. Although symptoms initially develop on one side of the body, they eventually become bilateral. In the advanced stage, the patient can suffer complete rigidity and immobility. Dementia occurs in about 50% of patients.¹ Depression is also common.

“The disease is characterized by loss of the pigmented neurons involved in controlling movement, which are located in the substantia nigra, locus ceruleus and other brainstem dopaminergic cell groups. This loss of neurons results in depletion of the neurotransmitter dopamine. The cause of the neuron loss is unknown, but it now appears not to be genetic, but rather induced through an as yet unknown external factor.”²

¹ The numbers on dementia in this reference are misleading. Dementia most often occurs in patients who are diagnosed with Parkinson’s late in life. The dementia usually begins about ten years after diagnosis. In other words, the dementia of Parkinson’s may well be the same, and seen in the same frequency, as the dementia of very old age. Part of the misunderstanding about the frequency of dementia, a symptom that strikes fear into the hearts of the recently diagnosed, is due to the way that all psychoses are sometimes lumped together under the heading “dementia.”

As noted in the *Parkinson Report*, Fall 2000, in an article “Hallucination and Psychosis in Parkinson’s disease,” Goetz, MD, “Although there were rare reports of hallucinations and delusions in medication-free Parkinson’s disease (PD) patients prior to the advent of effective drug therapy, these cases are exceptionally rare.” He goes on to point out that these problems arise in PDers in response to dopaminergic therapy (dopamine-enhancing drugs). He adds that, in some cases, psychosis can occur from complications of infection, dehydration, or drug toxicity. In other words, the psychoses are not due to Parkinson’s disease, per se.

For specific numbers on dementia, I quote from *Parkinson’s Disease, Questions and Answers*, by Hauser and Zesiewicz, Merit Publishing, Florida, 2000, p. 29: “Reported prevalence rates [of dementia] range from 10% to 80%, but actual rates are probably closer to 15% to 30%. In a series of 155 PD patients, 8% had severe dementia... In one sample of 139 PD patients in Norway, at least one “psychiatric” symptom was reported in 61% of patients. The most common psychiatric manifestations are depression (38%) and hallucinations (27%).”

From the above, the reader can see that dementia is often lumped in with other symptoms, many of which are drug related. This suggests that the true numbers for dementia are much, much lower than the 50% mentioned in the Merck Manual. James Parkinson, who admittedly worked with a much smaller sample size, was careful to make the point that “the senses and intellect remain uninjured.” I personally remember reading in the 1960s, before dopamine-enhancing medications were the norm, that the tragedy of Parkinson’s was having a completely alert mind trapped in an inert body.

² Berkow R (ed): *The Merck Manual*, Merck Sharp & Dohme Research Laboratories. Rahway NJ, 1992, pp. 495-500.

The illness is not genetic, except in the very rare familial form of a Parkinson’s-like disorder in the Contursi family. As for research looking for a genetic connection in Parkinson’s, a large, nationwide identical twin study of PD patients in the late 1990s came to the rare conclusion that if one identical twin has PD, the other is less likely than the national average to have PD. This indicates that PD is not genetic. More interestingly, it suggests some protective benefit from having a twin.

Considering that PD is often seen to run in families, we can make this hypothesis: the attitudes of “stiff upper lip” and stoicism that can be passed along from one generation to the next as family values or behaviors can contribute to the occurrence of Parkinson’s disease.

However, in an identical twin situation, one twin is usually more dominant, more protective of the other. In such a situation, even if the family tendency is towards a cold disdain of showing pain or emotion, the intimate relationship of the identicals may provide a safe haven, at least for the subordinate twin, from the emotionally rigid behaviors of the rest of the family. If the young subordinate identical is injured, he may hide it from the rest of his family, but he may avail himself of the sympathetic ear, maybe even a hug, from his closest sibling. The dominant identical may provide succor for his womb-mate in a family dynamic that otherwise would be hostile to the expressing of emotions. (Continued on next page.)

Signs and Symptoms

“A patient with PD may present with from three to all four of the variants of the symptoms below at the time of diagnosis. As symptoms progress, a patient may become wheelchair bound. PD is not fatal, but increased mortality occurs because of debility, aspiration pneumonia, and infections.”¹

The following are the four categories of Parkinson’s disease symptoms. I call them the Big Four.

1) Resting Tremor: The tremor is seen in 50% to 80%² of patients. The Parkinsonian tremor, in the early stage, is a "resting tremor," occurring when the affected body part is inactive: at rest.³

2) Poverty of Movement: “Bradykinesia [slowness and poverty of movement], akinesia [difficulty in initiating movement], and a reduction in automatic movements such as alternate arm swinging while walking”⁴ are characteristic symptoms.

3) Muscular Rigidity: Rigidity at the wrists, ankles, shoulders, and hips prevents smooth flow of movement; attempts at rotating the wrists and ankles result in jerky, "cogwheel" motions.⁵ The rigidity and slowness of movement combine to create a shuffling, labored gait.

4) Postural Instability: The increasing rigidity in the legs, loss of balance and coordination, and imbalance between left and right, combined with the postural forward stoop, lead to a tendency to fall forward.

Therefore, this genetics study, considered to be a failure in that it did not find a genetic connection, may in fact be an important lead. Certainly, the unheard of statistic that a person with an identical twin with an illness is less likely than average to have his sib’s syndrome suggests that something sociological is going on in Parkinson’s.

¹ The phrase “from three to all four” means that a person may possibly be diagnosed with PD even if all he has is one symptom from at least three of the four categories. As the disease progresses, he may have many symptoms from each category, but he must still have symptoms in three of the four categories for it to be diagnosed as Parkinson’s. The quote is from *Cecil’s Textbook of Medicine*, Wyngaard JB, Smith Jr LH, WB Saunders Co., Philadelphia, 1988, p. 2144.

² The percent of PDers that have tremor varies depending on which medical books or articles you read. There is no consensus.

³Tierney LM Jr, McPhee ST, Papadakis MA: *Current Medical Diagnosis and Treatment* (ed 35). Appleton & Lange, Stamford CT, 1996, pp 885-887.

⁴ Ibid.

⁵ Whoever named the uneven rotational movement of PDers’ wrists and ankles got his nomenclature wrong. A “cogwheel” is a smoothly-turning toothed gear. The word the writer was probably looking for was “camwheel.” A cam is a wheel that has one irregular lump on its circumference. As the turning camwheel rotates over the lump, it causes a pause and a thud in the circular motion of the wheel. This corresponds to the pause and skip that may occur when a PDer rotates the ankle or wrist.

Despite the original error, this uneven movement in PDers is now officially referred to as “cogwheeling.”

The above was the essence of understanding Parkinson's disease when I started the Little Project in 1998, and still is. What I call "The Big Four," the four categories of symptoms above, were derived from the work of James Parkinson and are still used today to determine a diagnosis of PD.

However, there are more details about symptoms that can help flesh out the description. These details are usually listed as being "in addition to" the Big Four, but most western-recognized symptoms are, in fact, derivatives of the Big Four.

Example of a Big Four derivative

For example, I found the following in a book on PD, after it listed the Big Four: "Another symptom of PD can be 'foot drop'." This symptom is actually not a separate symptom, but a derivative of poverty of movement. However, because the generalized Big Four does not always include many details, sometimes lists of details are included in books about PD. Often, these details about the symptoms make it appear as if these detailed symptoms are separate from the Big Four. However, no matter how many details are added in, it turns out, upon close examination, that these detailed symptoms for the most part are still derivatives of the Big Four: tremor, poverty of movement, rigidity, and poor balance.

Foot drop is an aspect of "poverty of movement"

Let's look more closely at the Parkinson's symptom called foot drop to see how it fits into the Big Four category system. During normal walking, the ball or toes of the affected foot may intermittently fail to be lifted clear of the floor during a normal stride – it can feel as if the foot sticks to the floor. The "stuck" foot causes the forward-moving patient to shuffle, or even to trip, falling face first. The falling is a symptom in the category of "postural instability," also known as "losing one's balance."

Patients may attempt to recover balance after a foot drop, but, now and then, a randomly occurring, utter inability to initiate large (normal) strides may result in a desperate attempt at walking that uses a multitude of tiny, labored, rapid steps, also known as "baby steps" or "festinating gait."

The tiny rapid shuffling steps of festinating gait are a (usually hopeless) attempt by the feet to catch up with the torso, which is still hurtling forward at the previously established speed. The torso is being propelled forward by momentum and downwards by gravity. When the slow-moving, baby-stepping feet fail to keep up with the forward moving torso, the upper body plummets to the ground or crashes into nearby objects, such as the wall. This forward and downward movement of the torso, accompanied by frantic, hurried-but-tiny footsteps that fail to catch up to the upper body, is called a festinating gait.

Though they may be listed as additional symptoms, foot drop and festinating gait are members of the Big Four of tremor, poverty of movement, rigidity, and postural instability: because of the falling and stumbling involved with the festinating gait, this problem fits into the postural instability category. Because this problem includes slowness of the footfall, inability to lift the foot off the floor (foot sticking) and slowness in making small subconscious movements that are needed to correct for imbalance, the problems of foot drop, small steps, and losing balance also fit the poverty of movement category.

This example simply shows that, although many "extra" or "additional" symptoms are sometimes listed in addition to the Big Four, these extra symptoms are actually Big Four derivatives.

The need to be informed

It is important that a person setting out to recover from Parkinson's disease has a very good understanding of what defines Parkinson's. If one understands the extent to which the recognized symptoms of Parkinson's are variations on the Big Four, one needn't memorize lists of seemingly unrelated symptoms, but need only understand the principles involved. Since every individual's Parkinson's disease manifests slightly differently, it makes more sense to understand the principles rather than a long list of symptoms which may or may not apply to any given PDer.

Recovery misunderstandings based on a TV show

As an example of how one's misunderstanding of the symptoms can lead to unnecessary worries during recovery, let me include part of a recent case study: I had one patient who, during recovery, was frightened by new sensations in her arm and the slow, rhythmic muscle extensions and flexions that spontaneously moved her bicep muscles when energy started returning to her left side. She became certain that tingling in her arms from increased sensation and improved range of movement were new symptoms of Parkinson's disease!

She was further convinced that her new ability to move easily was sign of worsening Parkinson's after seeing Michael J. Fox speaking to congress: in a televised program, Michael J. Fox stated for the cameras that his wildly flailing, dyskinetic arms (symptoms set in motion by *overmedication*) was what happens with his Parkinson's when the medications don't work. This statement may have been intentionally misleading. This was filmed during his successful attempt to show congress that more research money was needed for Parkinson's disease.¹

Misunderstandings based on observing overmedicated PDers

Others misunderstand the true nature of Parkinson's because they know someone who is medicated who "moves like a crazy person and doesn't know what he's doing half the time." Because of the rampant misunderstanding of the true nature of Parkinson's, based on uninformed people's experiences with *overmedicated* PDers, it is extremely important that I drive home the idea that Parkinson's is a syndrome marked by *decrease* in motor function: *less* movement, not more; *rigidity* over most of the neck, torso and legs, not limpness; *hesitancy and stiffness*, not rapid performance and increasing range of movement.

When the strange symptoms of recovery begin, it will be extremely important for morale that the ex-PDer has a good grasp of what constitutes symptoms of Parkinson's and what does not. Only in this way can one understand that the recovery symptoms, though annoying, sometimes painful, and even bizarre, are the exact opposite of Parkinson's symptoms. So now, back to the symptoms of PD.

¹ Why on earth, you may be asking, should Mr. Fox present symptoms of *overmedication* as if they were symptoms of Parkinson's? One reason might be that it makes for much better drama: a person whose medications aren't working is most likely to be hunched, drooling, not moving, and possibly even incapable of speech: not very dramatic or romantic. A charming TV actor might not want the public to see such a pathetic image.

Therefore, the TV presentation was possibly calculated to show something highly alarming: the wild, uncontrolled movements that can occur when a person's medications are grossly excessive. Mr. Fox's statement, in order to be correct, should have been "this is what happens when the medications don't work correctly, *due to excess dosage*."

However, the harm has been done. Millions of Americans now believe that Parkinson's disease is a disease of spontaneous, uncontrolled muscle thrashing.

More details about the Big Four

Again, while many of the symptoms listed in these headings may be listed separately in some books, one attains a greater understanding of the illness if one sees the relationship between the many symptoms and the four basic categories.

1. Poverty of Movement

Slowness, also called bradykinesia, and difficulty in initiating movement (akinesia) and a reduction in automatic movements such as alternate arm swinging while walking are characteristic PD symptoms.¹ While the arm swing may be consciously forced, temporarily, the arm will stop swinging as soon as the conscious effort ceases. Other symptoms include lack of coordination between arm swing (if any) and stride. Slow, shuffling steps, slow hand and/or arm movements, slow, muffled speech, and slowness in performing coordinated finger movements such as cutting up food, doing up buttons and picking up coins are forms of bradykinesia.

Micrographia, the extremely small, slow, and labored handwriting, is a form of poverty of movement, and can be characteristic of PD.

Unblinking eyes, sagging or useless facial muscles, inability to smile, poor swallow reflex/excess salivation, and inability to move the middle toes are all symptoms of poverty of movement.

2. Rigidity

There are two primary lines of rigidity. One starts at the back of the jaw, then goes down the front edge of the neck's sternocleidomastoid muscle, over the mammary line down the torso, crossing from the abdomen to the outside of the hip, continuing downward along the front (anterior) – outside (lateral) aspect of the legs down to the ankles and stops at the top of the foot. This line corresponds to the jaw-to-midfoot portion of the Stomach channel.

This rigidity makes it difficult to turn the neck from side to side or look behind when driving. The tightening along the front-side of the neck and over the clavicle pulls the shoulders forward into the classic hunched posture of Parkinson's. This type of tightening pulls the head forward and downward, as if the neck is shortening. The distance between the earlobes and the shoulders decreases.

It can look as if the shoulders are pushing upwards to reach the earlobes. This example may give you a good visual sense of this: many women have noticed that for several years prior to their diagnosis with Parkinson's they could no longer wear dangling earrings; the same earrings that used to dangle in space reaching halfway to their epaulets would now rest, slumping, on their raised shoulders.

Rigidity along the torso makes turning in bed more difficult. Ordinarily, a person trying to turn over in bed moves his shoulders, and then the torso, and finally the hips and legs. When the neck, torso, and hips start to move as a rigid unit, one must wrench the whole mass, from neck down to hips, as one piece. This leads, in the beginning, to the belief that one's mattress is not firm enough. After replacing the mattress and realizing that the bed was not the problem, people use a variety of methods to turn over: using the headboard for leverage, bringing the knees up and shoulders forward, making the body as compact as possible before pushing off

¹ LM Jr. McPhee ST, Papadakis MA: *Current Medical Diagnosis and Treatment* (ed 35), Appleton and Lange, Stamford, CT, 1996, pp 885-557.

against the bed with the shoulders or hips and heaving the whole unit over in one move, or asking the spouse to give them a shove.

This rigidity in the muscles that run over the collarbone may also make it stressful, painful, or impossible to raise the arms over the head for an extended period.

Another line of rigidity extends from the point on the wrist crease on the dorsum (the back, not the inside) of the wrist nearest to the junction of the index finger and thumb, and travels upwards past the outer end of the elbow crease, over the bicep, across the front-top of the shoulder, and over the neck to the side of the mouth.

This path corresponds to the above-wrist portion of the Large Intestine channel.

Rigidity at the wrists and ankles prevents a smooth flow of movement when making circles with the wrist or ankle; rotation of the limbs at these joints results in a jerky, “cogwheel” motion. Instead of rotating in a smooth circle, the ankle or wrist rotation motion features a pause and a skip in the vicinity of the thumb section of the wrist, or the front and anterior/lateral portion of the ankle. This pause and skip is due to rigidity in these two, very specific areas.

As Parkinson’s worsens, the increasing slowness and increasing rigidity combine to create a shuffling, labored gait and extreme constraint of movement, and difficulty turning to the side (moving the legs in the anteriolateral direction) when walking.

3. Tremor

The tremor of Parkinson’s often is, for the first few years at least, a “resting tremor.” Resting tremor occurs when the tremory limb is inactive, at rest. In other words, although an index finger may tremor against the thumb when a person is sitting still, activities using the hands will make the tremor stop. Once the limb is at rest, the tremor starts up again. Over time (months or decades), the tremor may worsen so that it occurs even during activity. It may get worse during times of stress or when trying to eat.

The most common form of tremor is the classic “pill rolling” tremor of the hand, in which the index finger rests briefly on the thumb and then bounces off the thumb at 4 to 8 cycles per second.

Sometimes the tremor extends up from the hand and involves the arm. A less common form of hand tremor occurs in the third and fourth finger. This tremor may cause the stiffened digits to vibrate in a fluttering motion or else make a back and forth motion at the wrist.

Tremor may occur in the lower limbs. Tremor can manifest in the neck or jaw. “Although it may ultimately be present in all limbs, the tremor is commonly confined to one limb or to the limbs on one side for months or years before it becomes more generalized.”¹

In times of calm, the tremor is a small quavering movement. A larger, back and forth, semi-rhythmic, involuntary movement with some power behind it can occur during times of stress or anxiety. This larger movement looks like an extreme exaggeration of the quavery, vibrating resting tremor.

4. Postural Instability

In addition to the festinating gait discussed earlier, many people with Parkinson’s have a tendency to fall. Some fall mostly forwards, some fall mostly backwards, some teeter from side

¹ LM Jr. McPhee ST, Papadakis MA: *Current Medical Diagnosis and Treatment* (ed 35), Appleton and Lange, Stamford, CT, 1996, pp. 885-557.

to side, and some find that, when walking, they tend to crash unpredictably into walls and furniture.

Most of these falls stem from the inability to send quick enough mental instructions to those muscles that are supposed to make tiny, balancing compensations: the brain can no longer initiate these automatic body balancing movements subconsciously. This inability to make movement corrections automatically, subconsciously, combined with extreme slowness of muscle response, may lead to frequent falls. A healthy person can, without even thinking, throw out an arm, leg, hip or neck to correct for some unbalanced movement. People with Parkinson's cannot make these quick, automatic movements. Their movements become increasing conscious efforts, and increasingly slow. As a result, the slightest off-center teetering is likely to lead to a fall.¹

This inability to compensate subconsciously is best demonstrated by the balance test that some doctors use for confirming a diagnosis of Parkinson's: the candidate stands with his back to the wall, about three inches away from the wall. The tester gives a quick nudge, or tap, on the shoulder, as if gently pushing the candidate towards the wall.

A healthy person will easily and automatically compensate for the nudge by moving the shoulders, arms, waist, hip, knees, and/or feet, in what are practically invisible movements, in such a way as to prevent falling backwards. A person with Parkinson's may go straight back, thudding into the wall, unable to stop himself. The test is most effective when the shoulder tap is done on the Side of the body where Symptoms First Appeared (SSFA).

Again, as a reminder, all of these symptoms are based on people with Parkinson's who are *not* taking medications. The frequent falls that occur when, due to medication, a person feels impervious to harm are somewhat different from the falls that occur in unmedicated Parkinson's.

FORMING A DIAGNOSIS

The official western medicine position on diagnosing Parkinson's is that a diagnosis of Parkinson's disease cannot be actually confirmed. There is an understanding honored by most MDs that, in order for a person to be diagnosed with Parkinson's, a person must present with symptoms from three of the four main symptoms categories (the Big Four). Again, the four categories are: poverty of movement (also called bradykinesia), rigidity, resting tremor, and poor balance. If a person has symptoms in only two categories, the understanding is that there should be several types of problems in both of those two categories before a diagnosis of Parkinson's can be made. Also, if symptoms from only two categories are seen, neither of these categories should be the postural instability. The most important category is the poverty of movement, slowness.

¹ Many well-meaning physical therapists teach classes in how to keep people with Parkinson's from falling. These classes are pretty much worthless if they try to teach PDers the importance of bending at the knees, leaning in the opposite direction, or any movement related technique. A person with Parkinson's, on the way down to the floor, usually cannot execute a conscious movement such as "I shall drop to my knees." For him to so drop will take a massive amount of conscious effort and the eventual movement so generated may not occur until long after he has already hit the ground. The most helpful advice from these classes is advice oriented towards emphasis on using a good walker or sharing the name of someone who you can hire to install safety bars in the bathroom. Well-meaning advice on the best ways *to move* in order to keep from falling, such as, "try to roll with it, break the impact of the fall," simply miss the point – these people can't move quickly enough to perform counter-actions, no matter how well planned they are.

A common misconception among the general public is that “anything that tremors is Parkinson’s disease.” This is not true. Many illnesses, ranging from blood sugar disorders to heart disease to post-polio syndrome, may cause tremor. Also, other tremor-specific disorders such as familial tremor or essential tremor are not related to Parkinson’s disease.

The list of known side effects of many drugs, especially the antidepressant and anti-anxiety drugs, include tremor or tardive tremor. “Tardive” means “shows up later.” The tremor from legal or illegal drug use or abuse may not even appear until decades after the user has stopped taking the drugs.¹ Therefore, just tremor, without symptoms from other categories, does *not* support a diagnosis of Parkinson’s disease. However, many people – and even some poorly informed MDs – do not realize this. They imagine that anything that tremors must be Parkinson’s. These people are not correct. Again, many syndromes include tremor.

Because of the uncertainty in a PD diagnosis, neurologists will usually request a brain scan of a person in whom PD is suspected. The brain scan cannot confirm a diagnosis of Parkinson’s; the scan is to rule out the possibility of a stroke or a tumor, events that can sometimes create symptoms similar to those of Parkinson’s. Both stroke (bleeding or blood clot in the brain) and tumor show up nicely in a brain scan – Parkinson’s does not. Therefore, if a person has several Parkinson’s symptoms in three of the four categories *and* no obvious sign of tumor or cerebral trauma, the doctor may give a diagnosis of Parkinson’s by default: no other diagnosis presents itself.

Atypical and non-classic Parkinson’s

If one’s doctor should say that one doesn’t have classic Parkinson’s, or has “atypical Parkinson’s,” bear this in mind: classical Parkinson’s takes time to develop. A person may have early Parkinson’s that does not *yet* look classic. However, with a degenerative disorder, it may be just a matter of time. Because Parkinson’s is degenerative, trying to diagnose it early, especially when the symptoms are still intermittent, is trying to hit a moving target. On the other hand, “atypical” may mean that one has all the symptoms of classic Parkinson’s *plus* some other symptoms that might indicate another problem is present at the same time. Most doctors do not bother to go much more deeply into the matter than whether or not they can fit a PD-ish label on it. They never, in our experience, bother to differentiate between drug- or toxin-induced parkinsonism and idiopathic Parkinson’s disease.

MISDIAGNOSIS OF PARKINSON’S DISEASE

Misdiagnosis in Parkinson’s disease is notoriously rampant. Even Parkinson’s specialists sometimes argue amongst themselves as to whether or not a particular person actually has idiopathic Parkinson’s. Aside from the basic definition of PD put forward by James Parkinson in 1817, there is currently much disagreement about what, technically, constitutes Parkinson’s disease.

¹ I spoke with a doctor who works primarily with VA in-house patients. He told me that thirty years ago, about 4% of the vets had tremor. Since the late 1990s, a majority of the vets have some form of tremor. He attributes this dramatic increase in tremor to the aging of vets who were given methamphetamines during WWII and the Korean war. The methamphetamines, which are dopamine-enhancing drugs, were usually used to help soldiers and especially pilots stay alert when sleep was not an option. Methamphetamine use is known to cause a tardive tremor that may not manifest for decades, or even until old age – long after the drug usage stopped.

Misdiagnosis discovered in autopsy studies

Depending on which study you read, somewhere between 25% and 30% of the people diagnosed with Parkinson's disease do not actually have PD. These numbers have been generated by various autopsy studies, in which it was found, during autopsy, that a supposed PDer had no Parkinson's-like brain cell modifications. In our own clinic, approximately 30% of the supposed PDers that came looking for treatment did not even begin to fit the standard, western medicine description of Parkinson's disease.

Differing opinions

We've had patients who went from one doctor to another, trying to get a firm diagnosis. One of our patients was told that he certainly had PD by one neurologist, and two subsequent neurologists said that they could not possibly support a diagnosis of Parkinson's disease. In this patient's case, because his family wanted a diagnosis of Parkinson's and the other two neuros weren't sure what he had but suspected Alzheimer's, the family decided to go with the first doctor, who insisted that he take antiparkinson's medications immediately. The medications did not help. In fact, his main problem, which was confusion leading to slow responses, rapidly worsened – probably due to the mind-altering properties of his medication.

Also, we have met a few patients whose misdiagnosis of PD by their neurologists might constitute acts of gross negligence or even malpractice.¹ As an aside, when we examined these glaringly misdiagnosed people, they clearly did *not* have a Qi irregularity in the leg.

PET scan controversy

Even the new PET scans, which can reveal areas of diminished dopamine receptor activity in the brain, but which do not measure dopamine levels or show changes in the substantia nigra cells, are *not* accepted as definitive by all neurologists. In fact, the scans have added a new level of complexity to the discussion: in one study, 14% of the people who had been

¹ In one class that I was teaching, one patient stood out. Her only symptom was a weak arm that didn't swing at all. Three years earlier, she had hurt her arm at work. She had woken up the morning after hurting her arm with a right arm that didn't swing and severe weakness in the fingers of her right hand. She was thirty-six years old. She had no other symptoms of Parkinson's. She saw a neurologist that week. The doctor told her that she must start dopamine agonist medication immediately or she would get worse. She had been taking the medication (at a very low level, because it had not worked at the higher level but she thought she should take something) for three years when she came to my class. It hadn't helped her arm to swing. She realized, looking around the classroom, that what she had didn't match what all the other patients had, in symptoms or in personality. The second day of class, after all the others had spoken in turn about themselves and their symptoms, she stated her case: "I'm not like the rest of you here. You have a certain way about you; I don't mean your symptoms. I'm just a girl with a bad marriage who works in a pub. I don't fit in here."

All the students in the class were able to confirm that the Qi was *not* running backwards in her legs.

After a one-hour treatment session with her the next day, during which I repositioned her arm in the shoulder socket – after which it swung normally – we determined that her problem had been a displaced arm caused by having improperly lifted a heavy basket of chips out of the fryer at work.

Another patient who was grossly misdiagnosed to the point of malpractice had a similar situation, except that his forearm immobility started when his "tennis elbow" surgery failed to heal correctly. The forearm hung limp, useless, trembling after the surgery. The surgeon told him that the operation had been a success, but that he could no longer move his forearm due to the overnight appearance of Parkinson's. Fifteen years later, his only so-called Parkinson's symptom was the unchanged immobility and trembling of the one forearm; he had lived fifteen years in dread of the sudden appearance of the other PD symptoms, none of which had ever appeared.

confirmed by a *panel* of Parkinson's *MD specialists* as having PD had PET scans that were perfectly normal. Does this mean that the doctors were wrong, or the scans?

I also had a patient who had tremor and no other symptoms of Parkinson's disease. Her western trained doctor, like me, was certain that she did not have Parkinson's disease. However, she was adamant that she needed a PET scan. The scan showed reduced dopamine receptor function. That was over a year ago. She still shows no signs of Parkinson's disease other than a tremor.

PET scans do not provide a definitive diagnosis of Parkinson's disease. In fact, their use is increasingly controversial.

As Dr. William Weiner, MD, Chair of the department of Neurology at the University of Maryland Medical Center, said in an interview with *Neurology Today*, "If I saw a patient who I thought had parkinsonism, and I sent him out for a scan and it [the scan] came back perfectly normal, I wouldn't change my diagnosis."¹

The L-dopa test

Recently, despite the fact that nearly anyone who is moving slowly for nearly any reason, including pain or depression, will move better under the influence of L-dopa (a powerful, mind-altering, mood-altering drug), some uninformed doctors have been using L-dopa as a test for Parkinson's disease. If a person with some Parkinson-like symptoms responds to L-dopa, these misguided doctors feel that their diagnosis of PD has been confirmed.

This specious reasoning ignores the fact that many disorders, not just PD, respond well to L-dopa. It also ignores the fact that drug-induced parkinsonism, a PD look-alike that can be triggered by many antianxiety and antidepressant drugs as well as many of the illegal mind-altering drugs sold on the street, will respond very nicely to L-dopa. However, in this latter case, if there is no underlying idiopathic Parkinson's disease, the L-dopa will not only mask the presenting symptoms, it can accelerate the permanent brain damage caused by previous drug use.

The final irony of testing for Parkinson's by using L-dopa is this: people with idiopathic Parkinson's usually do not respond to the medications for several weeks. If the drugs are dosed correctly, as the L-dopa manufacturers point out in their drug inserts, the full benefit of L-dopa may not be evident for even up to ten weeks. However, in a person who has some illness other than Parkinson's, such as drug-induced parkinsonism, depression, exhaustion, chronic fatigue syndrome, or any other illness in which dopamine may be temporarily reduced but the overall dopamine system (the dopamine receptors, dopamine transport molecules, dopamine reuptake enzymes) is still functional, the L-dopa might work very quickly. In idiopathic Parkinson's, the entire dopamine system is somewhat dormant and slow to respond to medication.

In other words, a rapid response to L-dopa might indicate that the person does *not* have idiopathic Parkinson's. A person with idiopathic Parkinson's will respond to L-dopa, but only after the medication has begun to accumulate in the brain. If correct dosage levels – levels that accumulate slowly over ten weeks – are used, a person with idiopathic PD may not have a response to L-dopa for several weeks. Yet, increasingly, doctors with little understanding of the illness or of how the drugs work are using a rapid positive response to high levels of L-dopa as proof of Parkinson's. Some of them give the patient a very, very high dose of L-dopa to "test" for Parkinson's. In these cases, the PDer may notice some mild benefit within a few days. Then

¹ "Study Examines Role of Imaging in Diagnosing Parkinson's Disease," *Neurology Today*, Aug. 2005, p. 47-48.

again, so will nearly anyone else. Also, PDers are particularly susceptible to the placebo response. Hopefully, this fad of making a diagnosis of idiopathic PD on the basis of a rapid (within a day or two) positive response to pharmaceutical dopamine – a fad unsupported by any good research – will soon fade.

Gender bias

We also learned that the motor problems of our male PDers had been immediately acknowledged by their MDs. Women, on the other hand, were often told that their slowdown of motor function, tremor, or rigidity was due to depression or dissatisfaction with life. Many of our female PDers said that it took several visits to the neurologist, spread over several years, before the good doctor admitted that a neuro-motor problem existed.¹

Incompetence

In addition to all this, it must sadly be admitted that many neurologists are incompetent to make an informed diagnosis of Parkinson's. Many cases of PD misdiagnosis are clear to the naked eye. The misdiagnoses run both ways: we saw one "PDer" who clearly had nothing worse than a bad outcome from a knee surgery. We saw another woman who had long had all the symptoms of Parkinson's but whose neurologist had, for ten years, refused to give her a diagnosis of Parkinson's, because, despite her rigidity, poverty of movement, balance problems, and tremor, she was still able to force a smile; this neurologist (wrongly) considered a frozen face and only a frozen face to be the gold standard for diagnosing Parkinson's disease.²

Drug- or toxin-induced Parkinsonism

Some patients have come to us with a long history of using antidepressant or anti-anxiety drugs and sometimes a large history of recreational drug use as well. Very often, their facial twitching and various spasms do not resemble in the slightest the classic tremors of Parkinson's, and, aside from these twitchings and ticcings, they have no classic symptoms of PD. And yet,

¹ One internationally renowned business-woman, only forty years old, with symptoms of fairly advanced Parkinson's (she was using a walker when I met her), was told by her neurologist that, if she would just get married, all her problems would go away. She did actually marry less than a year later. Her symptoms continued to worsen.

² I recall the case of the neurologist who performed the Babinski reflex test (testing the foot's response to a finger stroke on the sole of the foot) on a patient, in order to confirm a diagnosis of PD, and wrote up in his report that he got a negative result – and the patient had his hard-soled shoes on the whole time! The doctor had "tested" the soles of his *shoes* instead of the soles of his feet while looking for a toe-curl response!

Often, in these blatant cases of misdiagnosis, the "cure" to the actual problem is a simple one. Other times, the problem is completely baffling but, nevertheless, is not consistent with a diagnosis of idiopathic Parkinson's disease. Many times, doctors skirt the whole issue by declaring the person to have some kind of "parkinsonism," and then offering drugs for the illness as if it were Parkinson's disease. This is unconscionable: the drugs, when taken by a person who does not have idiopathic Parkinson's disease, can rapidly cause a decline in brain function, and do permanent brain damage. Usually, those people who rapidly develop dyskinesias from the drugs or who need rapid increase in dosage due to addiction or to compensate for rapid development of side-effects are people who do *not* have idiopathic Parkinson's disease. Their problems with the drugs are due to the fact that they did *not* have idiopathic Parkinson's disease. Only people with idiopathic PD or subclinical (not yet obvious) idiopathic PD can actually tolerate the drugs without having over-rapid appearance of side effects. Even PDers only do well with the drugs *if they are dosed correctly* – a remote contingency indeed, based on the hundreds of prescriptions we have seen.

they were given a diagnosis of Parkinson's disease by their neurologists. Interestingly, these patients never had backwards-running Qi in their legs.

Self-diagnosis

Self-diagnosis is very often misdiagnosis. Many illnesses can create some symptoms that seem similar to the written descriptions of Parkinson's and yet, to the trained eye, they are clearly not Parkinson's disease. Many of the self-diagnosed patients we have met did not come within a kilometer of actually having idiopathic Parkinson's disease.

I recall one obese patient with poor diet who had not taken any regular exercise since she was in her 30's, who decided that she had Parkinson's disease because, at age 86, her swollen legs moved slowly (although she talked a mile a minute and gestured very, very rapidly as she spoke). Also, she was increasingly stiff in the morning and had trouble turning over in bed. Furthermore, her arthritic hands caused her to write very slowly. She was adamant that I diagnose her with Parkinson's disease so that I could "fix" these problems. She had stumbled across my work and was so pleased to learn that there was a cure for her "condition." Her doctor agreed with me; she did not have even a hint of Parkinson's disease. She didn't believe him, either.

TREATMENT

Drugs

When I started my research in the 1990s, pharmaceutical (drug) treatment for Parkinson's disease consisted of a dopamine precursor (L-dopa), dopamine agonists (dopamine act-alike molecules), anticholinergics (drugs that stop acetylcholine, the neurotransmitter that transmits brain signals to muscles) and drugs that inhibited the breakdown of dopamine (MAO inhibitors). Shortly after I started this study, drugs that prevent the breakdown of L-dopa in the digestive tract and/or bloodstream were added to the list.

Although most MDs do not realize that the various drugs are best used for specific symptoms, the drugs do have different results. For example, if motor function is still good and anxiety-related tremor is the primary problem, anticholinergic drugs might be used to reduce the tremors by the mechanism of sedating mental and motor function.

If poverty of movement rather than tremor is the most problematic symptom, dopaminergic medications might be a better choice.

Dopaminergic drugs can have many adverse effects. Some adverse effects of the dopaminergic drugs (L-dopa, dopamine agonists, MAO inhibitors) are dyskinesia (erratic, uncontrolled and excessive movement), dystonia (excess muscle tension), insomnia, irregular heartbeat, and mind and mood alterations.

More problematically, these drugs are highly addictive and can cause death of dopamine-producing cells (parkinsonism) and a decline in the number of active dopamine receptors.¹

¹ The direct relationship between the amount of L-dopa dose and a decline in dopamine receptors was proven in the Elldopa study of 2003. For a discussion of the way that this finding was downplayed in the study, a study conducted in part by an employee of a company that is paid to test drugs for FDA approval, please see "Levodopa and the Progression of Parkinson's Disease," *New England Journal of Medicine*, March 31, 2005, p. 1386, Walton-Hadlock, J.L.

Increasing amounts of medication must then be taken to compensate for the addiction and drug-induced brain changes. As the dosage increases, the side effects of the drugs can become hellish – very often, the side effects become more problematic than the actual symptoms of Parkinson’s disease.

Brain destruction

Because patients eventually become unresponsive to drugs or develop intolerable side effects within five to ten years, a new field of experimental, highly intrusive procedures was being practiced even as late as the 1990s. Thalamotomy and pallidotomy, which involve killing brain tissue via electrocoagulation for the purpose of diminishing drug side effects, began in the 1980s and was discontinued by the end of the 20th century.

Stem cell implantation

Experiments with surgical implantation of adrenal medullary or fetal substantia nigra tissue has had, for the most part, disastrous results. One of the more curious results came from the placebo patients in one experiment. These placebo patients had surgeries, but unbeknownst to them nothing was actually implanted in their brains: they had “sham” surgeries. In the younger group of placebo patients, they obtained very good results: their Parkinson’s symptoms were greatly reduced for a long period of time (more than a year)!¹

Many of the people who received the actual tissue implants, however, had ghastly side effects. Some of these side effects included perpetually violent movement that resembled the dyskinesia of over-medication from dopamine-enhancing drugs. Some others had no dopamine-related changes, but the implanted cells developed into teeth and optic tissue. The best results were those obtained by the younger group that received the placebo treatment: surgery, but no actual implantation of fetal cells.

These early experiments have been, for the most part, ignored by those clamoring for stem-cell research money to “find a cure for Parkinson’s,” even though there is a strong feeling among most Parkinson’s researchers that stem-cells will not yield good results in Parkinson’s disease. After all, even if someone found a way to guarantee that the implanted cells would produce a controlled level of dopamine, it seems obvious that a body determined to induce dormancy in its own dopamine cells would be able to eventually extinguish the dopamine production in other, introduced cells, unless those new cells were growing out of control, like rogues, causing violent symptoms of dopamine excess.

Until the actual cause of the dopamine cell dormancy is turned off, there is little to be hoped for in introducing more dopamine cells. Then again, if the cause of the dopamine dormancy is known, it makes more sense to treat that *source* problem, rather than the side effect of decline in dopamine-producing cells.

¹ This otherwise inexplicable long-lasting improvement in movement may be explained by the fact that a brain-opening surgery constitutes a trauma. As such, the surgery and its sequelae may be able to create a trauma-induced increase in adrenaline that will not climb back down until the effects of the surgery have completely healed. This violent boost to the otherwise flagging adrenaline system may be enough to propel a person into somewhat normal movement, just as we see when, in an extreme emergency, an immobile PDer can move with grace and speed using his temporarily loaded adrenaline system. Oppositely, the surge of dopamine that occurs when a PDer feels safe, which occurs in response to placebos, “safe activities,” or maybe even “successful brain surgeries,” can also allow a PDer to initiate movement almost normally for as long as his heart feels safe.

The dying dopamine-cell theory

Younger researchers acknowledge that many PD symptoms do not appear to be dopamine related. Many agree that, since dopamine-cell death is not actually the problem, growing new dopamine-producing cells via stem cell or any other cell source is not the answer. In my very limited experience, it is the older neurologists who still are convinced by the dying dopamine-cell theory.

Meanwhile, since 2001, the National Institute on Drug Abuse has named dopamine the neurotransmitter of pleasure and addiction. As we learn more about dopamine, the failures of the PD-dopamine theory loom larger. In the 1950s, dopamine was thought to be the neurotransmitter of relaxation, the opposite of acetylcholine, the neurotransmitter that conveys muscle tension signals from the brain. This came about when doctors saw that people with Parkinson's responded to dopamine: they concluded, bizarrely enough, that PD must be a disease of excess strength: too much acetylcholine relative to the amount of dopamine. That didn't actually account for most of the symptoms of PD. Anyone who spends time with an *unmedicated* PDer can tell you this disorder is not caused by excess strength and vigor.

This theory had been completely abandoned by researchers by the late 1990s. However, many of the older clinical neurologists (clinical means working with patients, not doing research) have remained utterly unaware of the changes, in the last few decades, of our scientific understanding of dopamine.

The old PD-dopamine theories simply do not fit the facts of the illness. But in the absence of any new theories, some doctors continue to promulgate the old ones.

Brain stimulating implants

Ever since the beginning of the twenty first century, Deep Brain Stimulating (DBS) implants have been receiving excellent reviews from the company that makes the implants and from some of the doctors doing the very expensive implanting surgeries. The stories of those people who have done well with the implants are easy to find on the Internet. All of the write-ups of "satisfied customers" that I have seen were sponsored by the company that makes the implants.¹

Independent researchers are finding that the results of the implants are mixed, at best. The DBS can temporarily (up to one year) reduce the drug dosage need of the PDer. However, once the brain has grown accustomed to the DBS, the need for ever-increasing amounts of the drugs seems to continue to progress, just as it did before the implants.²

¹ I have had to read many newspaper articles *very* carefully before finding that the nationally distributed press releases were, in fact, releases from the manufacturer of the DBS system – advertising disguised as news. In one case, our local paper ran an article on a local man, showing how well he was doing with the brain implant. I had to dig a bit before I found out that the information for the article had been provided, not by an intrepid reporter, but by the doctor who had done the work and the DBS-making company. I am certain that many doctors push for these surgeries with the best possible motive. However, they do seem blissfully unaware of the risks. In my line, I am more likely to hear from people whose DBS surgeries were a disaster.

I recall reading a report from the Canadian public health system that explained why they did not support DBS surgeries: the results were very uneven and the very real risks were not worth the short-term benefit that a minority of people received.

² A strong proof that the doctors fully expect their DBS patients to need drug increases within a year was brought home to me via an early patient of mine who had recovered from PD after ten years on medication, who did

A new development in the DBS field is the finding that altering the brain disruption signal of the implant on a regular basis helps to maintain the effectiveness of the implants over a longer time period. Still, the long-term effectiveness and side effects have not yet been determined.

Our program and the DBS implants are not compatible.

Brain implants are effective, but do not increase dopamine

A very important and highly disregarded finding is that the DBS implants do not increase dopamine levels, yet they allow a person to move with better control. This fits in with the research that shows that dopamine insufficiency is not the sole factor in many Parkinson's symptoms. Again, and follow me closely here, the DBS implants often provide some short term improvement in movement control *without altering the dopamine situation.*

survive the hell of getting completely off his medications. Dominic's previously rigid body parts were limp, and his previously weak ones were getting stronger. He was moving much better than he had been when I first met him, but he was not at full strength yet and probably never would be due to brain damage from the medications. He was plagued by fears and doubts. When his fears got the better of him, a violent tremor would appear. (This is a common problem with PDers who were in our recovery program who used medications prior to working with us.) He lived alone: his children convinced him he should have a brain implant. (There was more to it; he had been on a waitlist for the implants since before he had recovered. His doctor pressured him by saying that if he didn't have the implants when his name came up, he could never have them. The doctor, humoring him, assured him that if he was truly recovering, he could simply turn off the implants if he ever fully recovered. And so Dominic decided to have the surgery.)

At a DBS conference two years after Dominic's surgery, the doctor told the audience that he had a patient who was completely off all medications. Not only that, continued the doctor, he had been off medications for two years. The audience response was one of universal disbelief. Dominic's doctor was accused of lying. No doctor in the audience had ever seen, *despite their assurances to patients to the contrary*, someone with an implant who actually no longer needed medication. This anecdote, told to me by Dominic's sons, should give the lie to the stories doctors tell about "permanently" getting off of antiparkinson's medication following DBS surgery.

As to why Dominic did so well, his Parkinson's was gone but he was living with an inability to initiate adrenaline *or* dopamine release when he was worried. (We had not yet figured out the dissociation part of the puzzle at that time.) The implants do not increase dopamine, they cause the release of adrenaline. Since what Dominic now had was adrenaline- and dopamine-release insufficiency *when worried*, and brain damage from drugs but not actual Parkinson's disease, the implant worked well for him: it encouraged adrenaline release.

However, in my own experience, all my DBS patients except for Dominic have had, ultimately, horrible experiences. Their problems ranged from relentless insomnia (no sleep in six months!) to a continual feeling of fire ants crawling through the skin. I have also heard of ambulatory PDers who became wheel-chair bound as an immediate result of DBS surgery. I will no longer work with a DBS patient. Our treatment protocol is not recommended for people with DBS systems. Recovery from Parkinson's, according to our hypotheses and experiences, requires that a person make the switch from the adrenaline system to the dopamine system. The DBS system works by relentlessly goosing the adrenaline system. Although doctors point quickly to the fact that the implant's electrical signals can be turned off, the implants themselves are never removed. Removal would be a very high-risk event, risking tearing and bleeding in the brain. Doctors do *not* perform removal of the DBS, once it is installed.

As long as the implants are in the brain, they are a mild source of trauma. In fact, they provide enough adrenaline-releasing trauma that most people notice an improvement in their symptoms immediately after surgery, before the stimulation is turned on. This may be related to the responses observed in the placebo patients who had sham brain surgeries: the stress of the cerebral intrusion may create enough fear deep in the organism that adrenaline levels rise for a considerable time. In the case of the implants, the intrusion is small. The brain soon accommodates to it, thus necessitating the need for the electrical shocks to provide further release of adrenaline.

Where is the research headed?

Increasingly, the old, 1950s decision that insisted on a *causative* relationship between idiopathic dopamine-cell decline and Parkinson's seems to not actually hold up. But even today, in 2007, these extremely important points suggesting that the dopamine change seen in Parkinson's is only a small part of the story have not made a dent in the ongoing clinical treatment of Parkinson's. The dominant clinical paradigm of the day still holds that all the symptoms of Parkinson's disease are due purely to a shortage of dopamine. This shortage is, in the current thinking, caused by an inexplicable decrease in the brain's production of dopamine.

The idea that the brain might be *intentionally* decreasing dopamine-making cells because the brain is getting few calls for dopamine release; because an environmental cause (coming from outside the body; injuries are considered "environmental" triggers) is inhibiting dopamine release; or because a dissociation response is causing a tilt towards adrenaline and away from dopamine, is never even considered.

These ideas would suggest that the body is actually behaving in the way that the body is supposed to behave during time of injury or dissociation-inducing trauma, except that the injury never healed and the mental signal to end the dissociation was never initiated. In other words, there is no actual pathology, no real physical illness present: the body is doing exactly what the mind is telling it to do. The result of these electrical and mental instructions is partial dormancy in dopamine-producing cells that weren't being used anyway. This hypothesis is consistent with the fact the researchers cannot actually find a pathology at work in Parkinson's disease.

Researchers *can* find the physiological *results* of reversed Qi in the Stomach channel: cell reundifferentiation in the substantia nigra, debris floating around in the dormant cells, and a decrease in the number of the heart's sympathetic nerve connections. What researchers aren't figuring out is the reason that the body is making these changes.

And so, despite evidence to the contrary, and a drastically new understanding of the role of dopamine, Parkinson's disease is still considered by most clinical doctors as an unfathomable case of, for no apparent reason, low dopamine and nothing but low dopamine.

SUMMARY: HOPELESSNESS

When I started inquiring into the syndrome known as Parkinson's disease in 1998, the view at that time was that dopamine-producing cells in the midbrain were dying, reason unknown. The ensuing dopamine shortage caused all the symptoms of Parkinson's disease: poverty of movement, rigidity, tremor, and balance problems. Brain cells were thought to be incapable of healing or regrowth, and therefore Parkinson's was incurable. Dopamine was considered to be a movement neurotransmitter, not because of research proving it to be so, but because L-dopa allowed movement in PDers.¹ L-dopa or other dopamine-enhancing drugs were

¹ In 2007, almost no one except for old timers in the field of Parkinson's disease considers that dopamine is a movement neurotransmitter. Everyone else has more or less accepted the research of the National Institute on Drug Abuse and the research in psychiatry, research that identifies dopamine as a major neurotransmitter in regulating mood and seeking behaviors.

The reason that pharmaceutical dopamine imparts movement in PDers is that it alters their mood, shifting their behavior into a dopamine system-dominant pattern. PDers' normal system for movement, the adrenaline system, is also still turned on, though it is increasingly operating on a "minimal release" basis. Sometimes, the sensations of joy from the pharmaceutical dopamine can create such a feeling of well-being that the adrenaline system is temporarily turned off. As soon as the drug wears off, however, the habitual thoughts that employ the adrenaline-nerve resume. Others find that their negative or cynical thought patterns are so engrained that even when

the treatment, but their effectiveness waned quickly. Surgical treatments sometimes provided short-term benefits, but as the brain continued to “deteriorate,” the effectiveness of these treatments also waned.

By the year 2000, it had been determined that, in fact, the substantia nigra cells in idiopathic Parkinson’s patients were not dead, but they simply weren’t releasing/producing dopamine. They had altered, reverting back to a different, rather neutral type of cell. Then again, in people with drug- or toxin-induced parkinsonism, these same cells *are dead*.

Regardless of this fact, the paradigm presented to the general public continues to declare that idiopathic Parkinson’s is caused by the “loss” of dopamine-producing cells. New research continues to be performed on a model (usually represented by lab rats) in which the brain cells are killed, even though the brain cells in people with idiopathic PD are not dead.

Throughout this book, I will be redundant with this “cells are not dead” motif because some PDer have a hard time registering this information after they hear misinformed MDs, or after they read inaccurate health articles caroling the old canard about dead dopamine-producing cells.

Also, by the end of the 20th century, it was recognized that dopamine was a major neurotransmitter. Not only was it a precursor molecule that readied the brain for activities such as movement, acting as the go between for consciousness and action, but it also had a role in regulating body temperature and mood, controlling appetite, integrating left and right brain activities, monitoring the immune system, and being the neurotransmitter of joy.

Curiously enough, addictive drugs and chemicals, including alcohol, cocaine, methamphetamine, the opiates, and nicotine, are all addictive because they all elevate dopamine levels. Dopamine is the neurotransmitter of addiction. Part of the reason that elevated dopamine causes addiction is that dopamine is one of the most carefully self-regulated of all neurotransmitters in the body.

Finally, a quick websearch can find solid, recent research showing that various physiological responses such as certain vision reflexes and speech reflexes are different in PDer than they are in control subjects. And these reflexes remain different whether or not the PDer is given an effective level of dopamine-enhancing drugs. In other words, dopamine is not the whole story – not by a long shot.¹

Old paradigms die hard

And yet, despite all the new, conflicting research, including much new research coming in that proves *many* Parkinson’s symptoms are *not* dopamine related, the old Parkinson’s model

flush with dopamine, their adrenaline system behaviors and thoughts are still apparent. In this case, anxiety from the still-turned-on, but not releasing, adrenaline system combines with the flush of joy and joy-induced movement given by the drugs.

Dopamine does not create movement, per se. Acetylcholine makes movement. Dopamine bridges the connection between the joyful idea of movement and the imaging of movement. After the idea has been created and transmitted by dopamine, the actual movement signals that travel along the nerves and out to the muscles are sent via acetylcholine.

¹ Examples of such articles are: Grande, Laura J., Crosson Bruce, Heilman Kenneth, et al, “Visual selective attention in Parkinson’s disease: Dissociation of exogenous and endogenous inhibition,” *Neuropsychology*, 2006, vol. 20, n3, pp. 370-382 and Jurkowski, A.J., Stepp, E., et al, “Variable foreperiod deficits in Parkinson’s disease: dissociation across reflexive and voluntary behaviors,” *Brain and Cognition*, v. 58, n1, p. 49-61.

remains the dominant clinical paradigm. In this model, dopamine exists to serve only as a movement neurotransmitter; the dopamine-producing brain cells are dying of “no known cause” even though research continues to prove that (prepare for a redundancy) they are not dead.

One of the most disheartening things I learned while researching this subject is that my patients’ MDs are, for the most part, uninformed about any research that has happened since they were in med school. To an alarming extent, their post-school information has been almost entirely provided by people with something to sell: drug companies and the deep brain stimulating system manufacturer. Most clinical MDs have no idea whatsoever of the Parkinson’s research findings that have occurred since they got out of school.

Even among researchers, the inertia in the field is widespread. For the most part, the discovery that substantia nigra cells are dormant rather than dead has been of little interest or even of negative interest to PD researchers; most of their work relies on a “Parkinson’s model.” A Parkinson’s model is a mouse with toxin-induced parkinsonism: a mouse whose midbrain cells have been killed. Again, this is a very different situation from that of idiopathic Parkinson’s disease.¹

Among clinical doctors who are out in the field diagnosing patients and prescribing medicine, the new research is more or less ignored. Most clinical doctors cannot keep up with the tremendous amount of research that is going on, even within their own field. There is simply too much information coming in all the time.

It can take up to twenty years for new research to infiltrate the medical community. The exceptions, of course, are medications or treatments that are heavily advertised by their manufacturers. Oppositely, research findings that might decrease the sales of drugs, such as the news that dopamine-enhancing drugs cause permanent brain damage, rarely receive large publicity even within the medical field. Also, unpopular research conclusions have a difficult time getting repeat funding. And so the inertia in the advancement of medical knowledge lumbers along.²

¹ In 2005, I read yet another article on how exercise appears to prevent the worsening of Parkinson’s. This perky article was based on a medical study that showed mice whose brain cells had been killed via toxins were able to improve their physical condition via exercise. This experiment demonstrates *nothing* about idiopathic parkinson’s disease. It does suggest that people with brain damage from toxins can benefit from exercise. However, people with idiopathic Parkinson’s do not have brain cell damage or brain cell death. People with Parkinson’s have an electrical signal in the head that prevents the release of dopamine, regardless of how much a person exercises. People with Parkinson’s, when they do exercise, tend to do it with adrenaline and intensity, rather than dopamine and joy. I have seen that, while exercise improves symptoms of Parkinson’s disease for several minutes or for several hours after the exercise, those people who exercise the most vigorously tend to have the most rapidly developing cases of Parkinson’s. In my limited experience, it appears that the more vigorously the PDer uses his incorrect adrenaline-dopamine relationship, the faster the illness seems to progress.

² For more information see: Walton-Hadlock, JL, *Medications of Parkinson’s Disease or Once Upon A Pill: patient experiences with dopamine-enhancing drugs and supplements*. Parkinson’s Recovery Project, 2003, Appendix 5, “Why Your Doctor Thinks The Way He Does: Fifty years of changing dopamine theories,” pp 555-575 and Appendix 6, “Dopamine Fallacies,” pp 577- 592.



*“Oh, East is East and West is West, and never the twain shall meet,
Till Earth and Sky stand presently at God’s great Judgement seat,
But there is neither East nor West, Border, nor breed, nor Birth,
When two strong men stand face to face, tno’ they come from the ends of the
earth!”¹*

- Rudyard Kipling

CHAPTER FIFTEEN

WEST MEETS EAST: PART I

This chapter will show that the western symptoms of Parkinson’s described in the previous chapter are the same as the symptoms of Rebellious Qi in the Stomach Channel From Unhealed Injury at ST-42. In addition to showing a Stomach channel relationship to the recognized symptoms of Parkinson’s disease, I will show that the auxiliary symptoms of Parkinson’s also fit the model of Rebellious Qi in the Stomach channel.

I can note the specific locations and natures of the western and auxiliary symptoms and place them as dots on a map of the human body. The line formed by connecting the dots is the same line that is classically described in Asian medicine as Stomach channel. By the end of this chapter, I will demonstrate this match-up: I will show this channel alongside a head-to-toe list of the symptoms. But first I’ll need to write a chapter’s worth of more explanations and another spot of Asian medical theory.

DEFINING “AUXILIARY SYMPTOMS”

Auxiliary symptoms are those symptoms that are somewhat common in PDers and somewhat uncommon in the general population. These symptoms are usually ignored in western writing about PD; they don’t necessarily fit the dopamine theory; they weren’t mentioned by James Parkinson in his book, *The Shaking Palsy*. Still, when listed together with the locations of

¹ Rudyard Kipling, “The Ballad of East and West,” *Ballad, Poems and Other Verses*, Fenno & Company, New York City, 1899, p.11.

This famous stanza, so often quoted out of context, means that although the continents of east and west will of course never physically touch until such time as the earth itself dissolves, yet when great souls of any race, social standing or nationality come together, all barriers disappear. I hope that both Asian and western medical researchers and practitioners can be “great souls” and throw aside their prejudices enough to consider learning broader ways of thinking.

I should not be surprised if someday we learn that what we now call the Asian medical model was used throughout the world in ancient times, just as the recent discovery of the frozen prehistoric man in Switzerland showed knowledge of acupuncture points existed in Europe; his back had been tattooed with acupuncture points known to be effective for the arthritis from which he suffered, as shown in X-rays.

In the same vein, I had two patients who were reluctant to receive treatment because they were not certain that Asian medicine was appropriate for Christians. One nun began treatment after praying for guidance on a Catholic retreat. The other began treatment after his Baptist minister approved it. The minister told him that all healing comes from God, and that he should not be put off by the vocabulary nor the country of origin of a medical process.

the more classic symptoms, they give a fuller picture of the forces at work in Parkinson's disease.¹

Collecting the auxiliary symptoms

I did exhaustive intakes on all my PD patients. Some of my intakes lasted for hours. Some continued week after week as I was doing foot treatment on the patients. I wanted full medical histories and any information that might or might not be of obvious import: anything the patient thought was unique or different about himself. Most of the auxiliary symptoms were told to me on the basis of location. For example: "I get a pain in my lower back molar on the right side; it comes and goes. X-rays say there's nothing wrong." These location-specific symptoms got added to my "Locations of auxiliary symptoms" list. Some patients also described body-wide symptoms and what they felt were unique personality traits. These symptoms got put on two other lists, the "Body-wide auxiliary symptoms" and "Attitudinal symptoms" lists.

I did not include every symptom of every PDer on the lists – only the symptoms that occurred in several patients and which occurred more often in PD patients than might be expected in the general public of about the same age range. These symptoms ranged from always being careful to exhale while chewing in order to prevent choking (often for years prior to diagnosis) to skin problems. The skin problems ranged from mere seborrhea alongside the nose to a history of (removed) cancers, melanomas, and large, worrisome moles.

Actually, the cancer and melanoma symptoms seemed unrelated to PD at first, but *many* PDers had this history. When doing physical exams, I realized that the PDers' scars from the removals of their cancers, melanomas, and moles were always located smack on their Stomach channels, or once in a while on the Large Intestine channels, on the same side of the body where PD symptoms first appeared. I added the cancer histories to the list.

When the highly uncommon childhood symptom of purposely and repeatedly ripping off the entire nail of the small toe came up in two of the first twenty patients, that symptom almost made the list. They both explained that they had ripped off the 5th (smallest toe) toenail on what

¹ Many MDs are utterly unaware of even the most common auxiliary symptoms of PD. For example, a PDer told me that when he called a Santa Cruz MD on her weekly radio talk show to ask about his toe pain and toe spasm symptoms, she assured the PDer that his toe curling was not related to Parkinson's disease.

More than half of my PD patients have had painful problems from toe curling and toe spasming.

Curious, I ran a cursory webscan of the literature. I immediately found four articles relating to this issue:

"The frequency and significance of 'striatal toe' in parkinsonism," King's College Hospital and Guy's, King's and St Thomas' Medical School, London, UK. *Parkinsonism Related Disorders*, 2002, Dec;9(2):97-101. This article stated that 7 of 38 patients with PD patients had striatal (extensor planar response) toes in the absence of any other signs suggesting dysfunction of the cortico-spinal tract.

"Do Parkinsonian symptoms and levodopa-induced dyskinesias start in the foot?" Vidailhet, M., Bonnet, A.M., Marconi, R, et al, *Neurology*, Sep;44(9): 1616-6. This article explained that symptoms of parkinsonism started in the foot in six out of 20 PDers.

The other two articles described cases of rheumatoid-like and psuedorheumatiod deformities of the feet associated with Parkinsonism.

I merely mention these articles because the MD on the radio had been so quick to dismiss a not uncommon auxiliary symptom of Parkinson's disease – toe deformities, toes curling under and toe spasming – simply because this "poverty of vitality and movement in the foot" symptom is not one of the frequently mentioned Big Four symptoms.

eventually became the PD side because the gentle sensation of throbbing in the foot was somehow soothing.¹

Some of these symptoms that were seemingly unrelated to movement and dopamine, such as insomnia or a tendency towards constipation (of a type that is not helped by laxatives), were recognized among western researchers as being symptoms frequently seen in PD.

However, there had never been any attempt to connect these symptoms and events with the Parkinson's itself. The main reason they are not considered important may be that they do not support the 1950's theory of Parkinson's disease being caused by dopamine depletion. Yet these "recognized, but unrelated" symptoms consistently added more weight to my growing hypothesis.

As an aside, while discussing their various foot symptoms, many PDers volunteered that they had intuitively felt that their mobility problems somehow stemmed from their feet. They had been assured by their MDs that such a notion was purely wrong.

DEFINING WESTERN SYMPTOMS IN TERMS OF LOCATION

To create a location list for the "known symptoms" of Parkinson's, I replaced the Big Four's generalized symptom descriptions such as "poverty of movement" and "rigidity" with the exact locations of the symptoms. To do this, I used symptoms of my early- and mid-stage PDers, before their slowness, rigidity or tremor became body-wide.

I replaced the general term "poverty of movement" with the exact *locations* of poverty of movement symptoms in my early-stage PD patients: the lips, the eyelids, cheeks, the index finger, the second and third toes, etc.

I replaced the term "rigidity" with a list of the specific *locations* where rigidity most often appeared: anteriolateral muscles of the neck, the torso muscles along the mammary line, etc.

For tremor, though it may eventually become system-wide, I used the most common *locations* of early-stage PD tremor.

And so I made a list of the specific locations of well-known symptoms from the Big Four. I combined this list with the location list of auxiliary symptoms.

Putting the symptoms together on a map

For many months I had no theory at all as to why my simple foot holding was reversing symptoms of what looked like Parkinson's. But I was acquiring a pretty extensive list of location-specific symptoms, symptoms that might or might not actually be related to Parkinson's disease.

I still don't know why, one morning, I decided to mentally rearrange my growing locations list into head-to-toe order. I realized, to my amazement, that my list was drawing a picture of the Stomach channel; nearly all the symptoms of Parkinson's disease, the classic western and the common auxiliary symptoms, were located at various areas along the Stomach channel – always on the side of the body where the Parkinson's first appeared. If the symptoms was bilateral, the other side of the body's symptoms were much milder, almost an echo of what was happening on the side where symptoms first appeared. When I added in the arm symptoms,

¹ Those two people told me (I will paraphrase): "I've never told anyone about this, it's embarrassing. It didn't hurt, but I knew it wasn't normal. But it felt so good, in a strange way, to have feeling throbbing away down in my feet." That seemed almost bizarre enough to include, but it was too small a number, percentage-wise.

they were all located on the Large Intestine channel – the channel that feeds into the Stomach channel – also on the same side of the body where symptoms first appeared.

Considering that there are two each of the dozen primary channels and eight extra channels – thirty two channels all together – I had to wonder, what are the odds of all the symptoms of an illness appearing on only two channels? If Parkinson's was actually a disorder stemming from neurotransmitter insufficiency, shouldn't the symptoms be spread throughout the body, or at least distributed over many channels?

If Parkinson's disease was actually caused by a dopamine deficiency in the brain, the decrease in dopamine should affect all motor function. But in PDers, the specific movement problems were due to the fact that some muscles worked and other didn't. For example, the legs of PDers had anteriolateral muscles that didn't work and medial and posterior muscles that *did* work. This is why even PDers who have trouble taking steps forward or turning to the side can often walk backwards somewhat easily – sometimes even going backwards without meaning to.

Then again, because the muscles of the Stomach channel are the ones used in a majority of motor functions – walking forward or turning, getting up from a chair or rolling over in bed – a problem in the Stomach channel can look like serious overall movement inhibition, but closer observation will show that the “overall” problem is actually due to the problems in specific muscle groups, not all muscle groups.

For example, the hunched posture of Parkinson's is caused by rigid, shortened muscles in the anteriolateral (front and towards the sides) muscles of the neck and torso and a relative weakness in the muscles along the back of the neck and spine. The overall look thus created is one of body-wide hunching, but the actual muscles causing the hunching are a very specific group.

Of course, over time, as Parkinson's progresses, the conglomeration of worsening symptoms may snowball. And the ever-increasing anxiety and depression can add yet another level of inhibition to neurotransmitter release. The increasing motor problems and the mental/emotional factors can combine and multiply until a person becomes completely immobilized (although he may be able to move somewhat normally if given a convincing placebo (positive thought) or a convincing emergency (negative thought)). But in the earlier stages of Parkinson's disease, most of the symptoms are highly location-specific – not body-wide.

It seemed logical that a midbrain neurotransmitter deficiency should create body-wide symptoms, affecting all muscles similarly. But an *electrical* illness (a channel problem) would affect primarily the specific muscles, blood flow, skin and nerves along the path of the channel. The other muscles, areas of blood flow, skin and nerves would be relatively unaffected.

Based on what I was seeing, Parkinson's appeared to be, in its early stages, an electrical disorder, a channel disorder!

Next, I noted on my imaginary map which of the symptoms were problems of rigidity and which were problems of limpness.

In my mind's eye I could suddenly see that the symptoms of rigidity occurred in the portion of the Stomach channel that ran from the back of the jaw to the center of the foot. Symptoms of limp muscles and weakness were in the portions of the Stomach channel that traverse the face and the portion of the foot between ST-42 and the toes. Rigidity in the arm extended in a narrow line from the front of the shoulder down to the wrist, following exactly the path of the Large Intestine channel. Weakness in the arm most often began at the wrist and extended to the tip of the index finger.

Rigidity versus weakness

An introduction to Asian theory of Excess and Deficiency

In Asian medicine, one always notes whether a symptom is due to too much Qi or too little. These plus or minus quantities of energy are referred to as Excess and Deficient conditions.¹

In muscles, Excess Qi can cause conditions of pain, rigidity, spasms, tightness, and/or heat. A condition of insufficient, "Deficient," Qi can cause numbness, flaccidity, weakness, paleness, and/or cold.

Whether or not unhealthy body tissues are either flaccid or rigid is a point usually ignored by MDs with regard to Parkinson's, but these two opposite conditions can signify very different underlying problems.

In either condition, Deficiency or Excess, when Qi is not running correctly, cell growth veers off from correct to incorrect. The effected area might respond in many ways ranging from atrophy to growing in an uncontrolled manner (cancer, tumors). In either case, whether Excess or Deficient, the tissues in the zone of influence of the incorrect Qi fail to grow correctly. In the term "tissues," I include skin, muscles, bones, blood vessels, and nerves.

In Asian theory, there are many Qi conditions that fit under the heading of Excess, ranging from fever to a nail through the foot. Rebellious Qi (backwards flowing Qi) is considered one of the many forms of Excess Qi.

Examples of muscles with Deficient or Excess Qi

A saggy eyelid: Deficiency

Consider the flaccidity in the lower eyelid of advancing Parkinson's disease, usually worse on the SSFA (Side on which Symptoms First Appeared). This weakness, with the lower eyelid sagging down a bit, can be so severe as to create the illusion that the eyeball on the SSFA

¹ A majority of people using our books and treatment plans are not acupuncturists. My first book was directed to acupuncturists, *not* because they have experience treating energetic blockages or doing Tui Na – they usually do not have such experience – but because they could most easily understand the theories involved.

However, it has turned out that most of the health practitioners treating PD with these theories are not acupuncturists. Therefore, this book includes an extremely rudimentary explanation of channel theory and the theory of Excessive and Deficient Qi. These theories are crucial in allowing anyone to make sense of the symptoms of Parkinson's disease, symptoms that present uniquely in each PDer. Without the theory, this book is only a one size fits all, formulaic cookbook. By understanding the principles, one can understand how to apply the same thinking even if any given PDer turns out to be slightly different from the norm. And since no two PDers are alike, the theory is more important than any specific examples or case studies.

To those acupuncturists who have written helpful letters telling me that my theory chapters are incomplete, I offer thanks and freely admit that I am giving a very simplified version of Asian medical theory.

is larger. There is no pain, no tension, no rigidity in the sagging eyelid. The eyelid hangs lifeless, drooping. The PDer may not be aware of the extent to which the eyelid is sagging because he can't really feel the eyelid tissue.

A rigid neck: Excess

Compare the limp eyelid with the situation in the neck muscles: the muscles of the front of the neck become very rigid and contracted in Parkinson's. This in turn causes the following symptoms: the head is pulled forward and downward by the tightening of the front of the neck. This creates the neck portion of the hunched posture characteristic of Parkinson's.

At night, in bed, the tightness in the neck muscles pulls the head forward, preventing the head from relaxing down onto the pillow. Sometimes three pillows must be used to bring the pillow level high enough to provide support for the head. Without the pillows, the extreme tightness in the neck is very painful, as gravity pulls the head backwards towards the bed but the tension in the neck pulls the head forward.

Painful coughing and choking can occur due to the pressure on the front of the neck from the muscles that are sometimes stiff and rigid, sometimes even spasming.

The differences between the limp, flaccid muscles of the eyelids and the painful, tight, rigid or spasming muscles of the neck are differences between Deficient and Excess muscle conditions, respectively.

These same principles can be applied to a problem in any part of the body. These principles can help a health practitioner understand whether or not he is dealing with a condition of Deficient Qi or Rebellious Qi. This distinction is extremely important when choosing a treatment plan.¹

Rigidity is not a sign of strength

In Parkinson's, body tissues in certain areas become unresponsive to brain command, shrink up a bit and sometimes even become hard and tough, like dried beef jerky. These areas are found on a line that runs from the corner of the jaw down to the center of the foot. The line varies in width: it ranges from a quarter of an inch along the front edge of the neck muscles to an inch

¹ Even first semester acupuncture students should know the classic warning that forbids strengthening (tonifying) a situation that is Rebellious. I get queries from acupuncturists, who should know better, asking me why they shouldn't just needle the Rebellious Qi to "straighten it out." If they considered the Rebellious Qi in terms of the ancient admonitions to never tonify an Excess condition, they would not need to have written to me. The western reader, possibly scratching his head over this jargon, may be able to relate it to the popular injunction, "Don't feed a fever." Fever is an Excess condition.

As for the specious argument that some types of needling can reduce or drain excess Qi, acupuncturists need to remember that this argument only applies in certain conditions, such as bleeding a point to remove hot blood, dispersing a knot of Qi by spreading it out, or draining one element into another element. When it comes to an actual excess of Qi in the channel, and *especially* if Qi running backwards, a needle inserted into the Excess area will usually serve to amplify the ongoing condition. Although an experienced practitioner may be able to temporarily redirect the Qi with a cleverly placed needle, the condition will revert to its old pathology when the needle is removed – and may be worse than before. Many PDers intuitively know that needles placed nearly anywhere in their body (except the Du or Ren channels) make them worse: they tell me that, while they can tolerate pain easily, they've felt deeply uneasy when receiving acupuncture. Many PDers who were needled at the most common points on the Stomach channel prior to learning of our program have told us that the needling caused a horrible, even terrifying sensation, like a body-wide electrical shock, or even needle shock (passing out). Many PDers have a severe aversion to needles, both Asian and western needles.

and a half in the area just below the knee. This toughness and hardness are especially palpable on the front-outer portion of the thigh.¹

I refer to this hardness as “rigor mortis-like” rigidity to suggest that it is a bad thing. I need to do this because too many Americans, unfamiliar with the Asian theory, assume that a rigid muscle is a good muscle. They are thinking in terms of rigid meaning strong as steel: a good thing. In PD, the rigidity feels, to a health practitioner doing some prodding, like cement or a piece of lumber. This type of muscle tightness suggests not only an Excess Qi condition, but a specific type of Excess: decades of Rebellious channel Qi: *not* a good thing.

The nature of the symptoms of Parkinson’s

As noted in chapter five, in Parkinson’s disease, the Stomach and Large Intestine channels have been shunted away from the face. This dearth of channel Qi on the face is the reason that the symptoms along the facial portion of the Stomach channel, starting at the eyes and going down to the back of the jaw, are symptoms of Deficiency. However, where Qi runs backwards, from the jaw down to the center of the foot, the symptoms are symptoms of rigidity, excess tightening and hardness in the muscles. From the center of the foot to the toes, where the Stomach channel Qi cannot flow, the symptoms are once again symptoms of Deficiency.

On the arm, the symptoms are primarily symptoms of Excess, except for the area from the tip of the index finger up to the point where the thumb meets the index finger: this area is deficient, even atrophied.

This brief introduction to the ideas of excess and deficient symptoms will have to suffice. Now we get to apply this theory to the symptoms of Parkinson’s disease.

Remember, Rebellious Qi is considered a type of Excess.

AN “AH HA!” MOMENT

It was while working with my fourth PD patient that I realized, much to my amazement, that the Qi in his legs was running wrong. At the time, I was still extremely dubious about Asian medicine. I had digested my course material in Asian medical school by interpreting its precepts through a protective screen of western science and biology. No one could have been more startled than I to discover that backwards-running Qi, Rebellious Qi, was just that: electricity-like currents that were running in the reverse direction.

I am blessed with extremely poor eyesight; an overdeveloped sense of touch is my compensation. Always keenly sensitive to electric charge or static in people’s skin, I could easily feel with my hands an electrical flow in my patients’ skin, but I had always dismissed it as mere static electricity.²

It was only when I realized that this PD patient’s leg electricity was moving in a reversed direction from my non-PD patients that I suspected that this electrical feeling might be related to that Qi that I had spent four years reading about in school.

In a moment, channel Qi went from being a theoretical concept to a tangible, measurable quality. There is no name for this discernable energy flow in western medicine or even in the

¹ Many of my PD patients have pointed these unyielding muscles on the anteriolateral side of the thigh and told me, “I’m really in good shape despite the Parkinson’s; look how strong these leg muscles are.”

² Actually, anyone can feel it. It just takes realizing what one is feeling. My advantage was that I didn’t need to try to feel for it; it jumped out at me.

entire field of biology. However, based on how it moves, short circuits, and is effected by nearby currents, it recalls to mind the principles of electricity and magnetism I learned almost forty years ago, in high school physics class.

Qi is real. Who knew?

As soon as I realized that the palpable sensations I felt flowing in the limbs of my patients was “Qi,” I could stop thinking of channel Qi as a principle, and start thinking of it as a physical reality. Channel Qi wasn’t some mysterious arcane and theoretical force. It was some energy that felt like static electricity sometimes, and at other times felt like a stream of electricity.

Suddenly, the concept of “Rebellious Qi” that I had learned in school took on a new meaning. I could easily feel that the currents running in this PDer’s Stomach channel was not going in the direction I had learned in school. Nor was it running in the direction that seemed familiar to me. (I had learned about the correct directions of channel Qi flow in Asian medical school, but I had never made the connection between this book learning and the sensations that I was accustomed to notice in patients’ skin.) Doubting myself, I checked his other channels. They were running in the usual directions: the directions that I had learned in school!

Back to the location map

By the time I constructed the location map of my PD patients’ symptoms, I had already seen that all my PD patients had backwards flowing Qi in some parts of the Stomach channel on the side that first exhibited symptoms of Parkinson’s. Other parts of the Stomach channel, the face portion and the portion downstream from ST-42, had no amount of palpable Qi.

Having noticed this already, I felt a giddy chill as I realized that the symptoms of Parkinson’s that featured rigidity were located on the portion of channel that was running backwards. The symptoms of Parkinson’s that featured weakness were on the portions of the Stomach channel that had no Qi at all. Suddenly, the combination of Deficient and Excess symptoms in Parkinson’s made perfect sense. The *nature* of the Parkinson’s symptoms in any given location depended on whether or not Qi was running Rebelliously or was not running at all, in the body part in question. This was glaringly logical.

This combination of Excess and Deficiency corresponding to areas of Rebellious and insufficient channel Qi suggested something new to me: Asian medical theory might be far more practical and objective than I had ever imagined. Also, whatever was happening with my Parkinson’s patients’ symptoms in response to my foot holding at ST-42 *might* have a tangible, logical explanation far beyond my earliest thought that “the Yin Tui Na at the foot allows the foot injury to start healing.”

As mentioned earlier, the classroom lectures on Rebellious Qi back in my school days had addressed superficial, fleeting issues of Rebellious Qi, such as coughing or burping. Rebellious *channel* Qi had never been discussed. (Remember, the very existence of channels had been disavowed by the Chinese government.) But now, for the first time, I understood that the pathologies of rigidity that I was feeling were the results of palpable Rebellious channel Qi on the tissues directly under its influence. Evidently, backwards flowing Qi, over time, caused muscle tissues to become wooden, distorted, or even cancerous. An absence of Qi, somewhat predictably, caused numbness, weakness, lack of muscle function.

I had to wonder if the picture of Rebellious Qi and absent Qi in the Stomach channel could lead me to an explanation of the symptoms of Parkinson's that *weren't* location specific. After all, I still needed a theory that would allow me to account for the few symptoms of Parkinson's that didn't fit on any channel. The dormancy of substantia nigra cells was at the top of my list of puzzling non-channel symptoms. But other body-wide symptoms such as poor temperature regulation and personality-based symptoms such as heightened wariness were also puzzling. The question then became, could Rebellious Qi in the Stomach channel also cause these symptoms? Could Rebellious Qi cause a dormancy in substantia nigra cells? If so, how?

Slowly I was able to put together a logical explanation of the channel routing changes that would occur in response to a long-term condition of Rebellious Qi flow. These Qi routing changes *did* explain the dopamine dormancy and most other body-wide symptoms. That explanation was given in chapter five.

In this chapter, I will show the list of western-recognized and auxiliary symptoms of Parkinson's alongside of a drawing of the Stomach channel – a sort of West meets East presentation.

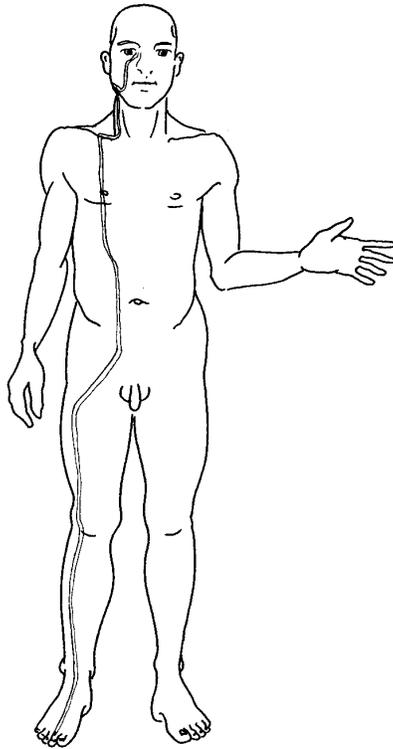
The list of all symptoms, by location

Key to the list:

- *The PD symptoms recognized by western medicine are marked on this list with a hollow circle.*
- *The auxiliary symptoms are marked with a filled-in circle.*

- no or slow lower eyelid blinking; sagging lower eyelid.
The symptom is worse on the Side of the body where Symptoms First Appeared (SSFA).
- sinusitis, problems with the sinuses, especially on the SSFA, including severe snoring and even sleep apnea
- seborrheal skin on the cheeks or along side the nose, especially on the SSFA
While seborrheal skin may appear prior to the diagnosis of Parkinson's, it more commonly appears in the years following diagnosis.
- loss of sense of taste or smell
- a feeling as if the roof of one's mouth is sinking down into the mouth at the back of the mouth, as if the sinus bones are collapsing downward inside the face
- inability to smile, worse on the SSFA
- inability to realize that facial muscles are not actually moving when PDer thinks that he is smiling
- feeling of deep cold inside the cheek, especially on the SSFA
- pain that comes and goes in the back lower molars on the SSFA
- excess salivation
- poor swallow reflex
- spontaneous spasming in the throat for no apparent reason, choking or coughing from "nothing," choking or coughing from saliva, choking easily when eating, spasms in the throat
This choking symptom may start decades before the diagnosis of Parkinson's.
- aspiration pneumonia from food going down the wrong way
- teaching oneself to always exhale before putting food in the mouth and not breathing once food is in the mouth to avoid a tendency for food to slide down the airpipe when chewing
This symptom may start decades before the diagnosis of Parkinson's.
- hunched posture, head pulled forward
- choked off voice, soft voice
- difficulty turning the head from side to side
- orthostatic hypertension (low blood pressure, insufficient blood supply to the head when standing up from a sitting position)
This is probably due to the pressure on the carotid sinus in the neck. The rigid tissues of the neck press on the carotid sinus, sending a false "high pressure" signal to the

sinus. The body correspondingly lowers the blood pressure.¹ This symptom, orthostatic hypotension, is commonly associated with adrenaline insufficiency as well.



The Stomach Channel

(The branch that skirts the lips is not shown.)

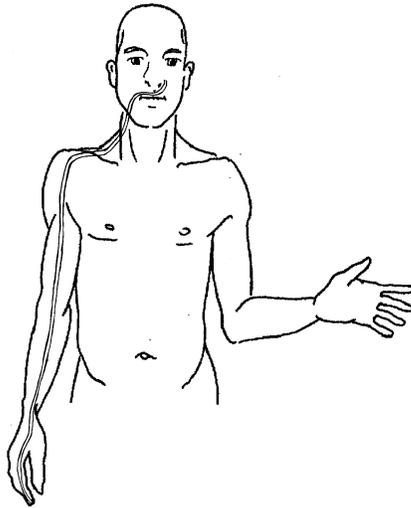
- discomfort, almost a feeling of suffocation or panic, if doing an activity with the arms raised, even briefly, over the head: taking down a shower curtain, getting plates down from a high shelf. This discomfort is due to the rigidity over the collar bone and chest; raising the arms causes the rigid chest muscles to push in on the lungs.
- pain or tingling between the shoulder blade and the spine when trying to sit up very straight with the shoulders back for any length of time, especially on the SSFA
- either a “cast-iron” stomach or a hypersensitive one
- difficulty turning over in bed at night, or turning from the waist
- in women, extremely deep, pathological abdominal stretch marks that formed during pregnancy from an utter failure of the skin to stretch

In one case, a pre-PDer experienced failure of the uterus to expand during her second pregnancy, necessitating a Caesarean section for a baby of low birth weight. Her first child had been carried to term in a fully expanded uterus.

¹ Many people with Parkinson’s are proud of their low blood pressure, never realizing that it is a part of their Parkinson’s pathology.

- either the ability to hold the bladder for an alarming number of hours, sometimes urinating only once or twice a day, if that, or else the opposite: chronically frequent, scanty urination
- chronic constipation – a type of constipation that does not respond to laxatives
 - Some people have the opposite: a long-time tendency to very loose, poorly controlled stools, even prior to the diagnosis of Parkinson's.
- pain in the groin, especially on the SSFA
- lack of hair on the legs along the Stomach channel, especially on the SSFA, even if the other leg or the rest of the leg has a normal hair pattern,
- extreme hardness in the anteriolateral muscles of the upper and lower leg
 - Many PDers point with pride to this steely bit of flesh, and imagine that it is supremely toned muscle. The energy movement and tone in these muscle groups, however, suggests the woodenness of rigor mortis rather than the tone of healthy tissues.
- difficulty in moving to the side, turning to the side while walking
- more difficulty in turning to the SSFA than in turning to the other side
- a sensation described as “woodenness,” “weirdness,” “buzzing,” “something irritating under the skin,” or “something not right” referring to the feeling in the anteriolateral portion of the legs
 - These feelings can be constant, but they especially might be felt at the end of a long day of standing.
- a rare feeling of momentary tingling or buzzing that comes and goes in the medial ankle, especially prior to the diagnosis of Parkinson's
 - This ankle feeling can be significant enough that one is prompted to pull down the sock and stare at the ankle, looking for the source of the irritation. But nothing visible is going on. However, severe vascular irregularities, varicosities, and skin staining may occur on the medial ankle, especially on the SSFA.
- cogwheeling in the ankles
 - The “cog” is at the Stomach channel point of the ankle, worse on the SSFA.
- foot drop, worse on the SSFA
- festinating gait due to shuffling steps, foot drop
- misshapen feet or toes, worse on the SSFA
- grey or purplish cold feet or toes, worse on the SSFA
- veins on the dorsum of the foot on the SSFA that do not run down to the toes
 - Instead, the veins often form a loop just distal to ST-42 (at the center of the foot), worse on the SSFA.
- tendency for cramping in the sole of the foot due to no muscle function in the opposing muscles on the dorsum of the foot, worse on the SSFA
- toes curling under the sole of the foot due to no muscle function in the opposing muscles on the dorsum of the toes, worse on the SSFA
- severe bunions and other displaced bones, worse on the SSFA
- smaller foot on the SSFA
 - The foot on the SSFA might be from one half to two full shoe sizes smaller than the other foot.
- toenail fungus, especially in the three medial toes, worse on the SSFA

- distinct toenail ridges that run parallel to the moon of the toenails
(Interestingly, unusually large ridges that run the *length* of the toenail may, in my limited experience, indicate a blockage in the foot portion of the Spleen channel. This can cause a floppy foot, an inability to lift the feet, and an overall look that is very different from Parkinson's disease.)
- inability to separate the 2nd and 3rd toe on the SSFA
- numbness on the medial side of the big toe (acupoint SP-3), or poor response when a needle is inserted at SP-3, especially on the SSFA
Needling this point should be breathtakingly painful in a healthy person.
- lack of proprioception in the feet and toes, inability to know where the toes are if shoes or slippers are on



The Large Intestine Channel

- lack of proprioception in the hands and arms, inability to know where the hand is and in which direction it is supposed to move when putting on sleeves that hide the hand from view
- atrophy of the muscle that pulls the thumb over to the 2nd metacarpal bone
- atrophy of the bicep
- pain or weakness in the bicep
- poor small motor skills: cutting food, picking up small things, doing buttons
- micrographia
- If micrographia is present, upper arm soreness during extended periods of handwriting

The PDer is using the upper arm to make the lettering instead of just using the very small muscles in the hand and wrist. This may be due to the lack of small motor function and proprioception in the fingers, particularly the index finger.

Inappropriately performing this small motor task with the large motor muscles causes the bicep area to tighten up quickly. And since the actual bicep itself may be somewhat or highly atrophied, other upper arm muscles will struggle to control writing movements that *should* be done with the fingers and wrist.

- cogwheeling at the wrist

- cogwheeling at the wrist most pronounced at the intersection of the wrist with the Large Intestine channel
- lack of arm swing
- prior to diagnosis, when arm swinging is/was still possible, a tendency to swing the arms in a peculiar manner, maybe unnaturally forceful, or with a side to side motion instead of the more normal front to back pattern, or with the hands rotated away from the normal position
- tendency for the arms to be crooked at the elbow when the arms are at rest, with the hands resting on or near the waistline
- pill-rolling tremor
- history of cancer, melanoma, lipoma, or tumor along the Stomach channel or Large Intestine channel

Summary of the location of symptoms:

As seen by the above list, the Stomach and Large Intestine channels are, with few exceptions, the locations of the symptoms of Parkinson's disease.¹

Looking at it the opposite way, the locations of symptoms of PD form a picture of the Stomach channel and Large Intestine channel. At first glance, this map of symptoms fit my budding hypothesis that a Stomach channel disorder was involved in the development of Parkinson's disease. At second, third, fourth and fifth glances, I started putting together the hypothesis of channel aberrations and rerouting that can explain the Excess and Deficient location-specific symptoms of Parkinson's disease.

Happily, this hypothesis also explained the *body-wide* symptoms such as dopamine-cell dormancy (see chapter five xxx), movement inhibition, and other body-wide symptoms that can be sequelae of an unhealed injury that's in a critical location. Those body-wide symptoms can still be related to channel aberrations. The next chapter will further discuss those findings.



¹ The primary exception is the medial ankle pain, varicosities, and discoloration. These medial ankle symptoms are located in an area where the Stomach channel can short circuit into the Kidney channel. This short circuit on the ankle often occurs in the vicinity of acupoints KI-2 and KI-3. In chapter 5, fig 5.1, page 62, a diagram shows how Stomach channel Qi on the foot can sometimes short circuit into the Kidney channel at KI-2.

“Through Thy grace the sudden shafts of wisdom will dispel error accumulations of countless centuries.”

Whispers From Eternity by Paramahansa Yogananda

CHAPTER SIXTEEN

WEST MEETS EAST: PART II

A FEW MORE CHANNEL ABERRATIONS

Much of this chapter may make more sense to a student of Asian medicine than to the person who merely wants to know about Parkinson’s disease. It is included because I sometimes receive queries from acupuncturists who want to know if any other symptoms of Parkinson’s are related to shifts in channel Qi. Much of what follows may seem obscure to the lay reader. However, even if the channel flow references are hard to follow, the gist, the summary, and the conclusion of this chapter should be clear.

Body-wide auxiliary symptoms

Of course, the *body-wide* symptoms did not fit a location, per se. Nevertheless, some of these symptoms corresponded with characteristics of specific channels. These channels, while not directly injured, might become caught up in the aberrations when Stomach channel Qi became Rebellious.

- extreme sleepiness between 11 p.m. and 1 a.m. (a time of day associated with the Gallbladder channel), with a tendency towards restlessness or insomnia during the rest of the night and fatigue or restlessness during the day

The fatigue may be subclinical during the day; situations that produce a sense of urgency can cause the tiredness to momentarily recede. Because of this fatigue, a PDer may sense a constant internal battle between alertness, restlessness and fatigue. He may draw upon his strong will power to get through the day, sometimes creating mental situations of emergency, fear of failure, or even fear of disappointing someone in order to produce enough adrenaline-based drive to keep going.

- time of greatest discord often between 7 a.m. and 9 a.m.

7 to 9 in the morning is the Stomach channel time of day. At this time of day, a PDer’s overall energy might be either at its height or it may be at the lowest level: he may be at his most dynamic and high-powered, or he may be utterly unable to get up and get going.

The list of body-wide symptoms suggests discord in the Gallbladder and Stomach channels. The Gallbladder- and Stomach-related symptoms support our hypothesis that channel Qi running perpetually backwards in one of the Stomach channels could eventually short circuit into one Gallbladder channel. During the day, and especially during times when the Stomach channel is being stimulated, the excess current in the Gallbladder channel would create a pre-sleep electrical signal in the brain on one side of the brain while the other side maintained a normal waking signal.

From 11 p.m. to 1 a.m. the current in the Gallbladder channel automatically increases greatly. This sends a go-to-sleep signal to the brain. In a PDer, the only time of day when both of the Gallbladder channels are sending the same signal to his brain is this two-hour period at night. This is when most PDers experience an extreme level of sleepiness.

During the rest of the night, the difference between the two sides creates an electrical imbalance, a vibration, an internal tremoring that can cause restlessness and insomnia.

Attitudinal symptoms

Attitudinal symptoms are similar to the symptoms that are “body-wide,” inasmuch as most of them don’t appear in a specific physical location. However, just as certain physical, body-wide symptoms are considered to be related to specific channels, the following attitudinal symptoms are also related to specific channels.

- aversion to or difficulty in visualizing, imagining, or even pretending to imagine either one’s entire body or certain body parts, as being filled with light ¹
- difficulty in creating a mental self-picture of oneself in the here and now
- fear of objectively incorrect behavior, such as tardiness or making an incorrect statement, or making a wrong turn or becoming lost while driving

A majority of my PD patients, before seeing me for the first time, drive to my office the day before the appointment to make sure that they know just where to park and to make sure they don’t fly into a panic/rage by getting lost looking for my office on the day of the appointment. None of my other patients that I know of, in all my years of practice, have done this.

- strong dislike for anyone, even a spouse, to see or touch the feet
- in conversation, often draws the subject away from oneself and towards the other person, or towards more neutral subjects such as philosophy
- very often has a history of dangerous or frightening childhood

¹ There will be much, much more about this symptom later. For now, let me expand on it via an example: one of my patients was a playwright and screenplay writer. He could mentally picture whole scenarios, from the backdrop to the facial expression of every actor in his plays. He could not, however, when doing a dopamine-stimulating exercise, visualize or imagine any light in his body. When asked to visualize his body as being full of light, he protested strongly. He insisted that it was impossible for him to do such a thing. Finally, after months of coaching, while he insisted he couldn’t do it, he did it. At the same moment as he was able to imagine light in his elbow, he inexplicably started weeping. He then cried for almost ten minutes: big, wrenching sobs.

On the other hand, one patient had gone to a hypnotherapist prior to her diagnosis to understand why the left side of her body wasn’t working. With the guidance of the hypnotherapist, she was able to mentally create a picture of the inside of her body. The right side was gleaming and functional. The left side, the PD side, was filled with rotting timbers and stagnant water. She was unusual in that she was able to see anything; most PDers can’t even imagine any image or activity going on inside their most effected body areas and are terrified even to take a mental look.

I first began to wonder about the significance of this after my first three PDers answered my not uncommon (in Asian medicine) intake question about childhood with an unexpected coolness.¹

- fear of anxiety or stress-inducing social interactions
- strong aversion to highly charged emotional situations, particularly situations involving anger
- high aversion to being in the vicinity of people who are being overly assertive, threatening, or who might become angry
- depression
- powerful mental attempts at staying busy to combat depression
- high capability for strength, speed, and stamina in his pre-PD years
- dread of making a wrong turn or becoming lost while driving
- inability to cry, especially during his youth
- high intellect
- a keen interest in gathering information. A local doctor in my community was known to tell his PD patients, “My Parkinson’s patients are the best informed of all my patients. Once they’ve been diagnosed, my PD patients usually know more about the latest developments in the field than I do.”

○ Parkinson’s personality

Briefly, the Parkinson’s personality may include emotional-harm avoidance tendencies, strong will power and intensity of purpose.

- strong moral or spiritual leanings, though not necessarily associated with a religious denomination. Many PDers are deeply spiritual. By “spiritual,” I mean that they often try to embody the qualities of service to others, selflessness, detachment from materialism, and asceticism. They often spurn activities that might be considered self-indulgent or flamboyantly self-expressive.
- absence of joy, absence of ideas as to what might bring joy.

Many PDers, when asked what gives them joy, answer that making others happy gives them joy. When asked what someone might do for them to make them happy, or what makes their own chest expand with joy, their own heart thrill with happiness, they have given me statements such as: “It’s been so long, I don’t remember,” “I don’t really feel joy,” “I don’t know,” and “I don’t think I even know the meaning of the word.” (Since joy is the essence of the spiritual life, there can be a certain irony here.)²

¹ Regarding this diffidence in talking about childhood, the intake scenario usually ran something like this: “...and what about your childhood? Anything interesting that you think might be of significance?” The PDers each replied to my query with more or less the same words, while becoming distant for the first time: “I don’t think we need to discuss that. I’ve worked that out.”

I’ve been a primary medical care provider for years; I’ve grown used to people answering my medical intake queries. This evasion on the part of my first PD patients when I asked about their childhood certainly piqued my curiosity. And yet, others were not shy at all; they told me stories of hair-raising violence and cruelty. But they told me these stories in a detached manner, and usually assured me that it didn’t matter anymore, that they had “dealt with it.” Of course, not everyone had a horror story from his immediate family or a childhood that called for a high level of stoicism. But even many of the people from happy homes recalled some injurious event during which they decided to put up a strong face.

² A swami, a monastic yogi, once told me that people with Parkinson’s disease are very advanced souls who have temporarily lost their way. A Tibetan monk stated that people with Parkinson’s are living in their heads; they have forgotten that God is also in their bodies. I have discussed the spiritual aspect of PDers’ lives with

Location of the attitudinal symptoms

At first glance, the Parkinson's personality and other attitudinal symptoms seem to be body wide, and therefore might *not* seem to fit onto a body map. However, the insufficiency of courage and absence of joy can be related, in Asian medicine, to diminished function of the Kidney channel – the channel that supports the adrenal gland and the pericardium. In Asian medicine, the adrenals and the pericardium are related to courage and joy, respectively. In chapter five, a map of the distortions of Qi in the foot shows how the aberrant Stomach channel may cause the Kidney channel to become deflected towards the floor. As noted in the previous section on temperature regulation, this and other aberrations in the Kidney channel can occur because the distortion in the Stomach channel in the vicinity of KI-2 and KI-6, near the ankle. When this occurs, the amount of Qi that flows in the overall Kidney channel is diminished.¹

hundreds of PDers and their family members. Some of these long talks have gone late into the night. The insights from people who have recovered from PD have been especially enlightening. Most recoverers have thought deeply about the personality changes that they experienced during recovery. I have written up the following conceit (extended metaphor) to share the brutally honest self-analysis and conclusions that arose in these many talks.

People with Parkinson's may feel as if they have spent lifetimes climbing up the steep mountain of self-discipline, seeking God attunement or spiritual progress. They have spent lifetimes practicing mind over matter and denial of the flesh. As they've climbed higher and higher up the spiritual mountain, they've felt a growing detachment from the people in the metaphorical valley below. Increasingly, they have come to view those who were not working at seeking Truth as being earthy, bawdy people, larking about in childlike ignorance.

After lifetimes of diligent exercises for self-discipline and/or spiritual practices, they have developed tremendous mental powers of concentration, superb physical control and strength, the capability to *detach themselves from the sensations of the body*. And then pride over these accomplishments has crept insidiously into their hearts. They have come to feel that they were somehow different from or superior to the less powerful people. When pride came, they lost their sense of direction. They tried to use their hard-won capabilities to fend off the freezing loneliness of their high spiritual altitude.

They magnanimously serve others from their state of spiritual elevation even while they feel apart from them. From high on their metaphorical mountain of spiritual attainment, they become responsible, they take charge, they pridefully give blessings to the underachievers, the incompetent people below. But in the loneliness and emptiness of their attainments, they remain assailed by their infinite fears.

They have forgotten that attunement with God is most easily found via the low-lying paths of humility, devotion, and the trust in God that brings spiritual fearlessness.

And so, by virtue of the Divine laws of cause and effect, they eventually develop this fitting illness. The symptoms of this illness are the logical conclusions of what they have long sought: they have denied the senses of the body for lifetimes, and so the senses become stripped away: taste, smell, and touch are all lost. Their mental determination to be unmoved by worldly events results, eventually, in an illness characterized by inability to participate in the world.

Finally, when they can no longer dress themselves, feed themselves, clean themselves, explain themselves, or even blink their eyes in wordless communication, they may realize that they are dependent for every aspect of daily activity on those very people that they once spurned: the common people down in the vale.

Eventually, after repeated lifetimes of struggling against the symptoms seen in Parkinson's disease, they *will* learn to accept, with humility and gratitude, the ministrations of the kind members of the community. Their minds will someday let go of their fear and pride. When this happens, they will suddenly behold God all around through their remaining senses of hearing, sight, and intuition.

They will hear God's love and sympathy in the gentle patient words of the people who care for them. They will perceive in their heart the good intentions of God flowing to them through the actions of the helpful people of the valley. They will see that the very bodies of even the most unsophisticated people of the valley are condensations of the light of God. The spiritual seekers had never been alone on their mountain after all: God was always there. And there never was a mountain that needed to be scaled.

¹ I need to remind the aggressive acupuncturist that merely needling the Kidney channel will not bring about a lasting benefit in this situation. As long as the Stomach channel is deranged, it will continue to disrupt the

The palpable decrease in Qi flow from KI-1 on the sole of the foot over to KI-2 and other foot points of the Kidney channel do correspond to decreased Qi flow in the Kidney channel. The decrease can have an effect on adrenaline release – which can influence courage. Also, the main branch of the Kidney channel becomes the Pericardium channel.

The pericardium muscles influence the degree to which the heart favors, at any given moment, the sympathetic system or the parasympathetic system. A decrease in the flow in the Kidney channel can lead to decreased function of the pericardium and the nerve function in the heart.

As noted in chapter one, in Parkinson’s disease, neurotransmitter receptor function in the heart is measurably decreased (measured with SPECT scans). This suggests a problem in the Pericardium channel.

Also, many PDers have difficulty in imagining their pericardium relaxing enough to allow the heart to rotate back into the parasympathetic heart position. PDers’ hearts tend to be physically *stuck* in the extremely sympathetic or even the dissociated position, a position determined by the pericardial muscles and the flow of the Pericardium channel.¹ This “stuck heart,” in turn, can prevent a person from resuming parasympathetic mode. The stuck heart can keep a person in sympathetic or dissociative mode. This, in turn, influences thought patterns and attitude.

In Classical Asian medicine, deficiency of channel Qi in the Heart or Pericardium channel is the cause of anxiety.

Therefore, although attitude may *appear* to be body wide, channel pathologies in the Kidney and Pericardium channel can contribute to attitudinal symptoms.

Electrical aberration at the end of the Stomach channel can be detected disrupting and diminishing the flow of the Kidney channel. This in turn diminishes flow in the Pericardium channel. The Pericardium channel can also be diminished by selective dissociation from the heart. A person with only psychogenic parkinsonism may not have any of the channel aberrations from foot injury, but he may still have inhibition of the Pericardium channel that *mentally* have been set in motion.

Many PDers in our experience have had both of these inhibitions: one from injury, one from selective dissociation.

Kidney channel. The underlying problem must first be addressed. Premature treatment of the Kidney channel can actually add energy to and thereby worsen the Rebellious situation in the Stomach channel. Also for the acupuncturist, the reason the Kidney channel is diminished overall is because the errant Stomach channel Qi gets shunted back into the Spleen channel at SP-6, leaving an overall deficiency in the Kidney channel.

¹ The reader who is baffled by the idea of a heart “position” might want to try the following exercise. Imagine that your heart has a spindle running through it from top to bottom. Imagine that the heart can rotate around the spindle. First, rotate the heart counterclockwise as far as it can go (counterclockwise while looking down at the heart, from the head.) Hold it in this position for a moment. Notice the tightness in the chest and the overall sense of tension and anxiety. Next, imagine the heart rotating the other direction as far as it can go. Notice a sense of heaviness and sleepiness. Finally, let the heart go back to its first position, “facing forwards.” Notice the sense of alertness and the feeling that something pleasant is emanating outwards from the heart. When I write about PDers’ deficiency in the pericardium (the muscles that surround the heart and also allow the heart to rotate counterclockwise or clockwise in response to sensory input or bedtime), I am referring to the way that many PDers’ pericardium seems to be stuck in the “anxious” position. Many PDers are unable to successfully perform this exercise.

PD personality: not for everyone

Not every person with Parkinson's disease has all the symptoms listed in this chapter – including the symptom of “Parkinson's personality.” I have met people with bona fide Parkinson's who do not fit the “classic” Parkinson's personality profile and do not the attitudinal symptoms listed above.

Even so, for the most part, people with PD might be described as ***being, or having been in their youth or at their moment of injury***, self-denying stoics who are willing to serve others but not themselves, and who would rather die than reveal to the wrong person that they have been physically or emotionally injured.

For example, you read in the case studies chapter about Gus, whose injury was received during a war-time battle: immediately before, during, and after the time of injury, his personality was, of necessity, very much like that of the classic, wary, PD personality. However, prior to the war and after the war he had a very easy going, almost happy-go-lucky personality. His stoic, dissociated personality was compartmentalized in his brain; it only remained with regard to his foot injury and that day of death. The stoic personality was locked up in his brain in the same brain compartment as his war injury. The rest of his personality was not typical of the Parkinson's personality.

Although his ordinary personality was normal and content, this one small part of his mind and memory remained ever under stress and dissociated from his conscious sense of self.

He did not develop Parkinson's until he was in his mid-80s. The lateness of his injury (early 20's) and his predominantly non-PD personality are possibly the reason for his very delayed onset of very mild Parkinson's.

Oppositely, people whose PD manifests early or hard and fast tend to fit fairly closely the generalities about the Parkinson's personality.

Symptoms that don't fit the map: exceptions to the rule

Some PDers had symptoms of rigidity, pain, dystonia, numbness or immobility at unique locations, locations that were not in common with other PDers. Very often, these unique points of pain did not fit on the Stomach channel or Large Intestine channel. These extra locations were usually scattered sparsely on one or two different channels and each scattering was unique.

Over the years, we learned that the extra problem areas were usually located at body parts that had received injuries at some time in the past: a broken rib, a badly sprained wrist, a surgery or a whiplash accident. These injuries were always present *in addition* to an unhealed foot injury. These injuries often felt, to the health practitioner's hand, as if they were still unhealed.

Clarifications and caveats

For research purposes, I did not include any PD patients in our write ups who had a significant, known medical situation in addition to their Parkinson's symptoms. People with a history of stroke, heart disease, or anything that might confuse the data were not included in the research.

Also, remember this: no one PD patient will necessarily have all of the symptoms, classic western medicine or auxiliary symptoms, described in this chapter. At his time of diagnosis, he may have only a few symptoms. The symptoms of Parkinson's disease usually develop gradually. At first they may even be intermittent.

And for those of you in the audience who might have Doctor's Disease or hypochondria, remember: although each of the preceding symptoms is often seen in conjunction with

Parkinson's, in and of itself it is *not necessarily a warning sign or precursor of Parkinson's disease*.¹

Finally, don't forget: the symptoms of idiopathic Parkinson's disease are somewhat different from the symptoms of drug- or toxin-induced parkinsonism; these forms of parkinsonism do not necessarily have the auxiliary symptoms, nor do the natures (excess or deficient) of the symptoms of drug- or toxin-induced parkinsonism necessarily conform to the nature of idiopathic Parkinson's symptoms.

Adding weight to the hypothesis: symptoms of recovery

Wonderfully, the *nature* of each problem area reverses itself during recovery. For example, during recovery, the flaccid muscles of the face may begin to spasm; this spasming and tension are signs of returning strength: the reversal of their previous limpness. On the other hand, the rigid muscle of the neck may become so limp that the recovering PDer can't hold his head up for several days.

The reversal of rigid and limp pathologies during recovery suggest that the nature of the symptoms is as significant as the location in understanding the pathologies of Parkinson's. The extreme degree of reversal suggests that the tissue damage is deep; nerves, muscles, brain connections all behave as if they have been dormant or damaged and need to heal. This suggests that most PDers have had ongoing damage along the Stomach and Large Intestine lines long before they were ever diagnosed. This also fits with our hypothesis.

The significance of Location-specific Symptoms

Although I now know many ways in which "death of the dopamine cell" theory does not explain away all the pathologies of Parkinson's disease, the map exercise was the first time it really hit me. The location-specific symptoms of Parkinson's suggested that Parkinson's disease is primarily an electrical disorder, a channel disorder.

Not only does a map of symptoms paint a picture of the Stomach and Large Intestine channels, it conflicts with the idea of a systemic dopamine problem. The symptoms of Parkinson's are located in certain areas and not others: a *systemic* dopamine deficiency could absolutely not explain why specific muscles consistently had problems and others did not.

For example, many PDers originally have tremor in their index finger along the course of the Large Intestine channel. The other fingers are usually not involved until the PDer begins to mentally dissociate from his now problematic (tremoring) hand.

Also, certain facial muscles are typically affected, but not others; a PDer cannot use the muscles of the face that create a "ball" or "apple" in the cheek while smiling but he can easily raise his eyebrows by lifting the muscles of the forehead.

PDers often have no proprioception in their middle toes. (Proprioception is the ability to feel where a body part is even when one's eyes are closed or the body part is outside of the field of vision). PDers usually cannot separate their 2nd and 3rd toes on the SSFA. They can often move and detect the location of their other toes.

If the problem was truly one of dopamine deficiency, all toes, all facial muscles, all fingers, should have equal or randomly occurring difficulty in proprioception and movement initiation.¹

¹ Doctor's disease is the tendency to imagine that one has the symptoms that one happens to be reading about. This not uncommon syndrome is particularly rampant in medical schools; some would-be doctors truly feel, and sometimes visibly manifest on their bodies, the symptoms that they happen to be studying.

This brings to a close the sections that show how the symptoms of Parkinson's disease can be best understood by recognizing the changes in channels that occur in people with Parkinson's. The rest of this chapter closes off the subject of Part I of this book: The Cause of Parkinson's Disease.

FINDING THE CAUSE OF PARKINSON'S DISEASE

The first map

The first time I mapped the symptom locations, I saw that the Stomach channel might provide answers for the underlying cause of Parkinson's disease. But at that early date, expecting a Stomach channel aberration to account for the brain changes seen in Parkinson's disease seemed to be asking too much.

Then again, these combined symptoms of Excess or Deficiency presenting in PDers were baffling. I could not think of any systemic condition in the brain that could create conditions of excess in some parts of the body and conditions of deficiency in others. *If all the symptoms of Parkinson's were due to a dopamine deficiency, all the pathologies should have the same deficient nature.*

Poor muscle tone from dopamine deficiency should produce limp muscles throughout, not the blend of limp and rigid that is seen in Parkinson's disease.²

In the meantime, questions remained. What about the brain's cellular changes? Was the stoic attitude the thing that prevented the foot injury from healing? If so, how?

The Stomach channel story could not, at first glance, answer all the questions. However, it did give a good thumping to the increasingly inadequate dopamine-cell death theory. Over the next several years, recovery symptoms answered more questions. The recovery symptoms led us to propose the set of channel reconfigurations that could account for all the symptoms of Parkinson's and the recovery symptoms. Once we knew what to look for, we saw evidence of these channel aberrations in all the PDers. Also, some of the more arcane teachings of Asian medicine, such as the Law of Midnight/Midday (the two hours periods of increased Qi in any given channel) suddenly became meaningful.

Most importantly, by merely treating the foot injury, some of our PD patients made a complete, easy recovery from Parkinson's disease.

¹ Of course, in advanced PD, the problem may eventually affect all five toes. And there are a few PDers whose feet are so damaged that they haven't been able to move any of their toes since their college days. Still, we were basing our generalizations on the situations seen in most early- and mid-stage PDers.

² The reason that dopamine-enhancing drugs improve movement in rigid areas is that they mask pain. If neck muscles are rigid, a person will refrain from turning his head from side to side because of the tension and pain. Under the influence of pain-masking dopaminergic drugs, he will not feel pain. He may move in whatever manner he wants to. When the drugs wear off, the pain in the rigid area resumes, possibly even increased by the movements that were done while he was under the influence.

The antiparkinson's drugs do not resolve any of the symptoms. They mask the symptoms.

Partial recovery and the discovery of the mental/emotional component

PDers who got stuck in partial recovery added a new dimension to the mystery. They no longer had no physiological symptoms of Parkinson's and had experienced some of the recovery symptoms, and yet they were emotionally susceptible to "attacks" of Parkinson's symptoms.

Working closely with these patients, and with the patients with psychogenic parkinsonism, we uncovered the shocking commonality of dissociation from the heart and an inability to feel safe. In the early twenty-first century, new research using mice proved that feeling safe is the trigger that causes an initial surge of dopamine release in the substantia, which then leads to seeking behaviors which then leads to further dopamine release.

Putting this all together with the foot story, we created a model that could explain all the symptoms Parkinson's disease. The model required two pathological patterns: a foot injury and dissociation from either the foot *or* the foot and the heart. The dissociation could be either held in place all the time or selectively activated in situations in which the PDer did not feel safe.

By treating both of these problems, the foot and the dissociations, we were able to eliminate Parkinson's disease in *some* partially recovered PDers, but not all.

Many patients dropped out of the program when they learned that they would have to change some of their mental habits. Others dropped out because they were convinced that they could *never* change their mental habits. Others felt that when we included the self-induced mental component to our theory of Parkinson's, we were "blaming the victim." We got some heated correspondence accusing us of inventing the idea of a mental component so that we could explain away those PDers who did not recover even after their foot injuries were gone and they'd experienced the foot-related recovery symptoms.¹

But we have stuck to our guns: one of the commonalities in recovered PDers is their conviction that they have become a "different person." In cases involving people who were stuck in partial recovery, the "different person" did not emerge as a *result* of recovery; by *becoming* a "different person," the PDer was able to recover.

One new idea

The reader may be wondering if all of my ideas about channel rerouting of Rebellious Qi are supported by Asian medical theory. The answer is "almost."

But first, remember: modern Asian theory did away with the existence of channels. Even so, many practitioners of Asian medicine have observed proof of channels and some of the older doctors secretly still make use of channel theory.²

Historically, learning about channels was the traditional starting point for any study of Asian medicine. Therefore, honoring tradition, some of the old channel theories are still taught, even though they are usually taught as historical relics. As for channels flowing backwards,

¹ When I first received hostile correspondence, I was surprised. But my son reminded me that paradigm shifts go through three stages. In the first, the new ideas are mocked. In the second stage, they are met with open hostility. In the third stage, they are accepted as self-evident.

² Twice in one year I attended classes taught by highly respected teachers of Asian medicine. Both of them made a statement at some point in the lecture to the effect that "it's OK if you don't needle the acupoint in the exact right place, as long as you get the needle on the channel." Both of them, when asked after class whether or not channels actually exist, said that they did not believe in the existence of channels: they followed the party line.

failing to flow, or flowing into other channels, those ideas are not my invention. Those are old, established ideas.¹

I did not invent any of the basic concepts presented in our hypotheses except for one, the jaw shunt. For the rest, if I accepted the idea that Qi is real, that channels are real, and that channel Qi follows the rules of electrical movement, then all of the aberrant channel flow seen in Parkinson's could be explained both in terms of Asian medicine and in terms of basic western physics.²

The only new idea that I had to add to my training in Traditional Chinese medicine was the idea of a Qi shunt on the jaw that served to prevent Stomach channel Qi from running backwards over the face and into the Du channel. Nothing in the traditional literature suggested that such a shunt system does exist. Even so, this shunt *does* exist: in PDers, it was palpable by hand.

Though it was not in the literature, I could feel the shunt on the jaw in my PD patients: the Stomach Qi in their necks ran backwards, up to their foreheads. It did *not* retreat back up the face path of the Stomach channel all the way to the point between the eyebrows. I had to hypothesize that, when Stomach Qi runs backwards, it is shunted away from the face. This built in shunt makes sense to me: surging, backwards-flowing Qi through the midbrain can cause death.³

With the jaw shunt in place at ST-6, Rebellious Qi from the Stomach channel, should it occur in response to any serious foot injury, is redirected up to the corner of the face where it should either cause headache or healing sleep. In mild cases of Rebellious Qi in the Stomach channel, Qi pools up at ST-8 and causes a headache, hopefully forcing the injured person to take a nap. In response to more serious injuries, the large amount of rerouted Qi building up at ST-8 may short circuit over into GB-4. When this takes place, a person may want to drop into a

¹ Even the newest student of Asian medicine is probably familiar with the principle of Wood Attacking Earth, in which the upward flowing energy in the Liver channel gets jammed up at LI-14 and palpably shunts over into the Stomach channel, thereby disrupting the downward flow of Qi in the Stomach channel. Though the word "channel" has been removed from the phrase "Liver channel attacking the Stomach (or Spleen) channel," a quick feel of the channels in the vicinity of LI-14 will show that the physical problem is the channel rerouting that occurs at acupoint LI-14. Channels have been removed from the nomenclature, but the classic pathologies usually imply the pathological action of Qi in the channels.

² I have received queries, sometimes hostile ones, from acupuncturists who were taught that Qi only runs in the lines that are taught in the basic textbooks. I always ask them to start feeling the Qi flow in their patients. They will soon have proof that, to paraphrase Tolstoi, "All people with healthy bodies in parasympathetic mode have channel Qi flow that moves in the same way. All people with injury and illness, including mental illness, have at least some portion of channel Qi that flows in its own, unique way."

³ I had seen a demonstration of this principle by accident. I was using gold and silver needles on a patient who had lack of focus and depression. I was trying to stimulate the Du channel through the head, so I put a gold needle on her forehead and a silver one on the back of her neck. The use of two different metals creates a small electrical current. By using one gold needle and one silver needle, I can make a very low powered "battery" that sends a current between the two needles.

I made a mistake. I should have put the gold at the back of the neck and the silver on the forehead. Instead of generating a current from the midbrain to the forehead, I was generating one that ran backwards through the head. At first, my patient said that she felt weird. Then she said, weakly, that something was wrong. As she started to pass out, I saw what I had done and removed the needles. I reversed the needles and within seconds she returned to full alertness.

healing sleep and take some time to live slowly, nursing his wounds. This jaw shunt is an excellent built-in safety/health mechanism for a normal person. It is the only Qi flow item that is original in our hypothesis. And I strongly suspect that it is not “original,” but was known by other doctors before my time.

SUMMARY

I have shown the extent to which nearly all the well known and lesser-known symptoms of Parkinson’s are located on just two channels of the body: the Stomach and Large Intestine channels. By showing that the *nature* of the symptoms corresponds to Rebellious and Deficient Qi patterns, I have shown that these symptoms may correspond to an unhealed injury and the ensuing pattern of Rebellious Qi in the Stomach channel, complete with the shunts and short circuits described in chapter five xxx.

Asian medical theory explains *how* these channels changes can lead to non-channel symptoms such as the inhibition of dopamine-release and the decline in dopamine producing cells.

The mental/emotional blockages known as dissociations, which have been widely studied by psychologists and psychiatrists, can explain the symptoms of Parkinson’s that are affected by mood, thoughts, and situations.

Another point that makes itself clear while looking carefully at the full list of recognized symptoms and auxiliary symptoms is that the current paradigm, the “dead dopamine cell” theory, simply does not match the limited locations or the mixed natures of the symptoms of Parkinson’s disease. Even the few symptoms that eventually become body-wide, such as slowness and depression, symptoms that might conceivably be caused by a systemic disorder such as dopamine deficiency, also happen to resemble a healthy response to severe injury *and* a selectively-induced dissociation-from-the-heart response.

The Big Four symptoms in Parkinson’s disease don’t have any logical connection. Also, the tremor and balance problems – two of the four – do not necessarily improve even if a PDer takes dopamine-enhancing medications.

Oppositely, the symptoms of sustained Rebellious Qi in the Stomach channel because of an unhealed injury make perfect sense – if a person is aware of the electrical schema that courses through the body and which provides instruction to every cell in the body from the time of conception.

Throwing away the dopamine theory for failing to fit the facts, we can say just this: Parkinson’s disease causes anatomical and physiological changes. The cause is unknown. Long-maintained Rebellious Qi in the Stomach channel due to unhealed foot injury creates the same changes. The cause is known.

IN CONCLUSION

Parkinson’s disease is the same illness as sustained Rebellious Qi in the Stomach channel From an Unhealed Foot Injury.

Many of my readers mistakenly think that Rebellious Qi *causes* Parkinson’s disease. No. Sustained Rebellious Qi in the Stomach channel and its inevitable shunts and short circuits is the

same exact thing as Parkinson's disease. They are both names for a syndrome that manifests itself as a particular collection of symptoms.

This bantering over a name is actually very important. The name "Parkinson's disease" is associated with incurability. One patient told me, "The day the doctor said the words 'Parkinson's disease' to me, the world changed."

As another example, I was examining a recovering patient and noticed that the Qi was finally running correctly in his legs. By his own admission, his symptoms were nearly gone. I said to him playfully, "Oops! Looks like you were misdiagnosed!" A few minutes later, he said to me, "The moment you said that, that I was misdiagnosed, I felt a shift go through my whole body. I felt a glow inside that I haven't felt since I was told I had Parkinson's. It's as if, in that moment, when you said that, that's when I really recovered. I know it's just a name, but until then, even though my symptoms were mostly gone, I could feel there was something wrong inside."

There is power in a name. Since Parkinson's is defined as incurable, doctors should be very careful in giving out that diagnosis. For myself, I'm reluctant to work with patients who have Parkinson's: Parkinson's is incurable.

I prefer to work with people who have Rebellious Qi in the Stomach channel From Unhealed Foot Injury, with possibly some Dissociation from the Heart on the side. That disorder is curable. The two syndromes are exactly the same.



PART II

RECOVERY SYMPTOMS

“There’s nothing either good nor bad, but thinking makes it so.”

Shakespeare’s Hamlet

CHAPTER SEVENTEEN

RECOVERY SYMPTOMS: AN INTRODUCTION

As PDers recovered from their foot injuries, many of them experienced unexpected, counterintuitive, and even bizarre physical and emotional changes. In the early days of the research, these events became as much a part of the puzzle as the symptoms of Parkinson’s disease. Although the smaller details of these events varied from one PDer to another, there was a baffling similarity in the changes that PDers reported to us, week after week.

For research purposes, our earliest PD patients almost never met with or spoke with each other: the probability of research “contamination” from patient-to-patient interaction was unlikely. Furthermore, since our hypotheses as to the cause of Parkinson’s disease had not been formed when the first pioneers began to recover, there is no way a patient could have “guessed” at appropriate recovery symptoms, and no way we could have suggested them. We were as stunned as the patients when one PDer after another manifested his own variations on these unexpected symptoms.

We named these events “recovery symptoms.”

When I first started noticing changes in PDers in response to my treating their feet injuries, I had no idea of any theory that might connect the foot injury to the symptoms of Parkinson’s.

In the end, the largest source of information about the cause and development of Parkinson’s disease was the collection of recovery symptoms. The recovery symptoms, together with my understanding of Asian medical theory, provided the clues that allowed us to figure out the processes that lead to idiopathic Parkinson’s disease.

“What the heck?”

The pioneers in our project expected any improvement from Parkinson’s to be pleasant. They assumed that recovery, if that was indeed what was happening, would consist of steady, linear improvements in motor function. They assumed, as well, that any benefits would take the form of a “return to the past.” In most cases, this would have meant a return to the previous way of life: high pain threshold; strong will power; adrenaline-activated thought and movement.

What actually occurred did not fit anyone’s expectations. Every one of the pioneers was shocked by the unanticipated, seemingly negative collection of new symptoms that occurred as their feet began to heal.

But as they began to see the positive flip-side of these sometimes painful or alarming changes, they started to appreciate that something incredible was going on: they were truly recovering from PD, not just feeling better by masking the symptoms. After all, the new symptoms, though weird, were the exact *opposite* of the symptoms of Parkinson’s.

The credo of many in this group soon became “I don’t know what the heck is going on, but it sure as hell isn’t Parkinson’s disease.”

Introduction to physiological symptoms

The physiological pathologies of Parkinson's are those symptoms that are apparent whether or not a person is trying to move. These symptoms may include coldness in the hands and feet, the numbness or lack of muscle responsiveness in the face and toes, the muscle tension that pulls the neck forward and inhibits rotation of the neck, the forward-leaning, shoulder-hunching tension in the overall carriage, and the steely rigidity in the anteriolateral muscles of the neck and legs. These conditions exist whether or not the PDer is trying to move. For example, if a PDer has an absence of musculature along the side of the nose or in the lower eyelid, it will be present whether he is sitting down, lying down, or walking.

The *physiological* symptoms of Parkinson's disease do not change with mood or expectation. They are caused by actual alterations in muscle tissue, nerve tissue, blood supply, and brain-to-muscle connectivity. These symptoms may *contribute* to the poor performance of certain movements, in the same way that a polio-shortened leg or missing thumb may contribute to poor performance. But they do not *cause* the mood-based variations in poor movement initiation or tremor.

The physiological symptoms are *not* improved by the taking of dopamine-enhancing medications because they are *not* dopamine-related symptoms. They are simply the result of long-term changes in nerve and muscle that result from the electrical disarray that is present in idiopathic Parkinson's disease.

Introduction to mental/emotional symptoms

In people with idiopathic Parkinson's, emotional and attitudinal postures that are most likely related to selective dissociation can trigger symptoms of automatic dissociation, which includes inhibition of dopamine release. This inhibition can occur whether or not a person has any sort of foot injury. People with psychogenic parkinsonism do not have physiological symptoms of Parkinson's, but they do have difficulty with rigidity, movement initiation, poverty of movement, tremor, and postural instability. Some people with idiopathic Parkinson's disease have *both* physiological symptoms and the same mental/emotional symptoms that are present in psychogenic parkinsonism.

Similarities between physiological and mental/emotional symptom

In some cases, severe dissociation from the heart can cause symptoms that were listed above as being caused by physiological damage, symptoms such as cold feet and hunching posture. While these symptoms can be caused by physiological damage, they are also characteristic of automatic dissociation. However, when these symptoms are being produced by dissociation, they will vary in intensity, and may even relax a bit while sleeping. When these symptoms are produced by injury-based damage to the tissues, what I am calling physiological damage, the symptoms do not vary.

For example, a broken leg bone is a physiological event. If a person with a broken leg bone is in an emergency, or if he has an overriding flood of dopamine from either internally produced joy or dopamine-enhancing drugs, he might *not* be able to notice the physiological damage. He might be able to move *almost* normally. However, the damage will still be there: the broken leg will not come and go in response to his mental or emotional state.

While a person has idiopathic Parkinson's disease, it can be difficult to determine whether or not a symptom is purely physiological or if it is mental. However, during recovery, the distinction often becomes more clear.

WHY THERE IS NO LIST OF SPECIFIC RECOVERY SYMPTOMS

In the first edition of this book, I included a list of recovery symptoms that had occurred in my first dozen patients. Because I was concerned that some symptoms were purely individual, and not characteristic of PD, I did not include symptoms on the list until several PDers had experienced them. But once I did list the symptom, I included on the list the specific muscle groups and the exact locations in which PDers had experienced those symptoms.

As the project grew, I kept trying to update the information, listing additional recovery symptoms as I observed them.

However, I soon found that the more exacting PDers that were coming into the program were using this information as a checklist. If their own recovery symptoms were not on the list, they panicked. And if a PDer *didn't* experience all of the items on the list, he was certain that his recovery had stalled. We did not yet realize that shutting down the heart can cause a PDer to favor a worst-case scenario.

Now, I no longer publish a list of all the specific muscles in which recovery symptoms have occurred. Instead, I want PDers and their health practitioners to understand the mechanisms involved.

The following chapters list only a demonstrative *sampling* of recovery symptoms, together with explanations of *why* the symptoms occur.

Linearity

In my original list of recovery symptoms, I listed them in order of which recovery symptoms had often, but not always, occurred first, second, and last. I learned that, no matter how many times I wrote "recovery symptoms did not necessarily take place in this sequence," some PDers panicked if their recovery symptoms were not in the sequence in which they had been listed. I repeat, we did not appreciate the inclination towards negativity that many PDers had acquired and/or cultivated.

Therefore, let me point out that toes may recover before fingers, or vice versa. Improvement in sensory function may precede the return of easy movement in some body parts, or vice versa. Toes may regain sensitivity prior to recovering full range of movement. Or vice versa. Oppositely, facial muscles may show increased movement long before full feeling returns to the face. Or vice versa.

The path of recovery was not predictable, and recovery symptoms did *not* follow a straight line.

THE TEMPO OF RECOVERY

Physiological recovery: somewhat slow, measured in days or weeks, not minutes

With regard to the *physical* symptoms, no one experienced a red-letter day when he woke up and said "Ah! The Parkinson's is gone! Today is The Day!"

Please remember, there had never been a distinct day when the PDer woke up and *suddenly* had idiopathic Parkinson's disease. The physiological symptoms of idiopathic

Parkinson's came on slowly. The physiological recovery also took some time. Trying to ascertain a single day on which the physical symptoms were gone was as tricky as trying to ascertain the exact day on which a broken bone is healed.¹

Once the injury has healed sufficiently, the *electrical* disarray can correct itself almost instantly, but repairing the damage it caused during the previous decades can take weeks, even months. It will take time to grow new muscle. It will take time to retrain new nerve connections. It will take time to return to full sensory function.

Mental/emotional recovery: as quick as flipping a switch

The negative emotional and mental patterns that inhibit neurotransmitter release can come and go as quickly as thought. Many PDer's have noticed that following a bit of bad news, such as receiving their diagnosis of Parkinson's disease, they suffered an *instant* decline or rapid daily decline in their ability to initiate movement. Oppositely, if they felt encouraged by some positive idea, they may have had an almost instantaneous, but temporary, improvement in movement initiation or speed of movement; in these cases, their movement almost instantly improved to the extent that their current physiological condition allowed. As soon as a negative thought arose, the movement inhibition returned within a few minutes.

When a PDer who'd been stuck in partial recovery overcame the habit of dissociation that had caused his mind-based symptoms, the tendency to slip into parkinsonism ceased. When the PDer made this breakthrough, the lasting disappearance of the mood-based symptoms of Parkinson's occurred as quickly as the flip of a switch.

How long will this take? How intense will this be?

The amount of time required for healing is variable, and I will not make estimates as to how long it might take for any given individual. I have had patients whose more obvious symptoms of Parkinson's disease were gone within three to five weeks of the foot injury starting to heal. I have had other patients who were still noticing tiny improvements in motor function a full five years after the injury was healed and the major symptoms of PD were long gone.

Also, the intensity of recovery symptoms varied from person to person. The recovery symptoms were sometimes subtle, sometimes not. A PDer might have experienced severe symptoms in some muscle groups and had almost no *noticeable* cues as to the changes that were gently occurring in some other body part.

Some people recovered from the *physiological* symptoms of idiopathic Parkinson's disease in a matter of weeks or months but lingered in a dopamine-inhibiting dissociation or negative mindset for years following the physiological recovery. If so, his symptoms during

¹ Drug- and toxin-induced parkinsonism *can* come on quickly, even overnight. The book, *The Frozen Addicts*, xxx describes the immediate or overnight onset of full-blown parkinsonism, complete with utter rigidity and constant tremoring, that occurred in a group of drug users that used a "bad" batch of synthetic heroin. These drug users suffered immediate, irreversible brain damage. Their condition has *nothing* in common with people who develop idiopathic Parkinson's disease. Nevertheless, *The Frozen Addicts*, and the hypotheses contained therein, was the primary source of the "dead brain cell" theory of Parkinson's. Today, most research on Parkinson's is done on lab mice that have been given drugs to kill their brain cells. These mice are referred to as a "parkinson's model," even though it has since been proven that people with idiopathic Parkinson's disease do *not* have dead brain cells.

In cases of tardive (delayed onset) parkinsonism developing from use of mind-altering drugs such as methamphetamine and use of pharmaceutical drugs such as some anti-depressant and anti-anxiety medications, the parkinsonism from the brain damage caused by these drugs may come on more slowly – in some cases, over decades.

those years resembled the usual presentation of *psychogenic* parkinsonism, plus any other symptoms that his mind had grown accustomed to activating.

One psychology report that I read claimed that it takes five years, on average, to get rid of severe selective dissociations. But we have seen people recover from heart-dissociation very quickly, in a matter of weeks. We have also seen people struggle with the heart-dissociation symptoms characteristic of psychogenic parkinsonism for *more* than five years. In these latter cases, sometimes they understood what they needed to do to change, but were so afraid that taking the first step would leave them vulnerable to emotional pain that they were unable to take the first step. Sometimes, the PDer truly had no idea of how to go about changing his negative mindset without also experiencing a painfully humiliating change in long-held beliefs. Many PDers were genuinely shocked to learn that a person's thoughts are not spontaneous and unalterable, but that research has proved that thoughts and self-control of thoughts are the products of mental training – whether positive or negative.

The format for the chapters on recovery symptoms

No two PDers had recovery symptoms in the exact same order. Even so, the recovery symptoms that tended to occur earlier are in the earlier chapters of this section and the recovery symptoms that tended to occur later on are in the later chapters of this section.

Each chapter has an explanation as to *why* these symptoms occur, a few specific examples of what these symptoms looked or felt like, and an example or two of the fascinating ways in which some PDers have used the recovery symptoms to mentally convince themselves that they were getting worse instead of better.

And did I mention that no two PDers have the exact same collection of symptoms? Each PDer's recovery was also unique.



“He jests at scars, who never felt a wound.”

Shakespeare’s Romeo and Juliet

CHAPTER EIGHTEEN

RECOVERY SYMPTOMS: NUMBNESS AND PAIN

Numbness vs. rigidity: a review

The locations of the physiological symptoms of Parkinson’s disease are listed in chapter seven, XXX. Most of these symptoms, it will be recalled, are due either to the absence *or* the reversal of Qi flow in the Stomach and Large Intestine channels. Most of these changes can be characterized by either numbness or rigidity. In areas where Qi flow has become absent, numbness occurs. In areas where Qi flow has been backwards for years, rigidity occurs.

When, in response to Yin Tui Na treatments, PDers’ Qi began to flow correctly in the Stomach and Large Intestine channels, the numbness or rigidity began to melt away. Areas that were previously numb became sensitive. Areas that were rigid became limp.

This chapter will address the recovery symptoms that occurred in body parts where Qi flow had been absent or minimal: areas of numbness. The next chapter will discuss recovery symptoms in body parts where Qi flow had been backwards: areas of rigidity.

STARTING WITH THE FOOT INJURIES

Most PDers wrongly assumed that the healing of a foot injury and restoration of healthy Qi flow in the feet would necessarily be pleasant. The *end* result was pleasant enough, but along the way, many PDers experienced pain.

The pain of injury

The FSR techniques, or any other light-touch massage technique, encourage a person’s awareness and even subconscious to revisit a long-ignored injury and thus initiate healing. When the mind decides to acknowledge an ignored injury, the unexpressed, dormant symptoms of injury may appear. These symptoms may include bruises, tenderness, pain, swelling and even heat.

If the PDer’s injury was only a dislocation or sprain, the reawakened pain wasn’t necessarily severe. If the reawakened injury was a broken bone, the pain was sometimes considerable.

As patients started experiencing the pain of forgotten injury, they often said, “My foot hurts! What should I do?” We were surprised by the fear and questions that arose. These people were adults; we thought that they should have known what to do. But even though they might have been capable of helping care for other people who were in pain, they often had no idea of how to care for themselves when they were hurt.

We had to teach them.

How to deal with pain

The appropriate thing to do, we explained, whatever sort of pain occurs, is to treat the injury the way that it should have been treated in the first place. Use common sense. If, following treatment, the foot hurts, then favor the foot for a few days. Limp a bit. If the foot itches, scratch it. If it wants to be rubbed, rub it. Listen to the body, and give it what it wants. Again, use common sense. Do not be stoic. Treat your “recently” injured foot in the way a well-loved child would treat an injured foot. This may even include (God forbid) asking for and accepting help. If it’s a severe sprain or dislocation, you might want to wrap it up or get a little extra sleep instead of going dancing.

In the rare case where it feels like a bone is broken, don’t be stoic and force yourself to walk on the broken foot. Use crutches for as long as necessary or even stay home and lie on the couch for a few days. Leave the stoicism behind.

We were surprised at the high degree of fear that some PDers had in anticipation of feeling old injuries. Some were afraid to let us touch their feet. One person traveled to Santa Cruz to be treated, knowing full well that the treatment consisted of foot holding. But when I asked her to remove her shoes, she balked. “Not even my husband has ever seen or touched my feet.” She went back and forth for about half an hour deciding whether or not she wanted me to even look at her feet. Then she made up her mind. She left. I never saw her again.

Others were terrified of what they might feel in general if they let their guard down. One PDer spoke for many when she explained to me, “The whole point of life is to avoid pain.”

Those PDers whose stoicism was zero or minimal tended to recover more quickly. They sometimes mentioned that they had been very stoic and guarded at the time of injury, or throughout childhood. But if they had learned, in their adult life, that it is good to experience sensations and that it is not “bad” to feel and respectfully tend for the body’s injuries, they responded more quickly to treatment. Some of these emotionally mature patients had consciously worked at learning, as adults, that it’s OK to ask for help. A few had even taught themselves how to cry. The PDers who were able to cry and mentally focus on the actual sensations of their own physical and emotional pain had fewer problems figuring out how to deal with any pain or bruising that arose when the foot healing began.

Many PDers, when starting treatment, were still proud of their ability to not feel pain. Some had stopped crying in childhood and had not been able to cry since. Some truly believed that non-feeling or perpetual wariness was superior to acknowledgement of physical or emotional pain. In general, these people needed more time with the foot holding before the foot injuries started to manifest. Often, these people were alarmed or at least anxious when the appearance and sensations of an old injury began to manifest. Although the pains were not necessarily severe, a few even veered into bouts of hysteria and paranoia when confronted with pain. It seemed as if they had no ability to face pain and process it. One patient had full-blown bouts of hysteria every evening for several months after he started feeling the injuries in his foot.

Then again, one (and only one) patient, within hours of being *diagnosed* with Parkinson’s disease, developed the same sort of daily hysteria, and it lasted for several months. He developed this hysteria a full month before he heard of our work and started being treated by us. His hysteria finally eased up after many months of foot treatment, psychiatric counseling, prescription-strength anti-insomnia drugs, and homeopathic remedies.

Eventually, we realized that many PDerers literally do not know how to deal with pain or fear. They may have never learned the usual pain assuaging mechanisms that young children slowly master during the years from around age three to age eleven (approximately). For many of our PD patients, the only fear- or pain-treatment mechanism they'd ever known was mentally blocking out the pain or fear – pretending that it wasn't there. The concept of *confronting* fear or *feeling* pain and consciously neutralizing it was absolutely new to them.

Looking ahead, chapter xxx explains how a healthy child learns to process pain so that the pain can quickly become mere sensation instead of being terrifying and seemingly life-threatening. Chapter xxx includes instructions on learning to correctly process pain for PDerers who truly do not know how it's done. But in the early years of the project, we just gaped in wonder at these strong, competent people who said, in all sincerity, "I hurt! What should I do?"

A lengthy aside: pain from other injuries

Some PDerers experienced pains in *other* parts of their bodies as they began to recover from dissociated foot injuries. These pains turned out to be other injuries that had also never healed. Sometimes the other suppressed injuries appeared at the same time that the foot injury started to show signs of bruising and swelling. Sometimes the other injuries appeared weeks, months, and even years after the foot injury healed.

Very often, the PDerer recalled the incident(s) that probably caused the other re-appearing injury(s). I was told things like: "I'll bet this hip pain goes back to when I fell two stories and landed on my hip; it sounded as if a bone had broken but my hip *never hurt*," or "Oh yeah! Eight years ago, when I moved the stove by myself, I thought I twisted something in my neck and I heard something pop, and my clothes have sat crooked at the neckline ever since, *but it never hurt*,"

Then again, sometimes a PDerer had no recall of any injury at all in the non-foot area that spontaneously manifested a bruise or soreness. It does not matter whether or not the PDerer is able to remember the painful incident. Some PDerers have had so many injuries that there is no way they will ever sort out which incident caused which injury. That's fine; this lack of memory will not impede recovery.

Some of the non-foot injuries only showed up as faint bruises with no pain. Some were full Technicolor bruises, featuring blues, yellows, and greens, with streaks of fresh red under the skin. Sometimes there was pain with no bruise. Sometimes there was bruising *and* pain.

Although unhealed broken leg bones were relatively rare, the people who had them experienced a tremendous amount of pain and swelling at the site of the break. Sometimes, in cases of very severe pain from a broken leg, body-wide tremor accompanied the pain and shock for a short time – as it should.

Staggered recoveries

Although some PDerers had arm, leg, neck or head injuries that appeared at the same time as the foot injuries came to the surface, it was more common for the various injuries to show up in a staggered fashion.

Research has proven that a person who receives severe multiple injuries cannot *simultaneously* feel the pain of all of his severe multiple injuries. In these cases, the body seems to "decide" which injury is the worst at any given moment, and addresses that one. When

appropriate attention has been given to that worst injury and healing has begun in that most problematic area, a different injury is then able to receive the body's foremost attention.

A person whose fall down a steep hill has bruised his ribs, sprained his knee, scraped skin from many places and thrown his back out, will know what I am talking about. Even though he may have a general sense that he hurts all over, only one injury at a time will *truly* stand out as being the thing that needs to be addressed immediately in any given moment. And as soon as that particular problem starts to feel better, another injury will move to the fore. It is not uncommon for people with injuries in multiple areas from a really bad accident to spend *months* healing. As soon as the neck pain starts to heal, the hip pain might demand attention. As soon as the hip pain starts to resolve, the knee pain might arise. When the knee healing is underway, the shoulder injury may start to squawk.

In some cases, once the PDer started being able to tune in with his body and feel long-forgotten pain, one or two "new" injuries showed up every few months, or once a year. In some cases, this delayed healing continued for several years even after completely recovering from Parkinson's. The recovery from Parkinson's did *not* require healing of all suppressed injuries. The recovery, including the person's ability to experience steady dopamine function, usually started up as soon as energy was able to flow correctly through the foot. The presence of other, smaller injuries – injuries that didn't interrupt the Qi flow in a significant manner – did not usually appear to inhibit the recovery from Parkinson's.

Summary of pain

We saw that old, unhealed injuries sometimes still had their pain trapped inside, as well as their bone breaks, tissue tears, and structural displacements. When the PDer began to mentally pay attention to the injured area, these pains, breaks, tears, and displacements became knowable, feel-able. PDers sometimes needed to learn how to process pain. For some PDers, the idea of confronting pain was terrifying, even life-threatening. For others, particularly those who had consciously worked on learning how to feel safe while experiencing sensations and emotions, the pain was not alarming – it was just pain.

IMPROVED CIRCULATION

Following the healing of the old foot injury, the next recovery symptom was very often an improvement of the blood circulation in the foot. The foot color often improved significantly. Blood vessels in the feet often became larger. Temperature regulation in the feet improved.

Before starting treatment, the skin on the feet of many PDers was mottled, sometimes purplish, or even a ghastly gray, with no or few distinct blood vessels. If a large vein *was* visible, it often formed a semi-circular pattern on the top of the foot instead of flowing all the way from the toes. Some PDers' ankles had severe staining (dark reddish brown discolorations) or angry, bright red varicosities. Following recovery from the foot injury, the skin color became healthier and healthy blood vessels stood out more. The semi-circular vein developed branches feeding into it from the toes or a completely new set of healthy veins appeared that fanned out over the toes. Sometimes, the dark "staining" lightened up a bit and angry-looking varicosities diminished in size and intensity. These changes sometimes coincided with or foreshadowed an improvement in temperature regulation in the feet, or even the hands.

Many PDers who'd had cold feet for years deeply enjoyed the novel sensation of having feet that could warm up easily after having been subjected to cold.

This type of lasting cold in the extremities, also known as Reynaud's syndrome, is not unusual in people with Parkinson's disease. One recovering PDer used to fear getting his feet cold because they remained cold for hours, if not days, after a severe chill. When he started to recover, one of the first behavioral changes that he shared with me was that now he would often sprint barefoot out to the snow-blanketed mailbox to pick up his letters and then scamper back into the house. He didn't mind that his feet got cold; rather, he was thrilled because, after getting back into the heated house, his feet would quickly warm up again! He was as pleased as a child at the novel sensation of his feet getting warm all by themselves after having been briefly chilled.

THE RETURN OF SENSATION

Another recovery symptom that was often set in motion by improved circulation was the cessation of numbness.¹

¹ Prior to entering the recovery program, many PDers insisted that their toes were not numb. As proof, they pointed out that they could feel their toes if they stubbed or touched them.

However, their proprioceptive awareness of their toes and their internal sense of how the toes feel (a concept that many PDers do not even understand) may be completely lacking. Their toes are usually quite numb. I have done many experiments with PDers in which I have been able to prove this numbness. So has Chris Ells, one of my colleagues.

Chris did his doctoral research evaluating numbness of the feet in people with Parkinson's. As part of the research, he also asked PDers whether or not they thought they had diminished sensitivity in their feet. Most PDers felt that they had reasonable, even normal, levels of sensitivity in their feet.

When the side of the body that first developed symptoms of Parkinson's was tested with a longish acupuncture needle inserted into Spleen-3, a famously painful point near the big toe, most of the PDers made rather a proud show of stating that they were able to feel the needle. When a matching needle was then inserted into their other, healthier foot, the typical response was much stronger: a sharp startle response, often followed by the slightly concerned query, "What are you *doing*?" When these PDers were told that the two needle insertions were exactly the same, and had been performed to determine relative numbness, the PDers, often, were stunned.

What they did not know is that, in a healthy person, such vigorous needle insertion at this particularly electrical location should evoke such a strong reaction that, very often, a healthy person lying down, relaxing, will jerk bolt upright in response to rude needling at this point. None of the PDers ever had such a powerful, visceral, *normal*, body-wide response to abrupt, powerful needling at this point (SP-3) until they began to recover.

When I taught a class on Parkinson's at the local acupuncture college, I would often needle new PD patients at SP-3 as part of their intake, to help me determine how numb their feet were. Many a student, knowing the extreme, body-jarring shock of strong needling at this point, would brace himself as he saw me picking up a fairly large needle and jamming it with a lot of power and wristy follow-through into the side of a PDers' foot.

Students usually looked worried, at first, thinking of the pain that I was going to inflict on these patients. The worry changed to stark amazement when the PDer failed to physically react against what should have been a powerful electrical shock. The PDers could overhear me telling the students that I was doing this to check for numbness in the feet. Not knowing the correct response that a healthy person would have to strong needling at this location, they were usually outspokenly proud of their keen ability to detect that I had touched their feet and inserted a tiny needle. Many said something along the lines of "I felt that. I could feel your fingers on my skin and I felt that you were putting a needle in: I'm *not* numb."

The true proof of their numbness came when feeling returned to their toes.

I think my favorite quote on this subject came from the recovering PDer who asked me, in amazement, "Did you know that a person can feel his own toes even when they're inside his shoes?! Did you know that toes can tell whether or not they have socks on *without even looking*?!" Of course, prior to starting the recovery program, this PDer had assured me that his feet were not numb in the slightest.

The sheer amazement in his face and voice as he asked me these innocent questions told the story: for as long as he could remember, he had never been able to actually know of the existence of his own toes if they were hidden by shoes. He had not been able to feel the soft texture of his socks against his skin. When he had started the program, he insisted that he could feel his feet: if he touched his feet, he could feel the touch of his hand – therefore, he did not have numb feet. However, if he was not touching his feet, he had no awareness of how his feet *felt*.

As PDer's foot circulation improved, foot areas that had been numb, such as the distal (toes) end of the foot and particularly the three more medial toes (the "big" toe, second, and third toes) experienced a return of sensation. In some cases, the sensation was mildly pleasant. In most cases, however, the return of sensation to long-numbed nerves was somewhat painful. Common PDer descriptions of the foot sensations that occurred during recovery from numbness were "tingling," "burning," "pins-and-needles," and "like the pain of recovery from frostbite."¹

One particular area on the foot tended to experience a very powerful sensation during recovery: the medial side of the ball of the foot (see the acupoint SP-3 in fig. 3.7, page xxx.) Many recovering PDer's reported a particularly strong stinging sensation right at this spot. Patients have said things such as: "I was certain I had a splinter in my foot. I took my shoe off six times to try to find the splinter but there was nothing there," or "I thought a bee was stinging me; I took off my shoe several times but there was no bee. The sensation lasted for hours."

However, some of the tingling that occurred when the nerves come back to life was less site-specific; the sensations came and went or moved around a bit. Many people described the sensations as "ants moving around in my toes." Another description was "someone's dragging a miniature rake back and forth over the top of my foot and my toes."

Interpretations of the "tingling sensation"

The emotional response to the reawakening of foot and toe nerves varied from one PDer to another. Some recovering PDer's found the tingling sensations to be amusing or even thrilling: proof that change was underway. Others were fearful that the pins-and-needles sensations would only keep getting worse, or even that the new sensations of pain were being caused, not by anything healthy, but by cancer, gout, imbedded nails or broken glass, or some rare and incurable foot disease. Some people enjoyed the reassuring feelings of once again having toes. Others found the awareness unfamiliar and even frightening.

As to the intensity, a few recovering PDer's told me that they were so shocked by or fearful of the stabbing sensations in their feet and toes that they screamed out loud during the more dramatic moments. Others said things along the lines of, "It was no big deal," "You got me worried for no reason," and "It's nothing; why did you even put it in your book?"

I cannot begin to share all of the descriptions and variations on the return of nerve function in the feet and face.

In general, people who were emotionally closed off or mentally oriented towards cynicism, negativity or self-control tended to experience the return of nerve function as terrible, painful, and even excruciating. Those people who were more curious than frightened tended to enjoy, or at least not mind, the sensations.

As the Bard's Hamlet says, "There's nothing either good nor bad, but thinking makes it so." The sensations of recovery from Parkinson's are merely that: sensations. Whether or not they are perceived as augurs of health or omens of suffering seemed to depend entirely on whether or not a person was in a predominantly positive or negative mindset.

Another sweet remark from a PDer who regained proprioception and feeling in his hands – even though, prior to recovery, he'd insisted that his hands weren't numb, was, "My fingers have eyes again! I can see where my fingers are even when they're inside my sleeve!"

¹ Frostbitten toes are actually painless – so long as they remain frozen. In fact, they often feel perfectly normal until they warm back up. *Recovery* from frostbite, on the other hand, can be quite painful.

Increased awareness of rigidity

Often, along with a cessation of numbness came an increased awareness of rigidity. This rigidity might have been there right along, but had not been felt. For example, many PDers had not realized that their wrists or ankles had been moving in a cogwheel motion until their doctor pointed it out. They couldn't feel the wrist or ankle very well (they were numb), so they didn't realize the full extent to which the ankle or wrist was rigid.

Due to improved circulation and the concomitant return of nerve sensitivity, many PDers noticed that their bodies were far more rigid than they had realized. As one person said, "I can feel my feet better and I'm walking better and faster, but I never knew my legs were so heavy and stiff!" Another patient said, "I think I'm getting worse. I have a limp." In fact, she had been limping since before she started receiving treatment. When reminded of this, she admitted that, for decades, she had *heard* herself limping, but had never *felt* it before.

THE RED RASH

(This section will take a bit more space than is possibly justified in terms of the percentage of people who get the red rash, but the fungus situation is significant to our project because it provides still more proof for our hypotheses.)

Many PDers have foot and/or toenail fungus. The toenail fungus causes the nails to be thick and gray, or even white. The foot fungus may blossom (present an itchy, red rash or sores) during the summer months or anytime that the feet are hot. The fungus may become dormant when the weather is cold.

In areas where the body is numb, the fungus can thrive, unmolested. When circulation improves, the body becomes able to notice the presence of the fungus. Once the body notices the fungus, it will take steps to kill it.

Fungus is everywhere. Our bodies are constantly being exposed to fungal spores. The white tip of a healthy fingernail is white with fungus residue: fungus is digesting the underside of the nail. But a healthy person's awareness of his body keeps the fungus from establishing itself in the pink, attached-to-the-nailbed section of the nail. The faint, very thin line of deeper red where the nail goes from pink (attached to the skin) to white (free standing) is the red of a biological battle line. These are the lines where a healthy body keeps the ever-present fungus at bay.

However, the immune system cannot not fight very well in those body areas in which a person has little proprioceptive self-awareness, poor circulation or sensitivity. Fungus can thrive in these areas. Because PDers have very little awareness of their toes and feet, they often have fungus invading and becoming established in the nailbed (inside the toenails) and in the skin of the feet.¹

¹ The numbness, poor circulation and poor body awareness that allows the fungus to slowly move deeper into the body may start decades before a person is diagnosed with Parkinson's.

For example, one PDer remembered noticing, at age seven, about two years after having her foot smashed in a car door, that she couldn't tell where her *fingertips* ended and the air started. She laughingly admitted to me that, in her twenties and thirties, she had taken this as a sign of her advanced spirituality – her body consciousness was contiguous with the universe. She never even realized that she had no idea where her *toes* ended. She developed toenail fungus in her late forties, just two years before she was diagnosed with Parkinson's. She only had fungus on her toenails, not her fingernails. Evidently, her toes were even more numb than her fingers – so numb that she didn't even know how numb they were.

In addition to having fungus in their toenails, people with Parkinson's often have thriving fungus in the skin of their toes, and the dead skin on the soles of the feet. These areas may break out into an itchy red rash when the weather is hot. If the leg is somewhat numb and there is poor circulation in the skin of the leg, the mycelium ("roots") of the fungus sometimes spreads farther up the skin of the leg, but it is usually not able to thrive and become obvious in areas where the blood circulation is sufficient to keep it at bay. Thus, its presence in the *legs* is generally limited to an asymptomatic sub-dermal invasion, if anything.

During recovery, circulation almost always improved quickly in PDers' feet. As circulation improved, it seemed as if the PDer's immune system began to detect the fungus, if there was any, and initiated battle against it. This often caused a reaction in the feet. The fungus, coming under attack, bloomed aggressively, in a final attempt to produce spores before being killed. The "blossoms" of the fungus were tiny, itchy, red bumps. If there were a lot of the little bumps, they looked like a red rash. Sometimes, if they were scratched enough, the bumps opened up, releasing a tiny bit of fluid.

As the fungus came under attack from the body's immune system, itching red sores broke out wherever the fungus was located. We were then able to see if the fungal infection had pervaded the skin of more than just the foot. In some cases, these sores, also referred to by us as "the red rash," extended up the lower part of the leg(s) and, in a few cases, even up the thighs.

The red rash could be hot and itchy. But in terms of treatment, none was necessary. The red rash always went away by itself. Once circulation was restored, the body was able to destroy the fungus that had snuck into numbed areas over the years. Some PDers found that an athlete's foot salve was somewhat helpful during this time, but others said that it was not. Others found that aloe vera lotion was useful in soothing the heat and itch until the battle is over. Others disagreed.

The red skin rash always ceased eventually. Even the fungus in the toenails often decreased, although this took much longer. Watching the healthy new nails slowly grow in, pushing the diseased toenails out, was a cause for gentle celebration. In some cases, the toenails returned to complete health.

I found it interesting to note that toenail fungus is an illness that doctors consider to be treatable but not ultimately curable – just like Parkinson's.¹

When our PD patients recovered from poor circulation in the feet their bodies seemed to suddenly recognize that a fungus was lurking. Their bodies waged war against the fungus, and won. The red rash and itching was a bit unpleasant at the time, but the upside was that it showed

¹ Some patients have asked about the internal (pill form) antifungal medications. Fungus in the toes and feet is not a dangerous condition. Internal antifungal medications, however, are extremely toxic and hard on the liver. In fact, the internal antifungal medications (as opposed to the external-use ointments and salves) are far more dangerous than the fungus itself. Most of the doctors I know personally will not let their own family and loved ones use the FDA-approved *internal* antifungal medications.

When a person uses internal antifungal medication, the *dormant* portion of the fungus doesn't die from the medication. The fungus will resume growth and thrive again within a few weeks after a person stops using the medication. In other words, the anti-fungal medications do not *eradicate* the fungus. The fungus can remain in the body, dormant, as long as a person is taking the medication. Therefore, the medication doesn't actually "cure" the fungus; it just masks the symptoms.

that the body of the PDer was coming back to life. The red rash battle sometimes lasted for a few short weeks. Sometimes it went on for over a month.

Only one PDer had the red rash go all the way to the top of his thigh. He was a neurologist. It was exciting for him to observe this utterly unexpected manifestation of submerged illness. It was clear that there had been something wrong with him that was not related to “dead brain cells,” and that his body was healing from some previously unrecognized numbness. I asked him if he would tell his colleagues at one of the top medical schools in the country about our theories and his treatment. He said that he would not. “They would think I was crazy if they knew I was seeing an acupuncturist.”¹

In closing off this section about the fungus, I want to mention why I have gone on at such length about such a seemingly unimportant side-event in the big picture of recovering from Parkinson’s. We can only hypothesize as to what is actually going on when a person recovers from Parkinson’s disease. Visible recovery changes, such as improvements in vein structures and the body’s restored ability to recognize and destroy pathogens such as fungus help provide the most objective presentation of what is really going on in Parkinson’s disease. The red rash outbreak on the feet and sometimes legs only occurred in approximately fifteen percent of the recovering patients. But even so, we could use this otherwise inexplicable recovery symptom to support our idea that PD is an illness of unhealed injury and the side effects of injury, including numbness and loss of awareness of the injured part(s) of the body.

RECOVERY SENSATIONS IN THE FACE: FEELING, TASTE, AND SMELL

Tingling in the face

As with the feet, the first changes to the face were often improvements in blood circulation. This led to improved color and warmth in the skin. The subsequent tingling, mild stabbing pains, and the sensation of bugs crawling under and over the skin lasted for many weeks, and in some cases, months.

Sensation often returned to the face more slowly and mildly than to the feet. But even if the facial sensations were milder, they were usually harder to ignore. Compared to tingling in the feet, the sensations of spiders crawling through the face and even the hair were often less sharp and painful but were far more startling and/or annoying for some PDers.

A stickler, reading this section, might want me to say whether the facial sensations were more like ants or more like spiders. In general, people who were more defensive or more negative tended to feel as if they had spiders and spider webs whereas people who were more curious and grateful tended to feel as if they had ants and loose hairs. I suspect that, in terms of nerve function, the actual sensation being transmitted was exactly the same. Whether the transmission was interpreted to be ants or spiders was merely a matter of perception: thinking made it so.

¹ We treated him before we understood the risks of the medications. Sadly, this brilliant neurologist became horribly addicted to his medications. During his recovery from idiopathic Parkinson’s, during which time he found himself emotionally unable to decrease his medications, he rapidly developed drug-induced parkinsonism and suffered greatly from adverse effects of the medication, including violent, rapid changes in blood pressure, passing out, and serious injuries from falls incurred when passing out. He was never able to decrease his medications.

When did the tingling occur?

The sensations tended to be intermittent. As with most of the recovery symptoms, these sensations were at their strongest when a person was awake and relaxed – in parasympathetic mode.

Some PDers were worried that they might have recovery symptoms when they were “in public.” This almost never happened. It seemed to us that, whenever a PDer was worried or frightened – and being in public seemed to be a cause for wariness for many PDers – his mind would switch over to dissociative or sympathetic mode. Recovery symptoms rarely, if ever, occurred during these times.

Bear in mind that in all people, not only PDers, parasympathetic (relaxed) mode is when most healing occurs. This is possibly why recovering PDers noticed the strongest symptoms of tingling or crawling bugs during those times when they were relaxing. Very often, the PDers felt their recovery symptoms most strongly while watching TV in the evening or just before falling asleep.

Numbness of sensory function in the face

In addition to poor muscle function, other symptoms of numbness and poor circulation in the face can include diminished faculty for taste and smell, and coldness and lack of sensitivity in the facial skin, including the lips. For example, many PDers dribble on their shirtfronts when they drink from a glass or cup because their lips are somewhat numb. They cannot accurately sense *where* to apply lip pressure to the rim or *if* they are applying pressure.

Just as with the denial of foot numbness, many of our PD patients denied that they had any impairment of taste or smell or tactile perception in the face. They assured me that their sense of taste and smell was perfectly normal. Others knew that their sense of taste, smell, or tactile awareness was declining or gone, but assumed that the loss was somewhat recent.

For example, one PDer admitted being humiliated for many years even prior to diagnosis because she was often told that she had a spot of food was sitting on her lip or that she had a “food mustache.” Also, she had increasingly spilled drops of her beverages down the front of her shirt because her lower lip couldn’t actually feel the contours of the drinking glass. She did not realize that her facial skin was more numb than normal. Instead, she had assumed that she was a clumsy eater. Her son-in-law had even asked her once if she was able to feel the bits of food stuck on her face. Ashamed of her “sloppiness,” she had assured him that she could, even though she couldn’t. At the time, she wondered to herself how anyone could *possibly* feel a bit of food stuck on the face. After recovering, she was able once again to feel when her facial skin was accidentally decorated with food.

When recovering PDers experienced the full range of healthy sensation that returned to face, tongue, and nose, they often asked questions like, “Did you know that wet asphalt has a *smell!*?” or “Did you know that different kinds of tea actually taste different?!” These queries suggested that the person’s sense of taste or smell had been numbed for a very long time. Some PDers admitted that they were experiencing common smells, such as the smell of newly cut grass, for the first time in decades. Again, when a person is numb, he cannot be a good judge of how numb he is: he is too numb to know.

Of course, there were exceptions. For example, one of my PD patients with lifelong extremely poor eyesight had a highly developed sense of smell even though her Parkinson's symptoms were coming on strong. Again, no two PDers are exactly alike.¹

The pleasures of recovery

Whether the recovery symptoms felt like ants or spiders, nearly all patients loved the *results* of these recovery symptoms: return of sensation and function. I have dwelt so heavily on unexpected recovery symptoms: the pain and the tingling. But the short-term symptoms were worth the effort. It was worth a spot of recovery symptoms to be able to experience the return of facial sensory function, and the ability to use the feet correctly while walking. Even in the earliest days of the project, when we could hardly dare to believe that we'd found a cure for Parkinson's, the patients were thrilled at the improvements in their faces and feet.

Family members especially were thrilled to see the return of full facial expression. One of the articles I published included before and after photos of a PDer who had not had facial expression for over twenty years.²

Exceptions to every rule

Then again, a few PDers grew angry when people suggested that they were moving better or looking better. These supportive compliments were interpreted as criticisms of how the PDer had moved or looked in the past.

I remember one recovering PDer insisting that he'd never had a lack of facial expression: he claimed he had *consciously* refrained from stupid smiles – just like the Buddhist monks that he'd briefly studied with, back in his college days. His face had expressed reserve; it *had* never been frozen.

His wife and I did not argue with him. He could not be argued with. Although he was a brilliant professor of sociology, he had the social skills of a six year old – including stubbornness and temper tantrums. He was emotionally utterly unable to deal with the idea that he had ever been imperfect – especially with regard to his facial expression.

But the fact was, over the last ten years, her friends had been increasingly afraid of him because he always looked as if he was glaring with disapproval. After muscle function returned to his face, he was clearly smiling at people once again, even as he continued to assume his lifelong look of unctuous “reserve.” Her friends were so pleased that he was finally “warming” to them, that he had become “welcoming.” His behaviors were actually just the same. So far as

¹ In our experience with hundreds of PDers, only one person developed malodoria during recovery. Malodoria is a condition in which *all* smells are perceived as offensive. When, in response to Tui Na treatment on her feet, this patient became able to detect smells, she was assailed each day by whatever the first smell of the day happened to be. For hours, she could not get rid of the stench of that particular smell, and when it finally ebbed, the next thing she smelled would take its place – and would be equally noxious. This patient was extremely cynical about the bona fides of everyone, and sometimes even paranoid. This was in the very early days of our project: she was taking a high level of antiparkinson's medications. When her foot began to heal and simultaneously her responses to her medications became suddenly violent, she tried for several days to decrease her medication, but found herself emotionally addicted. She dropped out of the program. Malodoria is extremely rare. It occurs when a person is deeply entrenched in sympathetic mode or dissociated. The “cure” is a return to parasympathetic mode. The variations in perception that naturally occur in the different neurological modes is discussed in chapter xxx.

² *Journal of Chinese Medicine*. Sussex, England. 2002, Vol. 69, pp.43-47.

he could tell, he was still giving her friends the same studied look of benevolent wisdom. So when any of us expressed delight with his new smiles, he grew angry, even belligerent.

He was very intelligent and witty. I'd noticed in the early days of treating him that, when he'd told jokes, his facial expression never changed. As he was recovering, his eyebrows, cheeks, nose, and mouth all danced with expression when he told his jokes and witticisms.

After his face recovered full sensation and the ability to move, the only thing that remained unchanged was his inability to admit that he'd ever had a frozen face – or been imperfect in any way.

His stubbornness and temper tantrums were somewhat unusual in our experience. In general, our PD patients tended to be people-pleasers: careful to not show emotion, and careful to prevent anger rising up in those around them. But then, there was a wide range of variations in the PDers' personalities, even if most of them had the characteristic emotional-harm avoidance traits of the "classic" Parkinson's personality.

THE SEQUENCE OF RECOVERY SYMPTOMS

There was absolutely no cut and dried pattern for the recovery symptoms. The following example will demonstrate. One PDer's unhealed foot was treated for nearly a year. After the foot healed, over the next many months, she regained feeling in her foot and her facial expression returned. Workouts at the gym helped restore range of movement in her arms. However, she still favored her left knee and hip and she still tremored. I asked her to take a course of homeopathic pills (Arnica Montana). This particular homeopathic remedy can hasten the healing of injuries by drawing one's attention to the injury. After taking the Arnica for about a month, at an *extremely* low dose, a mass of bruises appeared on both sides of her left knee and her left forearm. These bruises were consistent with the foot-in-the-bicycle-spokes knee-torque injury that she'd had at age seven. These bruises went away over a period of two weeks. Three weeks later, she announced, "I can feel my fingernails! They're like wind-chimes!"

Wind chimes? She had never been prone to flights of poetic fancy. Prior to full recovery, most of our PD patients were more comfortable and experienced with analytical thinking than with metaphor or poetry. I had to ask what she was talking about. She explained, "I can feel the tiny weights of my fingernails. I'm aware of my fingertips. It's as if I have ten little bits of weight at the tips of my hands: when I move my fingers, I feel them moving, fluttering like the little noise-makers on a wind-chime."

I'm including this example because she never experienced any tingling in her fingers prior to the return of sensation. Also, there was no specific sequence of events that could have led us to expect a return of sensation in her fingertips following the discovery and healing of a *knee* injury. And she'd never suspected that her fingertips were numb.

Recovery without tingling or pain

Many people recovered sensitivity and function in body parts even though they never noticed any of the negative symptoms of pain, tingling, or unpredictable muscle movements.

If, in this book, I have dwelled more on the recovery symptoms that did occur in some PDers prior to the return of healthy body function, it is not because bizarre recovery symptoms are the "normal" thing to expect. It's just that, in the cases where body part recoveries had no weird symptoms, there is nothing to write up. I have written up far more about the strange things that happened than I have about the things that *didn't* happen. I merely mention this, and will

mention it several times, because so many of our patients insisted on determining whether or not their Parkinson's was going away based on how many recovery symptoms they had.

The non-PDer reader may think that this is ridiculous: the way to know if Parkinson's is going away is that the symptoms of Parkinson's go away. However, many PDers are so out of touch with their bodies that they don't know whether or not a particular symptom is going away unless their attention is carefully drawn to that one problem. And in the same way that "a watched pot never boils," PDers who were fixated on a particular problem might not notice that all the other symptoms *except* for the "watched" one were going away. This seemed very bizarre to us, at the time. Also, many PDers who resumed perfectly normal movement refused to believe that they were recovering unless an MD said that they were recovering. Of course, no MDs ever said that a patient had recovered. Any patient who recovered had been "misdiagnosed."

One PDer who attended the free clinic once a week for just over a year was thrilled when her dragging leg, tremor, bent arms, facial masking, slowness and rigidity slowly went away. However, after her victory visit to her neurologist, she was furious – with *us*.

"My MD said that, if my symptoms went away, my problems must have been caused by something like a pinched nerve. I spent all this time coming to Santa Cruz every week, and all I had wrong was a pinched nerve! I never had Parkinson's to begin with! You people should have noticed that from the start!"

Eventually, after about five years into the project, we slowly began to realize the extent to which many PDers did not have any way of knowing how their bodies *felt* – they relied on visual analysis of their movements or the proclamations of their doctors to assess how their own bodies were doing. Over the next five years, we figured out what was going on in these PDers. But in the early years, we only saw that many PDers clung to the "checklist" of recovery symptoms. They used it so that they could analytically determine whether or not they were recovering: they did not have the capability of *feeling* the changes that were occurring in their bodies, feelings that might have confirmed that changes were afoot and that their symptoms of Parkinson's were decreasing.

THE DURATION OF RECOVERY SYMPTOMS

How long did the increased sensitivity and "crawling ants" go on?

It seemed as if the period of *heightened*, excessive sensitivity only lasted until the brain accommodated to the new incoming sensations – a few days or a few weeks.

As with recovery from frostbite, the tingling comes to an end when circulation is restored and the nerves are up and running regularly again. After that, conscious awareness of the toes is possible – and is situation dependent.

After the brain became re-accustomed to getting incoming nerve signals from the feet, it behaved as it does in healthy people: awareness of the feet only rose to the consciousness when something significant – positive, negative, or otherwise – was occurring to the feet. For example, a healthy person might not notice his feet during the course of the busy day working at the computer. But when he's on vacation, he might find himself focusing on the luscious feeling of warm beach sand sifting through the toes.

The timing of the tingling sensations was varied. For example, over a period of days or months, a PDer might have experienced a few *moments* of tingling, followed by a period of

calm. Or the tingling might have occurred a few *minutes* at a time. Or there might have been a period of a day or two – or several months – during which the feet or face seem to be feeling stabs of spiders, ants, or pins and needles for *hours* at a time. In other words, anything was possible. Again, the timing of the recovery symptoms had an enormous span, ranging from moments to months.

Asymmetrical healing

Many PDers noticed that most of the recovery from numbness occurred first in the healthier side of the body. If the PD symptoms had started on the right, the recovery symptoms might occur first on the left. It seemed as if, when the Qi started to flow correctly and the body started healing all the damaged bits, the parts that were less damaged recovered first. The recovery symptoms on the more damaged side of the body were often more severe and took longer to heal. Sometimes, the healthier side of the body would be completely returned to health and vigor before the side where symptoms first appeared even started to experience the pains, warmth, tingling, and the new movement that indicated a return to function.

Sometimes PDers were sometimes alarmed that their “healthy side” was tingling or wanting to move by itself. They assumed that their Parkinson’s disease had spread to the healthy side. Considering that the recovery symptoms were the exact opposite of Parkinson’s symptoms, this fear seemed illogical to us. But we still didn’t appreciate that some PDers were better able to anticipate problems than anticipate health or joy.

Attitude

Many PDers, prior to entering the program, have insisted on knowing exactly what their own recovery symptoms will be like. Of course, it was absolutely impossible to guess, and we never even tried. We soon realized that there was no way to predict who would experience what during recovery. We also learned that many PDers were *not* happy with my statements that such and such a phase of recovery may or may not be painful, and may or may not linger for an unknowable period of time.

Some PDers wanted as much hard, precise information as possible. They often wanted to know *exactly* what it was going to feel like and just how long it would last, so that they could be prepared for it. But the symptoms of *recovery* from Parkinson’s seemed to be as variable as the symptoms of Parkinson’s itself. But this just made sense: no two PDers had the exact same set of symptoms, or the exact same mindset.

However, in retrospect, I can say that attitude seems to be the best predictor of whether or not the recovery symptoms will be fascinating or hellish. An attitude of curiosity and gratitude allows one to see the symptoms as wonders, miracles: harbingers of recovery from an “incurable” illness. As for the “pain,” the PDers who felt safe noticed *sensations*, not *pains*. Oppositely, those who were more fearful or negative had symptoms that were ominous, painful, and anxiety-provoking. The latter ones did not think that recovery symptoms were necessarily signs of improvement – let alone recovery. They often told us that, just because they *seemed* to be improving did not mean that they were going to be “one of the lucky ones.”

DOUBTING THAT RECOVERY IS REAL

Fear when the tingling stops

Some recovering PDers actually worried when the tingling and/or the *heightened* awareness of the feet and face came to an end. When the tingling stopped, they pessimistically assumed that their feet had gone dormant again. Some even decided that the injury must have somehow returned, and that they therefore would never be able to recover – as they suspected from the start.

When a few PDers first said things like, “The tingling in my face has stopped, so even though my face has full expression again, and my sense of smell and taste has returned, I guess I can’t recover after all,” we were puzzled. This fairly common tendency to jump to negative conclusions seemed unwarranted and even illogical.

Over the years, after running many experiments, we were able to prove that this negative mindset plays an active role in maintaining the selective dissociation from body parts or from the heart that, in some cases, prevents full recovery. And dissociation from the heart contributes to a negative mindset. This negativity was not present in all PDers, and it was present to varying degrees in those people who had it. But in the beginning, we were baffled by these strangely negative conclusions. It seemed so strange to hear adamant statements such as, “Since my foot isn’t tingling any more, I’m going to be one of the people that can’t recover,” coming from otherwise intelligent, analytical PDers.

It took several years, during which we saw the positive-attitude people recover easily and the negative attitude people experiencing weird, mood based, come-and-go symptoms, before we understood the extent to which negative thoughts can become self-fulfilling prophecies.

Since these people had already started experiencing some recovery symptoms, we asked them why they were so determined that they could never recover. We suggested that they could get a foot massage, *relax* (if possible), and really focus on the sensations in the feet. If they did this, they realized that their feet had become far more sensitive than they used to be. Of course, if they were worried or wary, their awareness of sensitivity in the feet naturally and correctly decreased – because their minds were preoccupied with thoughts instead of noticing sensations. But despite the proof that they now could feel more sensation in face and feet by relaxing and paying attention to these areas, many PDers decided that, when the pain or tingling came to an end, they could not recover.

Also, many quickly forgot that they had ever experienced these changes. One PDer saw me once a month after the foot injury healed and she started to recover. But after about six months, she said to me, “I don’t see why you’re wanting to work on my attitude, you’ve never gotten rid of my foot injury. I’ve never noticed any improvement in my feet.”

I flipped back through her chart to her sixth week of treatment. “Do you remember saying, on June 4, “After the last treatment, my right foot hurt right across the top of the arch?”

“No.”

“Hmm. Do you remember saying, on June 11, “I’ve had a sharp pain at the base of my third toe all week.”

“No.”

“Well, do you remember saying, on June 18, “My foot pain is gone and my feet feel warmer lately, it feels really good.”

“No, but I believe you.”

“Do you remember saying, on June 25, “This last week, I stepped on a small piece of kitty kibble with my bare foot. It *really* hurt! There’s no way I would have been able to feel something like that in the past. It really *hurt!*”

“Well, OK. I’d forgotten about that. So why did I stiffen up when I found out that my grandson was going to prison? I’ve barely been able to walk since then, and my tremor’s gotten much worse.”

“Right. So let’s get back to work on the dissociation mindset you’ve always used for coping with stress.”

Despite experiencing recovery symptoms, the more skeptical PDer usually assumed they would never recover – and they didn’t, except for during those hours or days when, as they usually put it, they “forgot” to have Parkinson’s disease.

Some PDer who had only brief, minimal recovery symptoms decided that they hadn’t been through enough to *really* recover. They were certain that recovery should be dramatic.

One PDer, during his weekly visit, told me that he’d called all his old friends in New York over the weekend and told them that he was getting worse. Minutes earlier he had told me that he was doing much better, and he could hardly believe how much more feeling he had in his feet, and how much his walking had improved. When I asked him why he told his friends that he was worse, he replied, “There is no way I can be recovering so quickly. If I’m actually recovering from Parkinson’s disease just because you are holding my feet, well, I’m not ready to believe that. So I told my friends I was getting worse. I don’t want anyone to have false hopes.”

Some of the people who doubted their ability to recover, in spite of cessation of Parkinson’s symptoms, assumed that the Parkinson’s had merely become dormant. Why? Because they hadn’t experienced as many recovery symptoms as other people. These were the ones whose PD symptoms could go away for a long time, but when some distressing life event occurred, they would manifest their old symptoms again. The resurrection of these symptoms was usually deemed “proof” that they were never going to recover. The emotional blow of this “proof” often resulted in rapid worsening of symptoms.

We had to wonder. In these cases, was the short-term (weeks, months, or even years) *recovery* merely psychosomatic, or was the Return of the Parkinson’s psychosomatic? Something didn’t make sense. If Parkinson’s was based purely on physical problems, it shouldn’t come and go for long periods or be triggered by mood or environment. But if it was purely mental or emotional, how could we explain the similarities in the physiological recovery symptoms that unsuspecting PDer experienced after their foot injuries healed?

And yet, it was clear that PDer who doubted that they could recover also needed more time to recover – or they got stuck in partial recovery.

Illogical responses to pain

A few PDer who were doing well in recovery suddenly fell to pieces when some unexpected place in his body started to hurt with a nagging or a blinding pain. As they experienced the pains from unhealed, long-ignored childhood injuries, some of them started to tremor from pain and fear. They sometimes instituted a high level of body-wide dissociation, setting in motion a high degree of rigidity and extreme poverty of movement. This was somewhat understandable, but not helpful. Sometimes, in response to pain, they panicked and started talking antiparkinson’s medications; this was *not* reasonable.

Taking antiparkinson's medications to deal with severe pain from an old injury is a completely inappropriate use of this mind-altering type of medication. I have seen people start taking these medications to treat pain and then, succumbing to the lure of these extraordinarily addictive medications, cheerfully drop out of the recovery program. I've seen this many times.

The antiparkinson's medications are *not* designed to treat pain, reduce swelling, or accelerate healing. Most of them do not help with tremor, either, unless taken in doses so high that they cause the person to be stoned, out of touch with reality, and even *then* they do not always help treat the tremor. If they *do* ease the tremor, the benefit is short lived, and wears off as soon as the meds wear off. The Parkinson's meds are *mood* drugs, not anti-inflammatories.

Most PDer's in our program knew this. But even knowing this, PDer's who got slammed with unexpected pain often insisted that the pain must be coming from Parkinson's disease. This makes no sense, based on any reasonable understanding of Parkinson's, and yet the panic that accompanied the pain often led many PDer's to conclude, illogically, that the Parkinson's had suddenly returned or suddenly gotten much, much worse.

What the PDer very often could *not* do was say, "I'm in a lot of pain here. I will see some doctor who treats this type of pain (chiropractor, homeopath, acupuncturist, massage therapist, MD or naturopath). I will get an X-ray or CAT scan to learn what is causing the pain. We will figure out the best way to deal with the pain *and* the injury that is causing the pain. I might dash to the drug store for some homeopathic pills for injury or maybe I'll get some aspirin. I might also contact the doctor about something stronger to ease the pain, and maybe even something to bring down the swelling.

It seemed as if many PDer's were not even capable of *thinking* about the choices listed above. When a recovering PDer experienced intense pain from an injury he had received decades earlier, he almost never thought of how to *face* the pain and treat it. When pain appeared, many PDer's behaved as if death were imminent. Also, some PDer's were terrified of other people knowing that they were in pain, as if this would be a great risk. Sometimes they became rigid or tremored in response to the new pain, even if they had recently been becoming more fluid in their movements. They seemed incapable of understanding that most people freeze up, and some even shake, in response to severe pain – they interpreted the new rigidity as a sudden worsening or a reappearance of the Parkinson's.

They assumed that what was needed, after all, was antiparkinson's medications.

Sometimes, despite the pain, the PDer was still able to move better than before. Maybe his tremor was less and he was completely aware that his Parkinson's symptoms *were* ebbing. And yet, purely in response to the *pain*, he concluded that he needed antiparkinson's medications to treat the *pain*. This made no sense, but we saw this many times.

When it came to the ability to process pain, PDer's ran the gamut from mature and sensible to immature, almost infantile. The PDer's who were able to calmly feel their pain and decide how to treat it were also the ones who recovered more quickly. The ones who flew into a state of panic or who dealt with the pain by ignoring it or mentally blocking it out got stuck in what we eventually named "partial recovery."

Across the board, our Parkinson's patients were extremely intelligent and highly analytical – some of them, excessively so. But with regard to their ability to feel and process their own physical or emotional pain, some of them seemed emotionally arrested. In many cases,

the emotional immaturity was all the more striking because of the extremely high level of intellectual or analytical development.

Emotional immaturity

When some of the PDer's experienced the symptoms of a long-suppressed injury that dated back to childhood, they responded in an emotional manner that seemed more suited to a paranoid early childhood than to adulthood. Some assumed that no one could or would help them; some assumed that they would be at risk if anyone learned about the injury. It was almost as if these PDer's dealt with pain by using their *childhood* minds. In many cases, their childhoods had not featured a lot of emotional support.

Others responded oppositely, as if the pain allowed emotional doors to be thrown wide and they were finally able to express repressed rage or hurt. Some threw tantrums; others sulked.

One recovering PDer threw furniture at her husband, locked herself in the bathroom, and generally acted like a three year old – the age at which she'd been put up for adoption.

Another example

Another, according to his spouse, behaved after his birthday party “exactly like a nine-year old.” The PDer had used the *exact* same sentences that their child had used twenty years earlier after his fabulous 9th-birthday party was over: “Is that all I’m going to get to do for my birthday?” and “I never get to do what *I* want.” Over several more months, we all realized that this particular PDer’s life-changing shock had occurred when he was nine: at age nine, he’d come home from school and discovered his grandmother, dead of a heart attack, on the living room floor. He got out his Boy Scout book and gave her all the various treatments, including mouth-to-mouth resuscitation. He was still trying to revive her when his mother finally got home. The mother heaped praise on him for not having become emotional or given in to feelings at that time. For the *rest of his life*, his mother regularly praised him for not having given in to any feelings on the day that Grandma died.

Prior to starting to recover from Parkinson’s, this patient could not recall any negative events that might have led to emotional shut-down. To his mind, his behavior with his grandmother had been heroic and a high point in his life, for which he had received regular praise. It was only when he started to recover and started acting exactly like a nine-year old that his wife recalled the dead grandmother event and we put two and two together.

Even as many PDer’s *emotions* responded in ways that would have been more appropriate coming from a child, many PDer’s adult *minds* were fixated on the Dreaded Parkinson’s Disease. Therefore, when they started to hurt, they 1) didn’t know how to comfort themselves and help the healing along, and 2) assumed that all problems must be coming from the Parkinson’s.

Even though most of them had already memorized the list of symptoms that defines Parkinson’s disease, they usually assumed that *any* new problem, including pain from an old injury, must be a symptom of Parkinson’s disease. This made no sense. In most cases, no matter how many times I told a panicked patient that that the sudden appearance of pain and a large bruise in the spot where an injury had previously occurred was *not* a symptom of Parkinson’s disease, he flat out wouldn’t believe me.

Some of these people were convinced that, following a diagnosis of Parkinson's disease, any and all problems *must* be caused by Parkinson's. These were highly intelligent people, people who had read everything they could about Parkinson's disease. They knew what the symptoms of Parkinson's were, but when old injuries appeared, they panicked. These people often decided to drop out of our program and start taking the antiparkinson's medications that their doctors had offered them several months or several years earlier.

The reader may be thinking that this makes no sense, but we saw this over and over. The onset of pain in an area that had previously been numb was just one of the many recovery symptoms that many PDer's chose to misinterpret. But PDer's are not alone in this: some doctors also misdiagnosed throbbing, inflamed, injury-type pain as a symptom of Parkinson's disease – a disease that is characterized by numbness. These MDs should have known better. They should have known that most of the pains that do occur in Parkinson's are due to steadily worsening stiffness and rigidity – they are *not* pains from inflammation and injury.

An example

One patient who had thought she was recovering developed agonizing pain in both hips. The pain began when she first became able to imagine herself having hips. Instead of seeing a pain specialist, she went to the neurologist who had diagnosed her with Parkinson's. This patient was *not* taking any antiparkinson's medications.

Her neurologist, thinking of her only as a Parkinson's patient instead of a patient in pain, prescribed a powerful anticonvulsant (anti-epilepsy drug). This drug is often used to sedate brain activity in PDer's *whose medications are at excessively high doses*. High doses of dopamine-enhancing antiparkinson's drugs can cause enormously powerful and painful muscle dystonias. To counter the excessive level of brain activity that triggers these spasms, anti-seizure drugs are sometimes prescribed for PDer's *who are taking high, dystonia-inducing levels of antiparkinson's medications*.

This patient was not taking any medications. She was not having dystonias (muscle spasms). She was not rigid: she was exceedingly limp, a recovery condition that you will read about in the next chapter. Therefore, the anti-spasm medications prescribed by her neurologist were completely inappropriate. For over a year, she took the anti-convulsants that her neurologist gave her. The drugs made her even more limp, extremely groggy and confused. Because these drugs inhibit brain activity, they did reduce her pain a little: a *very* little. However, she was determined to keep as active as possible: she didn't want to "give in" to the pain.

After a year during which the almost paralyzing pain continued, she finally went to see a pain specialist. He took CAT scans of her hips, and discovered that both her psoas muscles were badly torn in the hip area – torn right through the middle of the muscle, from side-to-side – and inflamed. He prescribed medications to reduce the swelling and instructed her to minimize activity that used those muscles. As soon as she saw the scans, she suspected that these extremely unusual types of muscle tears had probably been received at age six during her father's "tickling games" in which he would have her lay down on the floor with her legs up in a fetal position. Then, he would force her knees apart to the sides. He would press her bent knees all the way to the floor and not let her get up until she cried. The "game" was to see how long she could go without crying.

This PDer was a highly educated therapist. She told me that she had no emotional feelings one way or the other when she realized that her father's "games" had probably caused

the hip muscle tears. I asked her if those “games” might have been a little inappropriate. She replied firmly, with no apparent warmth, “I loved my father. I looked up to him. *Everyone* admired him.”

Finally, after she started getting appropriate treatment and stopped being so stoic and driven, the muscle tears started to heal. It took more than a year for the lateral tears in her psoas muscles to heal completely.

Back to the point, she spent a full year in agony because she and her neurologist assumed that her pain was being caused by Parkinson’s disease. Her error is understandable: she trusted her doctor. The neurologist’s error was due to the medical professionals’ tendency to see every problem in terms of one’s own specialty. The neurologist, assessing her in terms of overmedicated Parkinson’s, assumed that her hip pain was due to either dystonia (permanent muscle cramp) or the violent spasms that PDer’s often get from excessive levels of antiparkinson’s medications, even though she had *no* muscle cramps and she was *not* taking medications.

I’ve written up the above vignette to demonstrate how patients who had been given a diagnosis of Parkinson’s – *and* their doctors as well – tended to assume that every PDer’s problems and pains must all be due to Parkinson’s. While I’m at it, I should mention that many recovering PDer’s who developed flu symptoms when the flu was going around assumed that they felt worse than usual because the Parkinson’s was getting worse. Sometimes, even if they ran a fever, they still insisted that the Parkinson’s was getting worse. And sometimes, their doctors agreed that the flu symptoms must be symptoms of Parkinson’s disease!

Inability to relate to normal aches and pains

Let me compare how a non-PDer and how a negative attitude PDer might respond to an ache or pain. I have *non*-PD patients who are middle-aged or older. Sometimes they come to see me because they are stiff or sore after a bout of unexpected activity such as spending all day gardening or from an habitual lack of activity or exercise. They want relief from their pain, so they get a massage or an acupuncture treatment.

But some recovering PDer’s, becoming stiff or sore after doing an unaccustomed activity or after spending several days doing nothing, panic. They called me to announce that the Parkinson’s is suddenly much, much worse.

Many recovering PDer’s have complained of increasing stiffness in their joints. I often ask them if they are doing daily stretching, or yoga, or Tai Ji, or anything to keep the joints supple. They often ask me why they should need to be doing such activities; prior to having Parkinson’s, they didn’t have such pains and stiffness, even though they hadn’t done stretches. When I point out to them that, prior to recovering, they were pretty much numb, using adrenaline, and only felt stiffness if the joint was so locked up that they couldn’t move it, they struggle to understand my point. I tell them that any person who is middle-aged or beyond, who fails to work at staying loose, is going to tighten up. This idea is usually met with disbelief, or the statement that, “I’ll start doing stretching exercises if the Parkinson’s goes away.”

PDer’s need to learn how to feel their bodies. They *also* need to learn that bodies can be stiff and sore for reasons *other* than Parkinson’s disease. Many PDer’s discovered, during the course of recovery, that a large part of their neck stiffness was due to old neck and shoulder injuries. These injuries often responded very well to gentle styles of chiropractic treatment or

craniosacral therapy. Parkinson's disease might have exacerbated these injury-based problems, but Parkinson's wasn't *causing* them. Many PDers said things such as, "That neck injury occurred decades ago and it never hurt. The Parkinson's is what's making the neck injury become a problem. Therefore, since my neck hurts worse than ever [since the foot injury healed and I've become more sensitive], it's proof that I'm not recovering from Parkinson's disease."

Eventually, we were able to figure out that the *fact* that it hadn't hurt at the time was also the underlying problem in Parkinson's: blocking out pain so that injuries couldn't heal was the problem. But we spent years trying to understand why some patients could not even consider this possibility.

On the other hand, increased awareness of body pains can become a real problem for some recovering PDers: they may develop fibromyalgia and hypochondria.

Swinging from stoicism to hypochondria

Ironically, some of the people that recovered from a Parkinson's went on to develop fibromyalgia or even extreme hypochondria during or shortly after their recovery.

These two illness patterns, fibromyalgia and hypochondria, are syndromes in which a person is oversensitized to every ache and pain. In these two syndromes, a person may be in agony, immobilized, worried, or even fascinated, with every physiological sensation that presents itself.

I do not want to imply that either of these syndromes are purely psychosomatic. However, it is curious that, from a brain point of view, these syndromes are very much the opposite of the stoic, even numbing mindset of Parkinson's disease. In Parkinson's, a person goes through life with a steadily decreasing ability to feel the events occurring inside his body. In hypochondria and fibromyalgia, a person has excessive sensitivity to the events that occur anywhere in his body, and no ability to screen the important pains from the unimportant pains.

Happily, when fibromyalgia and hypochondria did appear during recovery, they were not long-lasting. Within a few months or a few years, it seemed as if the patient's brain centers were reset to a happy medium in terms of stimuli- and pain-recognition.

REVIEW OF RECOVERY FROM NUMBNESS

In review, I will say that, after the Qi blockage had been removed and Qi flow resumed, circulation often improved, and then warmth returned. Tingling or stinging sensations sometimes occurred in areas where sensory nerves had been dormant. Some PDers noticed a rapid decrease in their Parkinson's symptoms at the same time.

It was obvious that these changes did not match up with the old "dead brain cell" theory of Parkinson's disease. These changes made it seem as if one of the causative factors in idiopathic Parkinson's disease might be circulatory changes and dormancy in various tissues and limbs: a dormancy that seemed to be caused by an old, unhealed foot injury; a dormancy that could be reversed when the injury healed up.



“We choose and sculpt how our ever-changing minds will work, we choose who we will be the next moment in a very real sense, and these choices are left embossed in physical form on our material selves.”

Dr. Michael Merzenich, University of California, San Francisco, brain researcher in the field of neuroplasticity,

CHAPTER NINETEEN

RECOVERY SYMPTOMS: RIGIDITY AND LIMPNESS

RECOVERY FROM RIGIDITY

As noted in Chapter Fifteen, body areas in which Qi has been flowing backwards for years can develop rigidity. In Parkinson’s disease, the areas of rigidity match the portion of the Stomach channel that extends from the back of the jaw to the top of the foot and, in some cases, the portion of the Large Intestine channel that runs from the thumb side of the wrist up to the side of the neck. These areas become rigid, not numb. These rigid body parts may feel hard to the touch or as if they are slightly contracted.

In addition to causing a *rigor mortis*-like muscle rigidity, the long-term backwards running Qi in these areas also inhibits the mind-to-muscle connections for these areas.

The primary zone of rigidity in PDers is along the Stomach channel. This contraction pulls the head forward, hunches the torso, and prevents the leg from moving easily. The secondary zone of rigidity, the Large Intestine channel, may pull the arm into a “bent at the elbow” position, pull the shoulder forward, and even pull the shoulder blade laterally.

BECOMING LIMP

Prior to recovery, many PDers imagined their rigid, even rock-hard muscles, and the anteriolateral leg muscles in particular, to be proof of healthy, well-toned muscle. However, these muscles were *not* “toned.” They were perpetually somewhat contracted and wooden. They could *not* relax. They were *not* vigorous.

During recovery, the tissue in these muscles softened. When the backwards-flowing Qi ceased to flow through these muscles, they lost their rigidity. They often became weak, limp or even mushy, for a while. They weren’t able to tighten and loosen in response to brain commands, for a while.

Actually, they hadn’t responded to brain commands during Parkinson’s, either, but because they were hard, they could be used as supports. The other, still-functional muscles could push off against them or compensate for them; and so many PDers had thought that the rigid muscles, especially those in the thigh, were healthy.

Although the softening was most noticeable when it occurred in the thighs, it sometimes was noticeable in other muscle groups that had become rigid. Then again, some PDers experienced an effortless return to healthy muscle tone in all or some muscle groups, with no noticeable period of limp muscles.

Not a question of atrophy

Some PDers wondered if the sudden flaccidity was atrophy. It was not. The flaccidity was due to the absence of the mind-to-body communication in these areas. Backwards flowing current not only creates rigidity in the area it traverses, it also can cause a disconnect between the mind and the tissues in these same areas. This disconnect is perfectly normal in the case of severe injury: the disconnect prevents a person from accidentally, thoughtlessly using some badly injured body part – a part that needs to be temporarily immobilized during the healing process.

But in people with Parkinson's, this mind-to-injured body part disconnect had very possibly been in place for decades. When the Qi began running correctly, the rigidity went away, but resumption of the mind-to-body part communication did not immediately kick in.

During PDers' recoveries, we could observe that the absence of a mind-to-body-part connection, and not atrophy, was the cause of the limpness: as soon as the mind-to-body connection was restored in these areas, the muscles instantly resumed somewhat *normal* tone. There was never any need for PDers to go to the gym to "build" the limp muscles back up. As soon as healthy awareness of the limbs resumed and the nerve connections ended their dormancy, the muscles suddenly had strength again.

"Conscious strength"

After having a few days to relax and reconnoiter, these muscles usually resumed normal tone and responsiveness. However, sometimes it took several months. I started referring to this resumption of normal tone and responsiveness as "return of conscious strength." It was a resumption of strength similar to that which a person gets when he returns to a fully awake state after having been deeply asleep or in a state of shock.

A most difficult phase

This weakness stage was the most difficult phase of recovery for some people. If a person had become mentally unaware of a particular region of his body, healthy muscle tone did not necessarily revive in that region after the Qi started flowing correctly. Instead, that region sometimes remained limp and non-responsive to brain commands for an indeterminate length of time. During this time, the muscles sometimes behaved as if they were made of limp rags.

We later learned that brain researchers have proven that healthy, non-emergency type movement is preceded by activity in the imagination center of the brain.¹ If a person cannot *imagine* himself moving a certain part of his body, he will not be able to initiate movement in that body part. Some recovering PDers were unable and unwilling to even try to imagine their bodies, or certain parts of their bodies. If they were unable to imagine certain body parts, they could not imagine initiating movement in those body parts. Those parts of their bodies became limp.

Sometimes, a PDer had rigidity in some areas and limpness in others. For example, if his foot injury was healing, he might develop limp muscles in his legs. But if he still had an unhealed neck and shoulder injury, he may have painful *rigidity* in his neck and arm while having such extreme *limpness* in his leg muscles that his legs could barely support him.

¹ The citations for the research are in chapter xxx.

It also seemed as if the relaxation that occurred as the foot started to heal made it possible for the PDer to feel more of his overall pain and rigidity than he had previously been able to feel. This made sense: a person who is locked into sympathetic (fear) mode does not fully feel his body's internal sensations, including his full spectrum of pain. When he relaxes even somewhat and reverts to even a slightly greater degree of parasympathetic mode, he can feel some of the pains that had been shut out during his state of mental, emotional, or physical emergency.¹

When combinations of limp and rigid occurred, it was sometimes extremely difficult for the PDers to know *exactly* what was going on.

Those PDers who could mentally relax were fascinated by these seemingly contradictory symptoms, if they occurred. But the PDers who tended towards living in their minds, which is to say, those who could not indulge in an adequate degree of chest expansion and heart-based sensory appreciation (also known as relaxation), or those who were *obviously* locked into fear, usually wanted to know *exactly* what was happening. Since we couldn't say for certain, some of them were terrified by any seemingly contradictory combination of symptoms.

LIMPNESS IN THE BODY, PART BY PART

Neck limpness

An example: one PDer experienced a softening in the rigid muscles in her neck. Within two days of her neck starting to go limp, she couldn't hold her head up. To hold up her head at work, she placed a stack of law books under her chin so that she could read her legal paperwork. Several days passed before she had enough conscious strength in her neck to use her muscles normally.

But another PDer *never* noticed a time when her neck muscles became weak. She only realized that the neck muscles were no longer rigid when she tried to look to her left side while backing up the car. She was accustomed to turning her head slightly to the left by putting lots of strength into the effort. But when her neck muscles loosened unbeknownst to her and she applied the usual amount of strength, she found herself looking *behind* her when she intended to merely

¹ Although high school level biology classes usually discuss the sympathetic and parasympathetic modes as if they were all or nothing, black or white, this is grossly incorrect. Anytime a person is breathing, he is using sympathetic mode. Any time a person is digesting food, he is using the parasympathetic mode. So a person who is breathing during a meal is clearly using both modes. When doctors casually use the term "sympathetic mode," what they really mean is "*predominantly* sympathetic mode." The same goes for parasympathetic.

Some doctors use the term "sympathetic mode" to refer to the *blast* of adrenaline that occurs during an emergency, a blast that causes an abrupt increase in heart rate, bronchial dilation, urine production, and the opening of the pores. This is a rare event – an event that should only occur during a dire emergency. Oppositely, full-blown parasympathetic mode occurs when a person is *feeling to the utmost*, as opposed to *thinking about*, all his ongoing sensory experiences. This condition is also known as relaxation. Many PDers did not know how to trigger the relaxation mode. Unlike healthy people, whose neurological modes, when awake, are somewhere on the spectrum between mostly sympathetic and mostly parasympathetic, some PDers did not seem to have the parasympathetic option. Instead, when some PDers experienced a decrease in sympathetic mode, they shifted into dissociated mode – a mode that seems very calm because it is the numb mode that kicks in when an animal is near death. Some PDers did have the parasympathetic option. Others could move a *little* closer to parasympathetic as the sympathetic declined. But those PDers who were emotionally unable to allow themselves to experience an increase in internal sensory awareness seem to drift towards dissociation when they had a decrease in sympathetic. In these cases, it seemed as if they were moving into a Nothing Land of not sympathetic and not parasympathetic: a condition similar to the pre-death condition of automatic dissociation.

look to the side. With almost no effort, her neck had pivoted as far as any healthy person might be able to turn the head. She had to learn how to moderate her head-turning strength to accommodate her new neck flexibility. But the point is, her neck muscles never became problematically, or even noticeably, weak or limp.

Another recovering PDer went through several days during which her head pulled to the right. This occurred when the rigid muscles on the left side of her neck began to melt while her right-side neck muscles were still rigid. Her Parkinson's symptoms had first occurred on her right side. Her right side symptoms were more severe, and they were also slower to heal. Several months later, the front and right side of her neck went limp and her head flopped gently forward for several days. After that, she experienced no more neck weakness.

An aside: asymmetry

As noted in the previous chapter, left-right asymmetry of recovery symptoms was quite common in recovery from numbness. Asymmetry also occurred during recovery from rigidity. Many PDers noticed muscles pulling to the left or the right as the opposing muscle group became limp. The less damaged muscle group was usually the one that became limp first.

Ankle weakness

Weakness in the area just above and below the ankles sometimes caused swelling in the ankles. This condition did not seem to be related to poor kidney function or any of the usual problems that might cause water build-up in the ankle. The root problem was *not* water build-up, per se. It seemed more as if the muscles above and below the ankle had become so limp that they did not present enough tension to force fluids back up the legs. Consequently, fluids built up in the saggy areas of the ankles. No exercise of this area was necessary to reduce the swelling: as soon as the muscles of this area attained healthy mind-to-ankle awareness, the ankles soon resumed their slimmer, pre-recovery lines.

Leg weakness

The legs often became *very* limp. Many PDers experienced a period during which their legs were so limp that they could not easily get up from a chair or from the toilet.

This inability to stand up due to mushy muscles *felt* somewhat different from the PDer's previous inability to initiate movement – if the PDer was able *feel* his legs, as opposed to objectively observing the functionality of the legs. The upper legs sometimes felt wobbly, or even like pudding. PDers nearly always decided that this new inability to stand up from a sitting position was more of a problem than their previous inability to initiate movement.

Before, when he still had Parkinson's, the PDer might have had difficulty in figuring out how to make his legs move. When the legs turned to mush, he might still have difficulty figuring out how to make his legs move *and* they were limp, to boot. Many PDers felt, at this point, that they had rapidly gone from bad to worse.

More leg limpness difficulties

Whether the condition lasted only a few days or a few months, the leg weakness was sometimes the most challenging phase of recovery from Parkinson's disease. Many PDers found themselves humiliatingly inconvenienced: needing help with bathing and toilet. This phase was exhausting for some caregivers, especially elderly ones. This phase was the one in which many recovering PDers began to doubt the wisdom of trying to recover.

As our understanding of Parkinson's grew, we realized that those PDers who could feel and imagine movement in their legs rapidly overcame this phase: mushy legs started to firm up in a few days or a few weeks. PDers who were unable to *imagine* their legs moving or who could not *feel* their legs sometimes found themselves stuck in this phase indefinitely. The many PDers who proudly said, "I don't do visualization; that's not who I am," might as well have said, "I don't do parasympathetic (relaxed) movement, that's not who I am."

As for *feeling* the existence of their legs, many PDers did not understand what we meant by the word "feeling." A not uncommon statement was, "I don't understand the concept of noticing how my body feels."

We had to explain that feeling is not a concept: it is a type of sensory perception, like tasting or smelling. Feeling, like tasting and smelling, cannot be *understood*; it must be experienced.

We finally figured out that some PDers did not *want* to be able to actually feel their legs or other body parts. Even more shocking, we discovered that some of these PDers genuinely did not comprehend what was meant by the word "feel."

The non-PDer may not understand what I am talking about. How can a person hope to move his legs if he can't feel them or imagine feeling them? The PDer who is reading this may be thinking the opposite: "How can anyone feel the "existence" of his leg? If someone touches your leg, you can feel that touch, but how can a person *feel* the leg itself if no one is touching it or it has no pain? What does the therapist mean when he says, "feel the existence of the leg"?"

- *Taking the stairs two at a time: an aside*

Prior to the relaxing of the leg muscles, many a PDer had been proud of his ability to mount the stairs two at a time, using his legs like rigid rods to hoick himself up. To mount the stairs in this fashion, a PDer leans forwards, getting his center of gravity over the stair riser that is *two* steps up. This is easy and natural, because the PDer's torso is already somewhat hunched forward, bringing his whole center of gravity forward. The PDer then thrusts his leg up and forward by pushing/pulling with the leg muscles that are not along the Stomach channel). Then, when his foot is on the riser that is two steps up, and his forward leaning body is centered over the higher riser, the PDer pushes down against the riser with his forward leg. The downward-pushing motion, which he *can* do easily (since it uses muscles that are not on the Stomach channel, serves to raise the leg that is still resting on the lower riser. As the lower leg swings up and forward, the PDer can very often use the follow-through of this movement to swing the leg up two risers, instead of just one.

Non-PDers, who use the balls of their feet and a forward leg swing to mount the stairs, may be a bit baffled by the above description. But I've described the above to many PDers. They have usually chuckled, saying, "I never thought of putting it into words, but *yes*, that is what I do."

During recovery, when PDers' legs began to go limp, they were no longer able to do this splinted form of leg lifting on the stairs. This special way of using the leg relies on the steel rod-like rigidity in the anteriolateral muscles of the leg. When these muscles became limp, the PDer could not brace against them for his pushing-down motion. For that matter, he very often was not able to lift the legs a fraction of an inch. Going up stairs was out of the question.

Some PDers appreciated that they were changing. Others, insistent that their two-steps-at-a-time feat was a sign of strength, could not accept the idea that most PDers, *even those with very advanced Parkinson's*, can usually mount stairs very well. Even when PDers can barely walk across the room, they can usually do stairs easily – even two at a time. As Parkinson's worsens, the ability to move only when challenged or stimulated makes it almost *easier* to mount the stairs two at a time than a mere one at a time. The very mild sense of challenge and concomitant adrenaline release experienced when confronted with stairs seems to *help* many PDers to mount them two at a time. A few patients with very advanced PD even told me (incorrectly) that their PD must be in the very earliest stage *because* they take stairs two at a time.

(There are exceptions to everything. We did have a very small number of PD patients who'd *never* taken the stairs two at a time.)

When a PDer abruptly lost his two-stairs-at-time ability, it was often deeply upsetting, leading to an immediate worsening of mood-related symptoms (slowness and tremor). On the other hand, it was sometimes – less often – appreciated as being a radical departure from his previous Parkinson's disease pattern.

Another aside: feet sticking to the floor with rigid or limp legs

The inability to lift the legs due to weakness in the anteriolateral muscles of the upper leg was different from the Parkinson's symptom of "feet sticking to the floor." PDers' feet might stick to the floor whether the legs are rigid *or* mushy.

The foot stuck-ness of Parkinson's can be caused either by aberrant Qi flow set in motion by an injury on the foot, *or* by a particular electrical pattern in the feet that is *supposed* to kick in during automatic dissociation – an electrical pattern that causes a terrified person to be "frozen to the spot."

If a person is even somewhat emotionally dissociated from his body, his feet may stick to the floor whether or not the injury is gone. In this type of sticking, the foot can feel as if it's actually attached to the floor, as if drawn by a magnet. Sometimes, the foot feels as if it is being sucked *into* the floor.

Bona fide *weakness* in the legs can also make it difficult to pick up the feet. This weakness does not feel as if the foot is glued or magnetically attached to the floor. This latter situation just feels as if the leg is too limp to raise the foot.

Differentiating between these two situations proved to be difficult for those PDers who could not or did not want to *feel* the sensations in his feet.

Torso weakness

PDers, often well before they were diagnosed, noticed an increased difficulty in rolling over in bed. A recovery landmark that was often disturbing was the *change* in how the PDer couldn't roll over in bed.

Typically, as Parkinson's worsens, a PDer lying on his back may need to turn over in bed in the following manner: he brings the knees closer to the chest and pulls the head further, as if mimicking a fetal position, and then in one strong movement he heaves his whole body, as a single rigid unit, over onto his side. As the Parkinson's progresses, he may need help to do the heaving bit: a strong shove by the spouse will usually shift him.

In recovering PDer's, the rigid muscles of the torso became limp. Some PDer's became unable to pull themselves into a fetal position. Instead, they could only lie floppy or limp on the bed.

It might not have been too difficult for a healthy spouse to shove a rigid PDer from one side to the other. It was *much* harder for the spouse to shift a PDer who had become limp, whose body felt like "deadweight."

This limpness while trying to turn over in bed was clearly *not* characteristic of Parkinson's – a disease that features rigidity. However, the new utter inability to turn over in bed often convinced PDer's that they were worse off than before.

Those with a curious attitude and an ability to feel their muscles appreciated that having the muscle tone of mayonnaise was a shift in the right direction. Some PDer's reported feeling genuine pleasure from the new awareness of sensation in the muscles of the torso and limbs, whether limp or strong. In the past, they had been accustomed to roll over quickly and efficiently without noticing the body sensations engendered by these movements. They began to appreciate a new way of moving: noticing the internal sensations that movement conveyed to the heart and mind.

These PDer's were able to figure out how to turn over in bed, eventually. One PDer described his new "turn over" routine. Starting at the shoulder, he very slowly moved one limp arm in the direction that he wanted to turn over in bed. Then he slowly oozed one leg over the other in the direction in which he wanted to turn. He slowly moved his head to the side. Slowly, moving one body part at a time, he was able to get enough body weight turned to the side so that his hips followed the arm, legs, and limbs, slowly pouring himself over onto his side.

Even doing this "slow motion" style of turn over, some PDer's needed help turning the hips until they regained conscious strength in those torso muscles that are used to turn the hips. But in time, these PDer's learned how to turn over *languorously*, a movement style that many of them had *never* used since childhood.

Oppositely, those who feared loss of autonomy and loss of control were usually terrified by the worsening ability to turn over in bed. As their minds increasingly raced, day and night, through worst-case scenarios, their tremors sometimes became violent, their slowness became glacial, their rigidity became painfully tight.

Some who rapidly developed worse symptoms of parkinsonism insisted that they were not afraid, that they were calm. But when we did mental exercises to hasten the mind-to-muscle connections, we discovered that they were using the word "calm" as though it were synonymous with "emotionally numb." And at the very root of this numbness was an enormous, paralyzing level of fear: in some cases, it was the fear of feeling the physical and emotional pain of life itself. Later chapters will share many PDer's exact words and physical and emotional behaviors that occurred in response to visualization or "feeling" exercises that led us to this chilling realization.

Back weakness

Many recovering PDer's had a spate of back weakness. Two recovering PDer's suddenly lost *all* muscle tone in the small of the back. They were utterly unable to stand up straight. They might pull themselves to a standing position for a few moments, but within less than a minute they were once again bent over from the waist. In these two PDer's, this loss of muscle tone in the back was so severe that the PDer's torso was parallel to the ground – if he worked at keeping his

head “up.” The work of supporting this overhang of torso required them to walk with their legs severely bent at the knees. They shuffled along with their heads a few feet above the ground. If, feet on the floor, they straightened their legs, their faces were facing their knees.

We did not figure out how to effectively treat this situation. We were never able to figure out how to re-activate their back muscles. These PDerS lived far away – one on the opposite coast and the other in Europe. We only saw them a few times. I wish that we’d had more opportunity to work with both of them.

We hypothesized a cause for this very rare pathology. In Chinese medicine, we recognize that the nerves that activate the adrenal gland enter the spine at the same vertebra as the nerves that activate these particular back muscles. Based on this, we guessed that, when the adrenal gland went into abrupt decline (taking a well deserved rest after decades of overuse), the energy in the entire vicinity of this vertebra, including the energy that activates those particular muscles, was also inhibited.¹

A more detailed discussion of back weakness is included in chapter xxx, in the discussion of the fleeting episode of low back pain that *all* recovering PDerS have experienced.

Working with the doctor when the limpness appeared

Multiple System Atrophy

Sometimes, people who were in the midst of limpness went to a doctor to ask, “What the heck is going on?”

Our patient’s were sometimes told, during this mid-recovery visit, that they evidently never had Parkinson’s disease in the first place: what they *actually* had was Multiple System Atrophy.²

Or, if the doctor was less brazen and less well-informed, he incorrectly decided that, in some people, leg and/or torso *limpness*, not rigidity, were the main symptoms of Parkinson’s disease. Or the doctor simply changed the diagnosis to “parkinsonism” (a catch-all term), or even “*atypical parkinsonism*” (an *extremely* vague catch-all term), and leave it at that.

Multiple System Atrophy is an *extremely* rare syndrome in which a person becomes steadily more limp and weak. Multiple System Atrophy is not in any way related to Parkinson’s disease. But if the PDer reminded his MD that, prior to entering a recovery program, he, the PDer, had problems of *rigidity*, not problems of limpness, the MD usually ignored these statements. Some MDs explained patiently to the recovering PDer that it is always hard to get a firm diagnosis in the beginning. No MDs, in our experience, changed their minds about the MSA when informed that the patient had been *rigid* prior to becoming limp.

¹ This posture was so impossibly bizarre that I found myself grateful for Oliver Sacks’ photos and descriptions of this exact same postural problem. In Dr. Sacks’ patient, the posture occurred, practically overnight, in one of his sleeping sickness patients after she was dosed with high levels of L-dopa. I suspect that her extremely high doses of L-dopa (thousands of milligrams per day) may have caused the energy in the area of her adrenal glands to shut down. The same problem of proximity of adrenal gland nerve and small-of-the-back muscle nerve may have been involved. Had it not been for Dr. Sacks’ documentation of this patient, I might not have believed my eyes when my two patients became bent double from small-of-the-back limpness.

² Although MSA is an *extremely* rare condition, I recall a one-month period in which three of my recovering Parkinson’s patients were re-diagnosed with MSA.

It was hard for PDeers to stand firm in their knowledge of what their own symptoms had been when the MD told them that they'd never had Parkinson's after all.

One of the first PDeers who received the altered diagnosis of Multiple System Atrophy was the neurologist mentioned in the previous chapter (the one who became addicted to his medications). When he started to be as loose as a blob of jelly, he asked his doctor for an assessment. His neurologist told him that he'd never had Parkinson's: he'd had Multiple System Atrophy right along. My patient was also a neurologist. He knew darned well that his previous symptoms had not been characteristic of Multiple System Atrophy. His symptoms had been those of Parkinson's. Even so, when his colleague told him that he had multiple system atrophy, he decided he could not trust his own judgment any more. (This was probably accurate: his mental clarity had become heavily fogged by his antiparkinson's medications.) He was willing to accept that his PD diagnosis *had* been incorrect even though, as a neurologist, he had originally concurred in every way with his diagnosis of PD. We never saw him again. Why would he come see us? He no longer thought he had Parkinson's, after all.

The first time this happened, I was surprised. PD and MSA are very different illnesses. After this happened several times, I realized anew the difficulty that doctors have in accepting the idea that a person can have Parkinson's disease and recover from it.

Now, we tell patients that if they *used* to have rigidity and the usual symptoms of Parkinson's, but following foot therapy they become limp and their doctors tell them that they were misdiagnosed and that what they've had right along is Multiple System Atrophy, it is time to celebrate: they no longer have Parkinson's disease. And in all likelihood, *despite* what the doctor says, they don't have Multiple System Atrophy.

Medical reports are not detailed enough

We had to explain to our patients that it was *not* reasonable to expect an MD, who might not have seen the PD patient in six months, to quickly examine him and decide if his symptoms had changed away from their original condition: most MDs do not make thorough enough notes to be able to tell if a person's symptoms have changed significantly, and memory of a fifteen minute visit, six months prior, is rarely precise. The doctor usually made a diagnosis of Parkinson's based on an overall picture of what seemed to be going on after having ruled out, via MRI, brain tumor and stroke. He may *not* have even noted in his records the exact symptoms or the exact location of symptoms that the patient presented. His notes might merely say something along the lines of "tremor, rigidity, cogwheeling, lack of facial expression." His notes may not even mention the *exact* location or the severity of the symptoms.

Therefore, if a patient has a 75% return of facial expression, but one corner of the mouth still turns down a bit, the MD may look at that corner of the mouth and say, "Continuing lack of facial expression." The MD will have no way of remembering that the condition used to be far worse.

We've had patients who'd lost their voice to PD but who subsequently regained the ability to talk, who'd been expressionless but had regained the ability to smile, and who had lost the use of their hands but had regained it again. They eagerly awaited the surprised look they expected to see on the doctor's face, only to be told by their MDs during their next bi-annual visits that "nothing had changed." If they pointed these obvious changes out to the MD, the MD might say something along the lines of: "You're having a good day today" or even, in the case of

one PDer who had never taken antiparkinson's medications in the three years since his diagnosis, "It's nice to see that your medications are finally starting to work."

We also had to remind our patients that the MDs may be accustomed to seeing *medicated* PDers. In these cases, he will *expect* the PDer to be moving better the second time he sees the patient – thanks to the medication. Therefore, he may not be surprised to see the PDer moving somewhat better than before: it's what he's used to. Even if he is reminded that the PDer isn't taking antiparkinson's medications, the significance of this might not sink in.

One recovering PDer who pointed out to his MD that he was once again making his own meals after years of not being able to use his hands at all, and that he no longer used a walker, which he'd needed for several years, was told, "Your symptoms aren't declining as quickly as before: your wife certainly is taking good care of you!"

"Do you want in increase in your medications?"

One recovering, *unmedicated* PDer became almost frantic when, after showing her MD how she could once again use her hands and how she no longer dragged her foot, was asked, "Do you want an increase in your prescription at this time?" The ex-PDer *restated* that she had never taken any of the prescribed medications and that her symptoms were nearly gone. The doctor then asked her if that meant that she did *not* want an increase in her prescription at this time. The patient nearly screamed at the doctor, "Why would I want an increase in my prescription?! I'm not taking any meds. Do you see any PD symptoms in me?" To which the doctor replied, "Fine," and assured her that he would see her in six months, at which time she could request an increase in her prescription if she so desired.

No amount of vehemence or patient explanations have ever, in our experience, caused a doctor to say that a person was recovering from Parkinson's.¹

To be fair, I must say that a few PDers' doctors have said things like, "Your case is baffling," and, "I would have expected you to be much worse by now," and even, "Your other symptoms are gone. If it wasn't for that one arm still not swinging, it would seem as if you hadn't actually had Parkinson's disease." But these comments, though observant of change and improvement, are not statements as to the reversibility of Parkinson's. Rather, they suggest that the doctor was considering a change in his original diagnosis: the patient had been misdiagnosed.

¹ We do have one exception, a case in which – for political reasons – a group of doctors officially stated that a person "might have recovered from Parkinson's disease." The case involved a French magistrate (judge). The reason behind the statement was this: because her Parkinson's symptoms were waning, she was facing a new diagnosis – a diagnosis of psychosis. From her doctors' point of view, the only possible reason that she could have previously exhibited "false" symptoms of Parkinson's disease for several years would be that she'd been having a psychotic episode that whole time. However, *if* her diagnosis was changed to "psychotic episode," she obviously could not remain in her position as magistrate. Psychotics cannot be judges.

(This was in the years prior to the "discovery" of the syndrome now known as psychogenic parkinsonism. Prior to the definition of this "new" syndrome, a person who recovered from PD-like symptoms was considered to have undergone a psychotic episode.)

The PDer asked me for advice. She was working with the top neurologists in Paris at the time. I suggested that she tell the doctors, in all honesty, that we had some patients in Germany who were recovering, and that the German patients would be more than happy if the first European recoveries from Parkinson's occurred in Germany, not France. When the patient presented this information to her team of doctors, her diagnosis was changed to "possible recovery from Parkinson's." She was allowed to continue working.

A PDer could not expect the neurologist to pick up on *recovery* symptoms and to notice that they are the opposite of PD symptoms. If muscles went limp, the doctor only noticed that the PDer was worse off than before. MDs are trained to focus on what is *wrong*. If a PDer became limp and weak, the MD focused on that, and added to his store of knowledge the incorrect idea that, in some people with Parkinson's, the condition causes limpness instead of rigidity. The doctor often said, accurately, that in terms of movement, the PDer was obviously much worse than before.

Trusting only the doctor

Some PDers who became stuck in partial recovery told me that they would only believe that their physical changes pointed towards recovery, and that they would only then (reluctantly) force themselves to do the visualization and the heart- and mind-retraining exercises “*if and only if an MD concurs that there has been some improvement.*” When the MDs invariably failed to do this, these people usually went into a tailspin of despair – and a rapid worsening of symptoms. Often, after these disappointing meetings with the MDs, their symptoms were “worse than they've ever been before” within a matter of days, or even hours.

On the other hand, some PDers who did the hard mind-retraining work even if it took months or years, and who then had a breakthrough to a healthy mindset, were suddenly able to move easily again. Following this breakthrough, they just as suddenly dismissed the whole subject of doctors. Their new attitude has been expressed, in a few cases, as “Who the hell are they [doctors] to tell me that I've got an incurable illness? What an idiot I was to have been so emotionally influenced by them! Why did I unquestioningly believe them?!” These people were able to feel the burgeoning changes within themselves, and had no reason to rely on any outsider's opinion as to whether or not they were doing better.

I was actually a bit surprised at the level of disgust, even anger, that many recovered PDers felt towards their doctors when they realized that doctors were fallible.

In general, it seemed as if, when the PDer's *minds* and *emotions* became healthy, the PDers knew how their bodies felt. Doctors became superfluous. Oppositely, those who were walled off from their abilities to feel and to imagine themselves moving, and who were therefore somewhat numb to the healing sensations going on within their own bodies, needed outside corroboration to confirm any changes or seeming improvements.

NO MORE ADRENALINE

Another event, that sometimes occurred before, during, or after this time, was a perceptible decrease in adrenaline. Pre-recovery behaviors, including physical, mental, and emotional behaviors, often suggested elevated adrenaline levels. When the foot injury healed, these behaviors often decreased or even disappeared in spite of having been dominant for much of the PDer's lifetime. During this phase, some PDers worried that they would never be as “intense” as they used to be. It appeared as if their adrenaline levels had been turned way down, or even off.

If the limp muscle phase coincided with the decrease in adrenaline, fear-based PDers sometimes were even more doubtful that life would ever again be worth living.

When the adrenaline declined, PDers sometimes found themselves accidentally being calm, even daydreaming. This could be terrifying if the recoverer felt that his very nature was

undergoing a change. During this time, “Who am I?” and “Who am I becoming?” were not unusual questions. This was sometimes a period of adolescent-like self-questioning or, in some cases, despair.

One recovering PDer said that, during this phase, her adult daughter showed up at the house, took one look at her, and asked, “What’s the matter?!” The mom replied, “Why should anything be the matter?” The daughter answered, “You’re sitting on the sofa.” The mom, genuinely puzzled, asked, “So?” The daughter said, “It’s just that I’ve never seen you just sitting around before.”

Another observed herself being so calm and content that she asked her husband and teenage son, “Do you still love me? I’m so different. How can you love me now, when I’m so mellow, if you used to love me the way I was before?”

The son answered, “Mom, we love you. Before, we loved you *in spite* of how you were.”

I have used the above line many, many times to console a PDer who found himself “turning into the people I used to look down on: the slow, inefficient people.” I will suggest to him that, “Maybe people didn’t used to love you *because* of the way you were: always so intense and perfect. If they truly loved you, they loved you *in spite* of it, not because of it.” This remark was sometimes well received. Other times, not so well.

PDers who used to feel that they were The One To Rely On during any situation were sometimes concerned by their new imperturbability and *laissez faire*. Some PDers wondered what would happen should an emergency arise. They feared that their “new” personality would most likely respond to an emergency by saying, “Emergency, shemergency; let someone else take care of it.”

Some admitted that nothing was ever as much of an emergency any more as they’d made things out to be, in the past. Small crises that previously would have been met with intensity and adrenaline were now met with, “It doesn’t really matter; it’s not as if anyone’s going to die.”

Many recovered PDers used the above quote. The phrase, “It’s not as if anyone’s going to die,” spoken with such firmness, by so many fully recovered PDers, actually helped us figure out the core emotion that was causing so many PDers to lock themselves into the semi-paralysis of pre-death, automatic-type dissociation.

Decrease in adrenaline in those who *could* relax

Prior to recovery, many PDers seemed to have been constantly relying on elevated levels of adrenaline.¹

When the injury crisis was over (the foot was healed) and they were able to relax, they sometimes felt as if they could never be in *predominantly* sympathetic mode ever again. Mild emergencies of the past, such as running late or forgetting an appointment, were no longer able to trigger even the merest drip of concern, let alone the heady response of an adrenaline surge.

¹ Adrenaline is sometimes described as the neurotransmitter of fight or flight. This is gross oversimplification. Adrenaline is more often employed as the neurotransmitter of wariness or mild thoughts of concern. Adrenaline activates breathing and heartbeat during times of calm, as well as during times of stress. Appendix xxx explains in great detail the differences between adrenaline- dominant and dopamine-dominant physiology and behaviors.

The sympathetic system, having been overused for years, now seemed to laugh off all petty emergencies.

And even in the few cases when genuine emergencies arose, they were able to deal with them calmly and efficiently, from the perspective of the parasympathetic mode.

Decrease in adrenaline in those who could *not* relax

PDer's who remained negative, cynical, or fear-based, dissociated from their hearts and living in their minds instead of using a blend of mind and body awareness, had a very different experience when the adrenaline decreased. The decrease in adrenaline that seemed to occur when the injury healed was a very large problem for people who were not emotionally able to drop their wariness and shift into parasympathetic, dopamine-releasing, mode.

Prior to diagnosis: using adrenaline every day

Prior to diagnosis and recovery, the PDer may have used his steady flow of adrenaline to power his movement. Some PDer's told us that they had intentionally imagined potential emergencies to get themselves going, if they found themselves slowing down.

For most of the PDer's life, he may have pitted his ability to generate adrenaline against the immobility from the injury (and from any mind-induced dissociation symptoms).

Although MDs still claim that PD becomes apparent when dopamine levels are too low, the truth is that the symptoms of Parkinson's disease become apparent when the PDer can no longer summon up enough adrenaline to override his ever-increasing immobility. When our patient's feet healed, their adrenaline (from injury) levels took a significant drop. If patients were still emotionally locked into fear-based dissociation when their adrenaline levels dropped, dissociation became their dominant neurological mode – not parasympathetic. Dissociation causes rigidity and inhibition of the movement neurotransmitters.

This concept confuses a few people, so I will repeat this: the unhealed injury had provoked a steady slow stream of adrenaline throughout the years.

In the example of a badly injured person running from a bear, it is adrenaline that enables the runner to override the immobility that would otherwise be created by the severe injury. Adrenaline can override the healthy inhibition of the dopamine-release system *and* the activation of the go-to-sleep system, both of which are supposed to be triggered by a severe injury, and both of which are supposed to cause immobility as soon as the injured person gets to a safe place.

When a PDer's injury was gone, his adrenaline levels receded. If his adrenaline levels went down but the mental links to dissociation remained or continued to expand – he found himself with a *significantly* reduced ability to initiate movement.

It seemed as if he no longer had enough adrenaline to keep himself fired up and fighting, but he did *not* have the feeling of safety in his heart that initiates the release of dopamine.

PDer's who fit this model behaved as if they had low levels of both adrenaline and dopamine: a condition that occurs during automatic dissociation. Movement-wise, they were far worse off than they had been just a few weeks before – back when they still had the trauma from unhealed injuries to keep their adrenaline flowing – overriding the tendency for dissociation. And in addition to low neurotransmitter levels, they sometimes had limp muscles.

When some PDers got to this decreased-adrenaline stage of recovery, in terms of movement *and* emotion, they very often perceived themselves to be *far* worse off than before. This was the general trend in those PDers who were unable to relax even after the injury healed and their injury-based adrenaline levels dropped.

DURATION OF LIMP MUSCLES

The amount of time necessary to recover strength in limp muscles varied from one person to another and from one muscle group to another. You probably knew I was going to say that.¹

But in general, recovering PDers could be divided into two groups: the people who recovered very quickly and the people that got stuck in partial recovery. Because the people who recovered quickly can be described very quickly, I will insert a short paragraph here about those people. The section on partial recovery will follow, and will take up a bit more time.

Fast recoverers

The people who recovered the most quickly did so without even *thinking* about it. As they recovered feeling in their bodies, they found themselves consciously enjoying the sensations of using body parts that hadn't been felt *or* used much in the past. Since it was enjoyable, they did it. They didn't have to *think* about doing it: they worked these healing muscles in the same way an infant automatically uses and enjoys his muscles. These fast-recovering PDers may have had limp muscles for a few hours or days, or a week or even a few months, at the most, and then suddenly recovered full function. It was not as if the muscles needed to grow strong: it was as if the nerves that connected the imagination to the muscles needed to be hooked back up.

However, I must note that even those who are peace with themselves, and who are enjoying the curious sensations of their unresponsive limbs, may find this stage to be particularly trying.

PARTIAL RECOVERY

After the Little Project had been up and running for five years, we had to admit that some people stayed in the limp-muscle phase for a long time: even years. That's when we decided to name this phase, if it lasted for more than a few weeks, "partial recovery." We could see, in retrospect, that the onset of the muscle weakness was the most common time for people to slip into partial recovery – even if the muscles did not become profoundly limp.

By the time a PDer began to experience muscle limpness, his body's physiology had usually already been experiencing recovery from numbness and other blatant recovery symptoms such as distinct changes in sleep patterns and other recovery symptoms that are described in later chapters. However, when the more overt recovery symptoms eventually ceased, the mood-based symptoms of parkinsonism sometimes increased with a vengeance.

While trying to discern the difference between PDers who recovered quickly and those who became mired in partial recovery, we observed that the partial recovery patients responded to the limp muscle phase with wariness and fear. It even seemed as if, in some cases, the return

¹ One of my many proof-readers was annoyed by the redundancies in these chapters about recovery symptoms. However, one of the proof-readers who was actually on the Parkinson's team said, "I'm so glad you're starting to be redundant about the most important bits. So many of our patients have been *so* resistant to the key ideas that they need to hear them over and over. So many don't even understand principles such as "each recovery is different" until they've heard it repeatedly."

of awareness of long-ignored body parts triggered or amplified the fear that had allowed or caused the body to block out the knowledge of the foot injury in the first place.

Healthy fear can cause a shift towards the sympathetic system, the release of adrenaline, and an enhanced ability to move with speed and power. Helpless fear, or a sense that one is dying and nothing to be done to prevent it, can cause a shift towards dissociation, the inhibition of both adrenaline and dopamine, and rigidity, slowness, and *weakness*.

When PDerS who were starting to recover experienced an increase in fear, they tended to go towards dissociative mode instead of sympathetic mode.

A few PDerS never even noticed much of a limp phase: they went directly from terrifying pain, which started when the injury began to heal, straight into amplified rigidity, slowness, and weakness. In these cases, it seemed as if the *fear of pain*, together with the decrease in adrenaline, caused an abrupt increase in rigidity. It might have been fear induced by the pain of remembering the foot injury, and/or the fear induced by the tingling in the previously numbed body areas, and/or the fear induced by spontaneous movements in recovering body parts and/or the fear of not being able to recover. Whatever caused it, these people very quickly became *very* rigid, slow, and *weak*. They became almost paralyzed with symptoms consistent with psychogenic parkinsonism and also consistent with frailty – a mentally influenced condition.

In other words, when emotionally restricted PDerS started to heal, they experienced a shift in the *cause* of their rigidity. Previously, their slowly developing rigidity had been caused by injury-induced, backwards-flowing Qi.

During the early weeks or months of recovery, they experienced a return of sensation in their feet and fact, improved circulation, tingling in the nerves that were coming back from dormancy, and spontaneous movement in toe and facial muscles – muscles that had previously been unresponsive. Clearly, these people were healing. But many of them also quickly attained a higher level of rigidity than before.

Weakness

Also, although people who got stuck in partial recovery might or might not notice a limp phase, most of them *did* notice the onset of weakness. Weakness is not necessarily a symptom of idiopathic Parkinson's. People with classic Parkinson's become rigid and slow. They often have problems initiating movement. But they tend to be driven and tense, rather than weak. And yet, people who got stuck in partial recovery often complained about weakness. The weakness usually started shortly after the foot injury healed up.

I sometimes wondered if the weakness experienced by people who were stuck in partial recovery was some sort of long-term variation on the limpness of muscles that was experienced by people who recovered quickly. However, my hunch is that the long-term weakness has to do with the negative mindset that locks a person into dissociative mode, and the limpness is more related to a mind-to-body part disconnect. Maybe some researcher with a SPECT scanning machine will study this someday.

Duration of partial recovery

How long did the partial recovery phase last? It's impossible to generalize. Some PDerS who got stuck in partial recovery steadily slogged through the mind-retraining exercises and recovered fairly quickly, in a matter of weeks or months. Some recovered slowly, over the course

of years. Some have not yet fully recovered. Of the latter group, many dropped out of the program. Others have not.

But the great thing is, some PDers who'd been stuck in partial recovery for more than a year did eventually make the stunning break through to feeling safe, relaxation and healthy sensory awareness of the body. When this occurred, any weakness, slowness of movement, or rigidity promptly disappeared. The sudden return of the healthy ability to move via imagining movement and anticipating the internal sensations associated with movement was as abrupt as the flip of a switch.

When this change occurred – after weeks, months, or years – the muscles had conscious strength. They were not limp. It may be that, during the months of partial recovery, the muscles re-established their mind-body relationship during those moments when the partially recovered PDer was sleeping or momentarily forgetting to be afraid. So when the dissociation ended, the body was ready to go.

At any rate, we suspect that the genuine limpness of recovery actually only lasts a few weeks, maybe a month or so, at most. After that length of time, the nerves that connect the mind and the muscle seem to be restored. Any weakness that remains after that span of time is more likely due to mindset than to a lack of mind-to-muscle neural connection.

Partial recovery could take the form of perennially limp muscles, dissociation-induced rigidity, or some combination of the two. Some PDers dropped out of the program when they got to this point.

Some of these partially-recovered PDers experienced many of the recovery symptoms mentioned in the previous and in the following chapters – symptoms that are the opposite of PD symptoms. Some of them even experienced periods of time – hours, days, or even weeks or months – during which they were moving normally. But at some point, when they relapsed into fear, disbelief of recovery, or revulsion at their new body awareness, they quickly, in some cases instantly, became horribly rigid and slow.

Some spent several years coming to terms with memories of hideous events in their past that they had blotted out. During this time, they might be uncharacteristically anxious, if not paranoid. These negative emotions, in turn, seemed to trigger the rigidity, slowness or movement, and tremor that are associated with Parkinson's disease.

In these cases, the PD-like symptoms were clearly psychogenic. We finally discovered the actual combination of mental and physical mechanisms that these PDers were using to invoke this condition. Based on our work with patients with psychogenic parkinsonism, a condition that looks exactly like Parkinson's but which is mood- or emotion-based rather than physically induced, we realized that psychogenic parkinsonism was exactly the same as what we were calling partial recovery from idiopathic Parkinson's disease.

Both psychogenic parkinsonism and partial recovery go away as soon as a person resumes the ability to experience the vibratory feelings in the chest that expand or contract in response to sensory perceptions. People with either psychogenic parkinsonism or PDers who become stuck in partial recovery will steadily worsen over time if the mind increasingly shuts out signals from the heart.

Most of our PD patients hated being told that their symptoms were the same as those of psychogenic parkinsonism. The word "psychogenic" suggests to the layman that the person is merely imagining his symptoms, which is not the case in partially recovered PDers. The

symptoms are very real, even though they are being triggered by a mental/emotional attitude. PDer's preferred the terms "partial recovery," dissociation, or "mental/emotional blockage." Honoring that preference, this book primarily uses the latter terms.¹

How long can a person stay in partial recovery? As far as we were able to tell, a person could stay in partial recovery indefinitely. There was no *physical* treatment that benefited these people. Those who eventually recovered fully after having been stuck for a while in partial recovery had not necessarily undergone any further *physical* change that opened the door to full recovery.

The people who overcame partial recovery all did it the same way: they *decided* to stop being wary. As soon as they refused to be wary, their bodies behaved as if they were healthy. If they were still processing pain from long-unhealed injuries or other physiological recovery symptoms, they continued processing them. But as for the rigidity, slowness, and tremor, those symptoms were over with.

Sabotaging recovery

As noted earlier, many patients respond to their recovery symptoms with "Yahoo! I don't know what the heck is going on, but it sure as hell isn't Parkinson's!" But more patients told me that recovery symptoms were only a tease, or that they couldn't really be signs of recovery. They were certain that, even if recovery were possible for some people, *they* could not be one of the "lucky ones."

An example of sabotaging one's own recovery

One deeply negative recovering PDer left this message on my answer machine: "I went to a church dance last weekend and for the first time in my life I was moving gracefully. I wasn't rigid: I felt like I was floating, it was glorious. I didn't care who saw me or what anyone thought. It was the best time I've ever had. People were saying that they've never seen me look so good. So tell me the truth: was that the Last Hurrah? From here on out, am I only going to get worse? I can't imagine that I will ever have such a great evening again. Was that the last time I'll ever move easily? Should I admit that I can't recover?"

I was stunned by his twisted thought processes. Movement-wise, he had recently had the best evening of his life. His conclusion, based on the fact that he didn't really think he could recover, was that this was a sort of "Last Hurrah," a spontaneous gift from God, and that, having used up these few hours of grace, he would never move easily again.

This man had been recovering wonderfully. In particular, his facial expression, the function of his hands, his loud voice and his ability to stand up straight had resumed. These were changes that he couldn't hide, even from himself. But he was also getting better at inducing a stunning level of immobility by the simple method of reminding himself that he couldn't really move. As soon as he remembered that he couldn't actually recover, he could hardly move.

He assured me that he believed that some people might be able to recover. But he was equally convinced that *he* could not. He knew that this was so because, during those times when he was *most* certain that he would never recover, his movement was the worst. The fact that he

¹ Western medicine has, for centuries, dismissed psychologically induced illness as "not real" or "less real." Asian medicine recognizes that nearly all illnesses, even susceptibility to germs and "measurable" pathologies such as myopia, have a mental component.

could induce immobility in himself just by dwelling on the impossibility of recovery *further* convinced him that he could not be recovering in *any* way. Despite his tremendous number of improvements, he could not shake the idea that he was doomed.

When he admitted that he had not once tried to do *any* of the homework (an assortment of relaxation or attitude-retraining techniques), he said to me, in all honesty, “Can’t you do that part for me?”

He had been off work, getting disability payments, but he admitted that he was now physically able to return to his teaching job. I told him that returning to work would be the best thing for him, but he replied he didn’t really want to go back to work. He also threw fallacious logic at me to show that he *shouldn’t* go back to work. He pointed out that, when he was in a negative mindset, he had increased problems initiating movement; since teaching *might* provoke some negative thoughts, even thinking about teaching could permanently worsen his condition.

I told him that his *mental* attitude, not his environment, determined whether or not challenges were interpreted as negative or positive, and that there was nothing inherently fear-inducing about going to work. He disagreed.

We’ve seen that PDers who keep working do much better than those who take time off to “focus” on recovering. The ones who take time off usually end up dwelling on their fears and feeling bad because they aren’t “doing” anything.

Throughout our nine months of working together, he had admitted that his extremities were healthier, had better color, were warmer, and had more feeling. He admitted that he had increased facial expression and better posture. He admitted that he had regained the use of his hand and voice. He admitted that, during those times when “I forgot to have My Parkinson’s,” he could move easily. And he consistently denied that these changes signified “any change in My Parkinson’s,” as he called it. There seemed to be a possessive quality in the way he always referred to his condition as “My Parkinson’s.”

Because he was increasingly able to immobilize himself with worry, when his negativity-trained mind took a twist in that direction, he was certain that “My Parkinson’s” was getting worse. In a spiraling fashion, the one thing that was most able to provoke a spate of immobility was thinking about how *he could never recover from Parkinson’s*. This circular bit of thinking, which made him fearful, which then made his movement ever more difficult, confirmed his worst suspicions and assured him that he could not recover, despite all his lasting physical improvements.

When I asked him why he indulged in such thinking, he said that his brain did it, not him. When I asked him who was in charge of his brain, he laughed and said that he wasn’t responsible for his own thoughts and didn’t want to do the work of changing them. He certainly didn’t want to do any work oriented towards positive thinking. He felt that positive thinking, in the face of an incurable illness, would not be “true.”

Once again, he asked me if *I* couldn’t simply get rid of his negative thoughts for him by using acupuncture or herbs, because his negative thoughts seemed to have a mind of their own. Although he laughed when he said this, he was dead serious. I had given him *many* techniques that can get a person out of negative thought loops, but he had never even tried one of them. He had no intention of doing the hard work of becoming the master of his own thoughts. He knew how to mire himself in negativity. He had unknowingly trained himself in it, and he was *very* good at it.

He only saw me one more time after his “Last Hurrah” phone message” During this last session, he denied that he had moved well at the church dance! I asked him why people had told him at the time that he was moving so well. He replied that he didn’t know.

He was also fascinated with the powerful effect that placebos have on PDer. I got the idea that he wanted a doctor to give him some placebo that would convince him that he was OK so that his symptoms would go away. I asked him why he didn’t just work on generating a positive attitude in himself. He laughed and said that he wasn’t interested in doing that much work.

Are these people crazy?

Again, a non-PD reader might think that I am making this up or that, in cases such as the above, I am dealing with a crazy person instead of a typical PDer. However, this man’s thought processes were fairly typical for a PDer stuck in partial recovery. Also, people with Parkinson’s are usually thought of as highly intelligent, responsible, and keenly self-monitoring: not crazy.

PDer who develop such clever ways to prove their expectation that they will get worse are not actually crazy – they are merely applying their long-practiced habits of harm avoidance. And these habits have, in many cases, grown entrenched over the decades. Many PDer have said to me, “I’d rather assume that I won’t recover. That way, I’ll be pleasantly surprised if I do. If I assume that I will recover but then I don’t, that would be painful.”

PDer often think everything through in order to avoid pain. This kind of thinking often means anticipating a worst-case scenario. But many PDer consider themselves clever, not negative.

Many PDer have cultivated for decades an elevated ability to anticipate worse-case scenarios. This attitude, in turn, keeps a person from “risking” the sensations that can occur if a person drops his fears and truly relaxes. Then again, this attitude also inhibits a person’s ability to relax – or release dopamine. PDer need to make a note of this: anticipation of risk or worst-case scenarios in a person whose uses dissociation as his response of choice to anticipated fear or pain will inhibit his own ability to release dopamine. A person who dissociates when he anticipates *not* being able to release dopamine will find that his expectation is instantly fulfilled.

The powerful irony here is that what some PDer perceive to be their greatest strengths, their ability to be analytical and to anticipate worst-case scenarios are, in fact, their greatest weakness and their physical downfall. This ability is a downfall because it has become habitually connected with selective dissociation from the heart; a habit that causes symptoms of automatic dissociation.

It may seem that this section on “sabotaging recovery” is wandering far from the point of this chapter: recovering from rigidity. But I am writing this chapter both to describe what we saw *and* to help future PDer who use this book as a guide to be able to recognize symptoms of recovery. One of the things that can make it difficult for a PDer to recognize symptoms of recovery is that a PDer’s mindset colors the events: he cannot be a neutral observer. A negative attitude may prevent recovery events from being recognized for what they are. Therefore, it is just as important to warn of the negative attitudes that might accompany sensory and movement changes as it is to write about the changes themselves.

PREDICTING WHO WILL RECOVER QUICKLY

There is no way of knowing who is going to stall during recovery. However, in our limited experience with just over two hundred subjects (a small number, by research standards), the five professional musicians in our program all breezed quickly through the phase of limp muscles. So did many others, including dancers and those who were able to enjoy the enhanced sensory experiences that occur during true relaxation.

However, other PDers were surprised to learn that, in healthy people, certain sensations or events might occur during relaxation: the chest might feel a sense of expansion; a feeling of movement or expansion might occur in the chest in response to music or visuals; the mind usually calms down and pays more attention to the perceptions of sensory events; the mind ceases its harried, internal monologue.

Still other PDers who *used* to know that relaxation could induce these shifts, even if they were no longer able to consciously induce them, were not as prone to slide into partial recovery.

Finally, those PDers who had no idea what was meant by the words in the above description of relaxation, who had no point of reference for the idea of palpable internal sensations such as “a feeling of expansion in the chest when experiencing something of great beauty or grace,” or who had no idea that these increased sensations were the hallmarks of true relaxation, and who instead had thought that “relaxation” referred to a welcome *cessation* of sensation – numbness – tended to slide into partial recovery and stay there.



CHAPTER TWENTY

RECOVERY SYMPTOMS: DYSKINESIA

Dyskinesia means incorrect movement. Dyskinetic movements may be somewhat constant, like the tension caused by a long-lasting spasm, or they may be intermittent, like a twitch or tic. During recovery, previously disconnected muscles regained a conscious connection to the brain. When this happened, the muscles sometimes behaved dyskinetically for a short period, until correct brain-to-muscle function was restored.

Spontaneous movement

The dyskinetic moments during recovery were delightful, inasmuch as they seemed like the very opposite of the forced, difficult movements of Parkinson's. They seemed to occur when previously rigid or numb muscle began to receive nerve signals once again. When this occurred, the muscle coordination was often poor, even spastic, at first.

When these muscles began to connect to the brain, so that the PDer could feel their existence, they often moved imperfectly, even immaturely: these muscle movements were sometimes like those of a baby.

A baby grimaces asymmetrically while exploring his facial muscles; he repeatedly, helplessly, bangs a spoon on the highchair tray while learning to use his arm. The recovering PDers found themselves experiencing asymmetrical facial expressions, strangely clumsy muscles, or even muscles that initiated repetitive movements much like those of a baby steadily banging a spoon on a tabletop.

We named this spontaneous, usually gentle, non-controlled movement "recovery dyskinesia." In appearance, it sometimes resembled the dyskinesias (non-controlled, and sometimes repetitive movements) that occur in PDers who have taken too large a dose of their antiparkinson's medication.

Recovery dyskinesia sometimes occurred in the large muscles of the chest and legs and sometimes occurred in the small muscles that give finger finesse or facial expression. Some events were one-time spasms followed by perfect conscious control. Sometimes they were gentle flexions and extensions of a single muscle, repeated a few, or a hundred times or so, every day for a few days. And they were everything in between.

Recovery dyskinesia is *not* to be confused with the uncontrolled movement of Parkinson's *tremor*. Fortunately, once you understand the causes and characteristics of each, it is not very difficult to distinguish between them. Parkinson's tremor, whether it is the resting tremor of the fingers, shaking of the arms, legs, or chin, or the "amplified tremor" that shakes the whole body, is always at its worst when the PDer is stressed or thinking of something worrisome, and it ebbs when he relaxes or doses off. Recovery dyskinesia, on the other hand, only occurred when a person was relaxed and feeling safe. In fact, it occurred most often when a person was watching TV in the evening or lying down to sleep.

Another key distinction is that Parkinson's tremor is generally somewhat vexing to the person experiencing it, while recovery dyskinesia was generally described as an enjoyable experience.

Finally, the movements themselves are different: Parkinson's tremor is usually a predictable, rhythmic vibrating or shaking, whereas recovery dyskinesia, as you will find in the coming sections, could be almost *anything*.

In some PDers, recovery dyskinesia was only a fleeting event that happened once or a few times or not at all in any given group of muscles. Some recovered PDers had no recovery dyskinesia.

Examples of recovery dyskinesia

In this section, I will describe only a few examples of recovery dyskinesia. I cannot possibly describe *all* the instances and styles of recovery dyskinesia that we saw and heard about.

Toe wiggling

One of the first places that recovery dyskinesia appeared was in the toes, shortly after the return of sensation. After the circulation in the toes improved and the tingling was over, a few PDers went through a very short phase in which their toes spontaneously wanted to move and stretch. It was as if the toes moved or "wanted" to move without conscious instruction: moving "on their own."

The PDers said that they *could* inhibit the toe movement if they wanted to. But they found it more pleasant *not* to get involved, for a change, and to allow the toes to simply express themselves. Even if the toes got into a mild cramp, they usually wiggled themselves free pretty quickly.

Movement in facial muscles

When PDers' long-dormant facial muscles began to function spontaneously, the resultant facial expressions were much more obvious than the spontaneous toe movements. The fleeting facial contortions often resembled those of an infant who cannot yet control his face.

Anyone who has spent time with an infant knows that babies can make some pretty funny facial expressions. Their facial muscles don't work in a coordinated manner for several weeks. The same immature types of irregular, spontaneous, or even spastic movements of the facial muscles occurred when the PDers' facial nerves reawakened from their dormancy.

While some PDers were scared that they would spontaneously make a goofy face while out in public, this rarely occurred. Again, most recovery symptoms took place at home, when the patient was relaxing or just before falling asleep.

Although some PDers worried about it, none of them experienced facial spasms while giving a presentation at work. It seemed that the body only took liberties with exploring new muscle function when the PDer was in parasympathetic mode: whenever or wherever he deemed himself "safe."

- Recovering facial muscles: an example of "safe"

One recovering PDer, after experiencing all manner of fleeting, small facial tics and spasms over a period of months, found herself having a major "face recovery" moment while lunching at a classy restaurant with her daughter. They were seated on the rooftop patio, the warm sun was shining down, and they were both laughing. She felt at peace with the world. Suddenly, the entire right side of her face went into a powerful spasm. Her right eye was squeezed shut, the right cheek muscles were bulging outward, and the right side of her mouth

was pulled up towards her eye. On the right side of her face, *all* the muscles that should be used during a heartfelt smile kicked in at once – at full strength. These muscles were still a bit clumsy, and hadn't worked together as a unit in over ten years. As the muscles locked into a powerful spasm for over a minute, screwing up and immobilizing the right side of her face, the recovering PDer finished her sentence by speaking out of the left side of her mouth. The daughter replied, and they continued chatting. After the facial spasm had been in place for what seemed like a full minute, the daughter said, "Mom, I'm not sure that that 'look' really works for you," and they both exploded into laughter.

After a few more moments, the spasm melted away. A new sensation of warmth and power pervaded the muscles in her cheeks and the skin of her face. From then on, her face was far more expressive and she was better able to feel the skin of her own face.

I chose the above anecdote as an example because many PDers have been terrified of having an obvious recovery symptom at a "wrong" time or place, such as when out in public, or in front of some judgmental person. The point I am making here is that, if the PDer felt safe, it didn't matter to him if a facial spasm appeared in public. But if a PDer tended to be self-conscious, public places would be "not safe." Therefore, he would be unlikely to experience a recovery event in which the body was playfully exploring the use of some long-dormant nerve group. In the above scenario, the recovering PDer *was* out in public *but* she was not self-conscious at all. She was having fun. She continued to enjoy herself while she temporarily looked ridiculous. She was able to laugh at her own goofiness. Very possibly, the delightful, calm circumstances helped her body choose such a moment for practicing some important muscle recovery work.

PDers only experienced recovery symptoms when they felt safe. Location, per se, did not make a place safe or not. What made a place "safe" was the attitude of the PDer.

A sudden arm stretch

One recovering PDer was practicing piano when suddenly her right arm straightened itself sideways, and extended to the keys at the high end of the keyboard. After a moment, the arm returned to its previous position. This happened several times that evening, and then never happened again. It was *not* a muscle cramp. The movement had been generated by a contraction of the muscles around the ball of the upper arm, where the upper arm sits in the shoulder socket. These muscles tend to be inaccessible to PDers. The movement had been powerful and graceful – and completely unexpected.

A neck pivot

Another recovering PDer was standing up singing in the choir when her neck muscles painlessly but powerfully pulled her head to the left. She found herself staring at the person next to her. The person next to her stared back.

"Cramp in my neck," lied the recovering PDer. After a split second, the neck muscle relaxed and she found herself facing forward again. It had not actually been a cramp. Cramps are overly tight and painful. This neck movement wasn't painful. It had been an involuntary, gentle contraction in a neck muscle that she hadn't used in years. Moments later, it happened again.

"More of a nervous twitch, really," she whispered.

After half a dozen episodes over the course of an hour, it never happened any more. Prior to this event, she had not been able to turn her head easily to the side. After this event, she could move her head easily.

Singing in the choir was one of her all-time favorite activities. She was not embarrassed by the “twitch.” She thought it was funny. *She was relaxed.*

The waving lady on a pancake house

Another recovering PDer described recovery dyskinesia in her arm. “Last night while I was watching TV, my right arm lifted up and started waving back and forth, back and forth. It wasn’t fast. It was like that plywood woman on the roof of the pancake house who mechanically waves back and forth. It kept it up for about ten minutes. Never went slower or faster. It didn’t hurt. I probably could have stopped it, but I didn’t want to. It was bizarre. My adult daughter was kinda worried about it, but I thought it was kinda funny. It felt nice. It wasn’t *anything* like tremor.”

Marching in bed

Sometimes, when sitting on the sofa or lying in bed, a recovering PDer’s leg(or legs) started rhythmically “marching.” Sometimes, the arms kicked in, moving in time with the legs. This was not “restless leg syndrome.” This was recovery dyskinesia.

Fixing the foot

One PDer felt an urge to bang the top of his foot against the bottom of the coffee table. Every evening, relaxing after dinner, his leg “wanted” to bang the top of his foot. So he let it. He would let his leg gently rise up a few inches, causing the top of the foot to get a gentle whack. The movement was steady and rhythmic. Each evening, the foot was whacked at least a hundred times or so. The whacking only stopped when his leg didn’t “want” to do it anymore.

He said it felt really good, as if his leg knew that something in his foot wasn’t quite right, and needed to be gently knocked back into place. The foot had received many Tui Na treatments. The foot felt completely healed, in terms of a return to suppleness and all the other things we look for when treating an old injury. And the PDer had no *conscious* understanding of something in particular that needed to be done to the foot. His leg just “wanted” to gently whack the top of the foot, that was all.

Shortly after the PDer noticed that his toes seemed happier and more flexible, he also realized that his leg was no longer wanting to bang his foot on the bottom of the coffee table in the evening.

Technically, this might not be considered a form of recovery dyskinesia. But the movement felt almost spontaneous, it was repetitive and rhythmic, and so I’m including it in this section.

King Kong

One PDer, relaxing at home in the evening, beheld her alternating fists beating on her chest as if she were doing the “proud gorilla” move in slow motion. Alternating, left, right, left right, her fists pounded her chest for nearly ten minutes. She was a very delicate, petite woman: the next morning she had bruises on her chest. She only had this experience this once, and afterwards, her arms were more relaxed.

Hand positions

In some recovering PDers, the muscles in the hand tightened for a while – never for longer than twenty minutes at a time. This sometimes caused various weird hand positions: dancer-like, stylistic hand positions. One person noted that these hand postures happened most often on her daily walk. Others noticed them while sitting around watching TV.

The voice

Some PDers' voices suddenly reappeared after months or years of being reduced to a whisper. The return of voice was not always a straight line. The muscles that opened up in the larynx sometimes opened very wide, without warning, and an embarrassingly big booming voice issued forth for several minutes – or a day or two. And then, sometimes a few days later, the voice temporarily disappeared altogether. Eventually, the big voice became consistent in those PDers who fully recovered.

One PDer called his sister when his voice returned after nearly ten years of inaudible speech. His sister accused him of being a prankster. “You’re not my brother; my brother only speaks in a whisper.”

Chest muscle spasms

Several patients grew mildly concerned while experiencing recovery dyskinesia in the chest. In Parkinson's disease, the muscles that run down the mammary line of the torso become rigid. During recovery, these muscles sometimes went through recovery dyskinesia: either they tensed up for about twenty minutes and then relaxed, or they tightened and relaxed over and over again, somewhat quickly. Either way, the event usually lasted no more than twenty minutes.

During the tightening up phase, recovering PDers sometimes felt as though they could not take a deep breath. A few PDers wondered if they were having a heart attack. They decided to go to the hospital. By the time they arrived at the hospital, the tightness had ceased. One PDer stuck around at the emergency room for a full heart work up, to reassure himself that nothing untoward had occurred.

An aside: I always told patients to see a doctor immediately if they felt that they were having a heart attack. I did not want them to ignore symptoms that gave cause for concern. If it turned that it was only recovery dyskinesia, and not a heart attack, no harm would have been done. None of my unmedicated patients had heart attacks during their recovery from Parkinson's disease.

During the *relaxation* phase of chest-muscle recovery dyskinesia, the opposite occurred. Any PDer who experienced this felt his chest was so wide open that he hardly needed to breathe. One PDer said, “I was enjoying my shower when I suddenly realized that I hadn't inhaled in a really long time. I was a little concerned, but I just didn't feel any need to inhale. Finally, I did inhale and exhale, and again, a long time passed before I felt the need to take another breath.”

Recovery dyskinesia of the head

Recovery dyskinesia often occurred the neck, the face, and the inside of the head.

Recovery dyskinesia in the tongue

Many PDers experienced a bout of clumsy speech: the tongue felt as if it was moving on its own or it didn't move the way it used to. Some PDers thought, for a short while, that they

might need to relearn how to use the tongue. However, the speaking skills usually came back very quickly. In some cases, the tongue seemed to be positioned differently. This may have been caused by a change in sensory awareness of the tongue or a change in the position of the palate.

Many PDer's bit their tongues and/or the inside of their cheeks during this phase.

One PDer had a curious tongue situation develop during recovery: when concentrating deeply on anything, she would stick her tongue out on the right side of her mouth, ever so slightly. Just the tip of the tongue was visible. She had been doing this for several weeks when she remembered an old photograph of herself playing the violin when she was age twenty. In that photo, her eyes were closed and she wore a look of deep concentration – and her tongue was sticking out the right side of her mouth. She knew that she had not put her tongue out to the side for decades but now she was evidently reverting back to her youth.

After a few months, after her palate lifted and her teeth spread apart, she no longer stuck her tongue out to the side. She was certain that her age twenty tongue-to-the-side had not occurred when she was healthy, prior to age seventeen. She suspected that she had started doing it shortly after the weird buzzing experience on the side of her head at age seventeen, after which she always felt a little more tense inside.

In other words, the age twenty tongue asymmetry and protrusion had been a very early symptom of left-right imbalance and internal tension – a very early symptom of pre-Parkinson's disease – and not a healthy idiosyncrasy. When her tongue started staying inside her mouth even when she was concentrating, she said it felt very good. She could remember her tongue feeling comfortable in her own mouth when she'd been a youngster, prior to her brain shift at age seventeen. ("Brain shift" will be explained in an upcoming chapter xxx.)

Recovery dyskinesia in the palate

A person's upper palate is held in position by muscles. These muscles sometimes changed their degree of tension during recovery. If the muscles above the upper palate gently tightened, lifting the lateral (outer) sides of the upper palate, a gap appeared between the two front teeth, as if the teeth had suddenly moved apart. This sudden, very visible movement of the teeth was painless. The teeth had not actually moved in their sockets: the two sides of the upper palate had lifted up, and the teeth came along for the ride. The roof of the mouth sometimes felt as if it had assumed a new position. Again, this movement in the upper mouth was not painful. Just the opposite: it felt pleasant, if it felt like anything at all. It seemed to be caused by a return of vigor in previously weak cranial muscles.

Sometimes, the lift in upper palate position brought about a welcome reduction in snoring or sleep apnea.

Many PDer's, *prior* to recovering, had experienced a sensation as if the palate was falling down into the mouth now and then, partially blocking the airways and the sinuses. The resumption of healthy muscle tone in the muscles of the palate often cured or improved this problem.

Recovery dyskinesia in the eyes

During recovery, changes sometimes occurred in visual acuity. Many PDer's noticed a sudden, extreme improvement in vision that lasted for a few minutes or a few days, which was followed by a return to normal vision or a temporary worsening of vision.

For example, one patient said, “I swear, yesterday, I could see a bird sitting in a tree half a mile away!” A few days later, the same person was hardly able to focus her eyes. Over a period of a month, this occurred several times. After that, her vision settled back down to exactly what it had been. Based on everything else we’d seen, we guessed that the muscles of the eyes had been going through tightening and relaxing exercises. The muscle tensions had caused the temporary changes in vision.

After this occurred several times in several recovering patients, I started telling patients not to rush off to the ophthalmologist or optometrist for an eye exam or a new pair of glasses if the vision suddenly altered. In our limited experience, once the recovery dyskinesia in the eye-focusing muscles was over, the vision always settled back down to something very close to what it had been prior to recovery.

Not uncontrollable

PDers nearly always wanted to explain to me that they probably *could* have stopped the movements in the arms or legs or hands if they’d wanted to, but they didn’t *want* to. It’s as if some muscle wanted to work out or stay in a certain position for a while, and the PDer felt good when he allowed it to do so. He enjoyed feeling the sensation of the moving limb or vigorous muscle contraction *and* he enjoyed the general good feeling of expansion in the chest that came from paying attention to how the sensation felt.

These repetitive types of recovery dyskinesia usually lasted somewhere between a few moments and twenty minutes. Even the most relentless recovery dyskinesia usually petered out after about twenty minutes.

In most cases, recovery dyskinesia was amusing. If it ceased to be amusing, it soon stopped. It occurred when a recovering PDer was relaxed or enjoying himself. If he stopped being relaxed or became stressed, the movements soon ceased.

Speeding up

Some PDers had the strange experience of the body moving faster than they could control. While doing an ordinary, mundane and repetitive chore, such as chopping vegetables, it felt as if the *correct* arm muscles for the chore suddenly kicked in. The muscles moved very quickly, effortlessly, in a rapid manner. The *effect* was that the knife seemed to be suddenly chopping the vegetables much faster than before. The *feeling* was effortless, as if the arm was “moving by itself.”

This “speeded up” movement happened while PDers were doing all sorts of activities, ranging from practicing piano scales to chopping celery. The commonality was that it only happened while a person was relaxed or enjoying himself.

Then again, not everyone experienced any events in which limbs seemed to move unnaturally fast.

Variations in recovery dyskinesia

I’m about to be redundant: no one experienced all the types of recovery dyskinesia. Some experienced none. Some people had a few very powerful experiences, some had only a few, mild moments. Some slept through their few moments of recovery dyskinesia and were told about it by an amused spouse. Every recovery was different.

Enjoying the body

Recovery dyskinesia is wonderful. Several ex-PDerers have said that they felt as if they were coming back to life when the body starting relearning, on its own initiative, how to feel and use muscles that had been rigid and somewhat numb. The feeling that one is starting afresh, growing a whole new body, is so very delightful that a few people have even said that they feel as if they have lived two lives in one lifetime: they had their first life, and they were dying from it; then they were given a second chance and they got to start again from the beginning – learning what it’s like to *enjoy* having a body, and learning how to *enjoy* using it.

Wanting to have a body versus enjoying a body

Some PDerers have protested my phrase “learn to enjoy having a body.” They have declared that they *do* enjoy having a body, or always used to. Now they just want to get their old bodies back.

I recall one PDer who was adamant that he loved his body. After we’d been working together for about six months, he told me the following anecdote.

The previous weekend, he’d spent an hour floating in his daughter’s swimming pool, lying on one of those floating rafts with arm rests and a holder for a beverage. His mind was blank and the sun was shining down. He felt a wave of peace flood over him, and he realized that he was feeling good. He realized that he could feel the sensation of the sun on his skin, and the sensation of floating. It was a rare moment. He marveled at the sensations that he was experiencing.

Then, his mind kicked in. He immediately wondered to himself if this was the type of “enjoyment from the sensations of the body” that I was always badgering him about. His next thought was, “Heck, I can’t be expected to *feel* this much *all* the time. That’s ridiculous.” As soon as he thought this, he realized that he was no longer able to be aware of the same sensations from the sun and the floating.

The moment of pleasure from body awareness had snuck in to his consciousness as he’d felt it with his body, but he’d quickly brushed it away with his negative thoughts. As soon as he thought to himself, “Heck...,” and re-immersed in negative thinking, he realized that he was unable to feel his body in the same way. He was unable to recapture the sensation of sun, the feeling of floating, and the good feeling inside his own body. He certainly was no longer *enjoying* it in the manner that he’d done a few moments earlier.

That’s when he began to understand why I frequently told him that he must learn to enjoy his body. He had incorrectly thought that his desire for a *functional* body was the same thing that I called “*enjoy* the body.” These were two different concepts, but he couldn’t understand that until he actually enjoyed how he was feeling, for a few seconds, floating on a raft in the swimming pool. That’s when he realized that a desire for a functional body was, in fact, very different from enjoying the sensations of one’s own body for more than a few moments at a time.

But even when he understood what I was talking about, he refused to accept that he could allow himself to pay attention to his body’s sensations just by relaxing, taking the time to notice how he felt, and most of all, shutting out any interfering negative thoughts. He told me that such a course of action would be impossible. When I asked what was impossible, he replied, “I don’t know what to do! Tell me and I’ll do it!” I said that he had to practice relaxing and noticing how he felt, the way he’d done in the swimming pool. Then he yelled at me, “So what should I do? Tell me what to do, and I’ll do it!” I said that he should practice enjoying the sensations of his

body. He threw his hands as far in the air as he was able, and said again, “Tell me what to do so I can do it!”

I have had many patients who have insisted that they always enjoyed their body, who realized, during recovery, that they actually hadn't. They may have enjoyed *using* the body like a tool, they may have enjoyed having the body obey their instructions when they worked it hard during sports, but they hadn't actually noticed or enjoyed the sensory experiences produced by the body, or hadn't in a long time.

For some PDers, the spontaneous, gentle movements of recovery dyskinesia and the heart-felt relaxation that occurred when the injury healed were like beginner's lessons in how to *feel the body* and *enjoy* having a body.¹

An infantile demonstration of repetitive, dyskinetic movement

Once, in the early days of this project, I was on the road, breakfasting in a pancake house. I was musing about the unexpected symptom of recovery dyskinesia that had recently occurred in a few of my patients. At the table next to me, a baby was sitting in a high chair. I watched as he was handed a spoon. He had little control over his movements. As he focused on keeping the haft of the spoon clutched in his fist, his bicep began to contract and relax.

The result was that his arm, and the spoon, started moving up and down, up and down. The rhythmic movement continued for several minutes. The proud parents said that he was banging the spoon on the tray. But I could see that he was not banging the spoon intentionally: he clearly did not have the coordination necessary to bang the spoon so rhythmically on his tray. What was happening was this: his non-controlled muscles were firing off on their own. The muscles in his arm were flexing and extending, flexing and extending. The infant was not doing it consciously. He looked happy and surprised at the movements occurring in his arm.

As he continued helplessly “banging the spoon on the tray,” the proud parents beamed as his movements became more vigorous. His other arm started moving up and down in time with the first. He was clearly not in control of these movements, and he was obviously enjoying himself. Within moments, his legs started moving in time with his arms. His torso started flexing and extending at the waist. All the muscles along the baby's Stomach channel and Large

¹ More than a few PDers have been certain that sensory awareness of the body and/or enjoyment of the body was self-indulgent, or even “not spiritual.” They needed to learn that the sensations generated with a healthy body are, like flowers and beautiful music, something that can and should be enjoyed. The spiritual injunctions against excessive body attachments have to do with the problems that arise from wrong identification: when a person imagines himself to *be* the body instead of being the soul. Also, *excessive* fascination with the inherently restless body, which occurs when the mind is undisciplined, can be a distraction when trying to stay focused on inner stillness or on uplifting activities.

However, the sensations that arise from having a body, which include vision, hearing, taste, smell, and feeling, are not inherently bad. These sensations can be rightfully enjoyed, because they are just awareness of manifold expressions of God's vibratory presence, a presence that underlies and permeates the universe and all objects, even bodies. So long as a person does not *identify* himself as his ever-changing body or become overly attached to it, he risks no sin in enjoying the sensory experience of being able to feel his own arms and legs. Then again, many PDers cannot even fathom what is meant by the phrase, “Feel his own arms and legs.” A lengthy explanation of what is meant by the word “feel,” and instructions on learning how to feel the existence of the body, are presented in later chapters.

Intestine channel were flexing and extending, flexing and extending. After several minutes, he stopped moving. His parents kissed him, fussed over him.¹

He had not been making these moves consciously. His brain was just starting to form awareness of muscle control. The muscles were starting to learn how to respond to signals from the brain. It would be many months before the child would be able to exhibit elegant motor control over his fingers and arm muscles. But the process had to start somewhere.

His spontaneous, repetitive movements, set in motion, perhaps, by the curious sensation of the spoon in his hand or maybe by his desire to imitate the hand-to-mouth movements he had seen performed by his parents, looked exactly like the several instances of arm or leg recovery dyskinesia that I had witnessed.

This vignette makes two points: the repetitive, spontaneous movements that occur in specific muscle groups during recovery from Parkinson's are not pathological: they are perfectly natural; they occur as the brain is becoming familiarized with specific motor functions. Second, with the right attitude, this movement can be fun.

FEAR OF RECOVERY DYSKINESIA

Although I always made the point that recovery dyskinesia felt good, many potential patients were afraid of it. More than a potential patients were far more worried about momentarily "looking like a fool" than they were about having Parkinson's disease.

Some potential patients, having read through the earlier edition of "Recovery Symptoms" concluded that they didn't want to risk experiencing a recovery symptom while out in public. They told me, in all seriousness, that they were afraid that, in a moment of weakness, recovery symptoms might occur that would make them look "stupid," "wrong," or "sick." Several PDer have admitted that they would rather believe that I am wrong – that Parkinson's *is* incurable – than risk recovery incidents in which they might lose face. After all, Parkinson's is a respectable illness.

The non-PDer reading this might think that I am joking. Surely, no intelligent adult would rather be rigid and shaking than have a few moments of perfectly understandable loss-of-control. But be assured, non-PDer, that many PDers are reading these same words and thinking,

¹ The infant was learning to control the muscles of the Stomach and Large Intestine channel: the two channels that are not functional until birth. The muscles regulated by these channels are the last muscles to come "on line" in the developing child. These channels do not even start to be activated until the baby starts to breathe. This makes sense when you consider that these two channels drive the peristalsis of the gut. An infant in utero should have no gut movement whatsoever: any activity in the Large Intestine might eject sticky meconium (fetal fecal matter) into the amniotic fluid. If meconium gets into the fluid, and thence into the lungs of the unborn baby, it can be fatal. The baby has no need for activity in the gut: all his nutrition comes in through the umbilical cord and goes directly into the baby's bloodstream.

All the other channels, the ten primary channels and the eight extra channels, are functional in utero. Only the Stomach and Large Intestine channels are dormant until the first breath. All of the functions that are nearly impossible for a newborn (thumb and index finger control, eye control, arm control, standing, walking, holding the head up, bowel control, and so on, are regulated by these two channels. In contrast, the muscles that move the lungs, the back muscles, even the muscles under the skin that regulate the pores, and so on, are all operational at birth.

It was no coincidence that recovering PDers with long-dormant Stomach and Large Intestine channels found themselves performing movements that brought to mind the spontaneous muscle activities of infancy. It is also no coincidence that a severe injury almost anywhere in the body will trigger a chain of electrical events that cause these two channels to shut down until such time as it is "safe" to heal: until conditions are safe, a person does not want to initiate healing, nor does he want to be distracted by non-essentials such as eating and digestion: activities triggered by the Stomach and Large Intestine channels.

“Yeah. I’d hate to lose facial control or have my arms jerk around while someone was watching. I’m not sure that this program is for me.” I have talked with these people, argued with them, and, eventually, learned to be OK with it when they decided they would rather stick with the Parkinson’s than risk unknowns and potential embarrassment.

For that matter, some PDers told me that they would rather not recover if recovery required a shift in emotional attitude. Having to change long-held fears or give up a lifetime of cynicism would be the same as admitting that they have been holding to an incorrect mindset – admitting to doing something “wrong.” Many have eloquently, if illogically, explained that they would rather stay how they are than make such an admission.¹

And so, after we discovered the mental/emotional component, I wasn’t too surprised when a few PDers said that they would rather believe that I am wrong, and that there is no cure for Parkinson’s, than admit that they have been cultivating a pathological attitude. At first glance, this attitude was wondrous strange. But we had not yet discovered the *nature* of the mental/emotional blockage: a blockage that often includes or creates a dread fear of criticism and fear of being “wrong.”

Some PDers were very resentful when we started broaching the subject of a mental/emotional component. For example, in the first years of our website, we only posted information about the foot injury aspect of Parkinson’s. We were flooded with inquiries. After 2005, when we posted new information on our website that described what we’d discovered about the mental component, the number of PDers making inquiries about our program dropped to a trickle.

RELAXATION: THE PREREQUISITE FOR RECOVERY SYMPTOMS

Or: Department of redundancy department

As you’ve been reading along about recovery symptoms, you’ve no doubt noticed how often the description of the symptoms was followed by something along the lines of, “These symptoms only occurred when the recovering PDer was relaxed or enjoying himself.” In other words, PDers did not have to “take steps” to initiate recovery symptoms: as soon as the PDer was in parasympathetic mode, recovery symptoms occurred.

As soon as the foot injury had healed enough that energy could circulate through it correctly, no prompting was necessary to get recovery symptoms going. All the person needed to do was feel safe and relax or enjoy himself; the recovery symptoms would kick in automatically. It seemed as if, once the foot (and any other significant) injury was gone, the recovery symptoms were wanting, waiting, and willing to manifest. The only thing that could hold them back was the

¹ It never made a difference when I tried to explain that, with a degenerative disorder, people can’t “stay how they are.” I was genuinely shocked at how many people insisted that Parkinson’s wasn’t so bad and that they didn’t mind having Parkinson’s disease, because the symptoms didn’t really interfere with their lives too much. They honestly did not believe that their symptoms would *ever* get worse. I had to wonder what these people didn’t understand about the word “degenerative.” I sometimes asked these PDers if they’d ever been to a Parkinson’s support group. I was often told something to the effect that, “I went once, but the people in the group had a different kind of Parkinson’s.” or “The PDers in the group were a real mess: they weren’t like me. My Parkinson’s isn’t the severe kind, like theirs is. *Mine* isn’t a problem.”

I repeat: PDers are usually considered to be highly intelligent people.

PDer himself. The PDer could prevent recovery symptoms by being in sympathetic or dissociative mode.¹

The body of an injured person does not activate healing processes while running from a hungry lion or while dissociated. In these conditions, healing is put on hold. Ex-PDer, when in fear- or dissociation-mode, seemed able to keep all healing symptoms on hold. PDer who went back and forth between contented and anxious noticed that they did *not* manifest symptoms of healing during the anxious times. But when recovering PDer were genuinely able to feel safe and relax, the recovery symptoms proceeded as quickly as they could.

Not in the car

A PDer should *not* be concerned that recovery dyskinesia will occur while in a stressful situation. It never occurred while PDer were driving or doing any job that triggered the slightest bit of extra adrenaline.

Chapter summary

Recovery dyskinesia can be a pleasant symptom of recovery from Parkinson's. I have dwelt on it in some detail so that 1) a recovering PDer will be pleased, and not frightened, if it occurs and 2) it is so utterly opposite of the controlled, conscious movement of Parkinson's, and was, in many ways, so similar to the movement that sometimes occurred in medicated patients in response to a dopamine excess that it constituted yet another proof that our patients were recovering from Parkinson's – or at the very least, the dopamine was flowing.

When we came to better understand the processes at work in idiopathic Parkinson's disease, we concluded that there is no way that a person will experience recovery dyskinesia *if* he still has idiopathic Parkinson's disease. Recovery dyskinesia is a unique form of muscle recovery that only occurs when the brain begins reestablishing contact with muscles via the long-dormant connecting nerves. The motor movements feel uncharacteristic of PDer motor function: they feel effortless, as if they are being performed with dopamine, not adrenaline.

However – this does not mean that a person who experiences recovery dyskinesia will not be susceptible to the mood-based symptoms of Parkinson's. If a PDer has recovery dyskinesia but, afterwards, allows himself to slip into anxiety, he may well stiffen up, tremor, and find himself unable to use the limbs that, so recently, were moving effortlessly.

If a person was stuck in partial recovery and wondering if he was truly recovering or not *and* he had experienced one or more episodes of recovery dyskinesia (or for that matter, if he'd experienced several other of the other recovery symptoms), he was up against the uncomfortable conclusion that his remaining bouts of rigidity, slowness, and tremor were no longer being caused by an unhealed foot injury. His *body* was healing. His *mind* was still looping through negativity. If the PDer had recovery dyskinesia in some limbs but was increasingly susceptible to mood-induced PD-like symptoms, the logical conclusion was that he *had* genuinely had

¹ Sometimes, in addition to the foot injuries, other body parts had to be treated before energy could run in the right direction. In some cases, scar tissue, which is electrically non-conductive, was straddling one or more channel pathways, impeding the flow of energy. This problem was easily remedied using acupuncture to recreate conductive pathways through the scar tissue. In still other cases, multiple injuries needed to be treated before healthy flow of energy returned. But the larger point being made in the footnoted paragraph, that the body will initiate healing processes on its own as soon as the currents cease to flow backwards and the person resumes the "I am safe" emotional stance of the parasympathetic mode, still stands.

idiopathic Parkinson's, to start with. But now the physiological parts of the illness were healing. What he was left with, at this point, were symptoms of mind-based, psychogenic parkinsonism.

As I have mentioned before, we were not expecting any of these recovery dyskinesia symptoms. They took us by surprise. They also further proved for us that the problems of Parkinson's were *not* just symptoms of dopamine-cell death. These recovery symptoms reflected both a return of brain-to-muscle function in muscles that had not been used in a very long time and the effortlessness of dopamine-based movement.



CHAPTER TWENTY-ONE

RECOVERY SYMPTOMS: MUSCLE SORENESS

Arm swing: bicep soreness

When, during recovery, PDers' arms first started to swing, the bicep muscles hurt. This arm swing pain was *not* recovery dyskinesia. Dyskinesia means “abnormal movement.” Dyskinesia occurs when a muscle first gets back on-line and clumsily starts relearning how to respond correctly to the brain signals.

Arm soreness occurred after the arm muscles become *correctly* operational and started being used a few thousand times a day.

Prior to recovery, many of our PD patients no longer had the synchronized electrical pattern that automatically swings the arms – using the correct muscles. When, during recovery, the biceps, the muscles that *correctly* perform arm swing, started to automatically contract in synchrony with the opposite-side leg stride, the arm muscles felt sore at first, even a bit painful, when walking. The reason was this: the muscle contraction automatically occurred with every single step.

Even if the new arm swing was very tiny, almost invisible, in the beginning, that tiny movement was the result of contraction in the feeble biceps. This muscle might not have been used for years. During recovery, the biceps automatically – without the PDer thinking about it – made a healthy contraction a split second prior to every opposite-side footfall, thus forming the arm swing.

If a recovering PDer walked fifty steps, the convalescing biceps contracted and relaxed fifty times. If he walked for half an hour, the bicep contracted and relaxed more than a thousand times.

When the biceps began to work, they might have been going from zero contractions per day to many thousands. The biceps usually felt sore from the unaccustomed work out. Over the next few weeks, walking often became difficult because of the accompanying soreness in the biceps. Whether the PDer *tried* to swing the arms or not, the reconnected biceps did their healthy contraction in time with the footfalls.

Several recovering PDers who previously mourned the loss of arm swing reported the irony of trying to *prevent* their arms from swinging. “My arms are *so* sore. When I go for my morning walk, I cross my arms over my chest and try to pin my biceps down so that they can't move. Sometimes that helps. But sometimes, even though the arms can't really move, the darned biceps are still doing their repetitions in time with my walking. It's getting so that the arm soreness is limiting the distance that I can walk. I used to go three miles a day. I've cut back to half a mile a day because of how the soreness builds up in my arms. I have to admit, it's a good pain. It's like the soreness I used to get when I was in high school sports, after doing those conditioning exercises. And I can tell that my arms are getting a lot stronger. But this last week, after half an hour of steady walking, I was so sore in the arms that I just wanted to go home.”

This soreness and pain eased up when the muscles in the arm became accustomed to the thousands of daily repetitions that a healthy person makes while walking. But for a short period, maybe a few weeks or a month, the new arm swing and the resultant workout for the budding biceps was a bit painful.

Prior to recovery, PDer's often assured us that they were still swinging their arms, or could if they thought about it. They were surprised when the arm soreness began. Truth was, even if they'd been swinging their arms or doing biceps building reps at the gym, they hadn't been using their biceps.

A healthy arm swing uses the bicep. In Parkinson's disease, the bicep muscle tends to become atrophied and not responsive to brain command.

We'd observed that our PD patients whose arms still *did* swing a tiny bit were usually *not* using the normal arm-swing muscles. It looked to us as though, when a PDer's biceps atrophied, he used other arm muscles to create an arm swing.

When other muscles take over the job of an injured muscle or help stabilize an injured area, it is referred to as muscle splinting. Muscle splinting is a common, normal occurrence.

For example, some PDer's generated an arm swing by *throwing* the arms forward using muscles from the torso and the back of the shoulder. Others had an arm "swing" in which the hands and forearms stayed in front of the torso, sashaying from left to right, in time with the footfall. This is not correct arm swing. In these cases, various muscles were splinting for the inaccessible biceps. The correct arm swing is generated when the arm is *pulled* forward, from the front of the arm, using the biceps.

Although I wrote in a previous chapter that limp muscles were not atrophied, the bicep *was* atrophied: an exception to the rule.

(The chronic tension in the upper arms that causes the arms to stay bent at the elbow seems to be the result of rigidity in the muscles on either side of the biceps: muscles that, in happier days, splinted for the non-functional biceps.)

Prior to recovery, no amount of gym work on the part of PDer's ever helped the biceps get strong. If a PDer worked out in the gym, doing exercises that are supposed to develop the biceps, the only muscles that got stronger were the ones that splinted for the biceps. As long as the biceps remained out of reach of the mind, no number of weight lifts did anything for the biceps. The biceps did not work.

One PDer said that, ever since she was about twenty-five, she had not worn a sleeveless shirt. She knew that her arms didn't look right. She was very fit, and her arms were strong. She hadn't been able to explain exactly what was wrong with the look of her arms. After she recovered and her biceps returned, she suddenly realized what had been wrong with her arms: they'd been missing the bulge of the biceps. She started wearing sleeveless shirts again – not only because her arms looked normal again, but also because she no longer *worried* about whether or not she looked "right."

But the main point in mentioning the biceps situation is this: the biceps hurt when a PDer started unconsciously using them again. The pain eventually eased up as the biceps got accustomed to doing their correct job.

Back soreness

Up until now I have frequently stated that any given recovery symptoms might or *might not* have occurred in and given patient. “Every PDer’s recovery is as unique as his symptoms!” has been my constant cry.

But when it came to back soreness, this recovery symptom was almost universal. In our limited experience, all but two recovering PDers had back soreness.

The small-of-the-back soreness was usually referred to as back “pain,” but it was not pain in the classic sense. It was soreness, like a gentle ache or pulling in the small of the back. Standing up straight was an instant “cure.”

The back soreness probably occurred because the muscles in the small of the back had been over-relaxed, over-stretched. They possibly became overstretched over the years as the muscles in the front of the torso became contracted.

During recovery, when the rigid muscles along the front of the torso began to go limp, the muscles in the small of the back were once again able to contract. These back muscles might not have functioned correctly for years. When they began to contract a bit, they were over-keen at first; they tightened up slightly more than necessary.

Then again, they sometimes tightened up only briefly, then relaxed, then tightened again and relaxed again: the back “pain” came and went as the muscles played at finding their correct tension level. The soreness was felt when the muscles were tightening – *if* the PDer happened to be standing in his habitual, forward-leaning posture. When the lower back muscles tightened up (while bending forward), only one maneuver would stop the mild soreness that occurred: standing up straight.

This soreness was *not* true back pain. Back pain that is due to injury or pathological spasm usually causes a person to stand crooked, in such a way as to minimize the pain. The small-of-the-back muscle tightness that occurred during recovery from Parkinson’s disease did *not* feel better if the PDer stood crookedly to “favor” the pain. Chiropractic treatment, hot or cold packs, and other back treatments did nothing to reduce the new health and vigor in the back that was causing the soreness. The only thing that made the healthy back muscles feel good was standing up straight, allowing the back muscles to assume their correct position and strength.

This back soreness lasted for weeks or even months. During this time, leaning forward aggravated the soreness. By leaning forward, I mean the type of gentle leaning forward that occurs while washing the dishes or reaching across the bed to straighten the blanket. These gentle, forward-leaning movements caused a mildly unpleasant pulling sensation in the newly firmed up muscles. However, this problem was not severe, and could be easily remedied by throwing the shoulders back and employing good posture.

We asked complaining PDers to keep in mind that, if a person has back soreness that can best be remedied by throwing the shoulders back and standing up straight, this is not a real problem: this is your body teaching you to stand up straight once again. This is a recovery symptom.

Cases of no back soreness

As mentioned in chapter xxx, in two patients, the back muscles did *not* pull tight. Healthy tension in the back failed to automatically kick in. These recovering PDers found themselves bent far forward, with the face nearly touching the knees.

In these two cases, we did try many acupuncture and moxa techniques to strengthen the low back. These techniques did not work, even temporarily. Because both of these cases occurred prior to our discovery of the mental/emotional blockage that caused partial recovery, we did not explore the possibility of psychological inhibitions in these two patients.

Frequent urination: oversensitivity of the bladder nerves

At about the same time that a PDer experienced small-of-the-back soreness, he often felt drastic changes in his sensitivity to bladder signals.

In his youth, the pre-PDer may have had powerful bladder control. He may have been able to be out and about all day without feeling a need to urinate. This was not because he had a “strong” bladder. In these PDers, this lack of “need” to urinate occurred because his bladder nerves were somewhat numb. Only the very strongest signals from a *very* overstretched bladder could generate a strong enough signal to get through the relatively numb area in the low back, near where the bladder nerves enter the spine. The feeble bit of signal that got through to the spine and then traveled to the brain was all the PDer might have gotten, even if the bladder was actually saying “I’m really overstretched down here.”

Also, when an animal is highly dissociated from a high degree of trauma, when he is behaving as if dead, the animal might not register signals from the bladder. Those signals can wait until a safer time.

The combination of dissociation and/or an overstretched, numb area in the small of the back may have been the cause or at least contributed to this not uncommon symptom of Parkinson’s disease.¹

Oppositely, a few PDers had a lifelong weakness of the bladder, with a need to urinate every hour or so. This need for very frequent urination can be related to a high level of fear – the sympathetic system rather than the dissociative system.

At any rate, during recovery, as the spinal nerves of the low back began to be invigorated, at about the same time that the back soreness began, the nerves from the bladder also began to return to healthy sensitivity.

During the decades that the PD was silently worsening, the PDer’s brain had, in many cases, become accustomed to a very low signal from the somewhat numb bladder nerves. Because the “fullness” nerves from the bladder (the “stretch receptors”) had barely gotten any signal through to the brain, the brain had slowly trained itself to respond to the least little signal, if any, from the bladder. The brain learned that even the smallest signal from these nerves indicated a very full bladder.

Some PDers actually got few or no bladder signals during the day, unless the bladder was horribly full. The only time they felt genuine bladder fullness might be at the first urination of

¹ One PDer told me how, during World War II, his buddies highly resented his ability to stay in a foxhole all day. The other soldiers had to leave the safety of the fox hole every few hours in order to use the latrine trenches, thus exposing themselves to risk.

I heard many fascinating bladder stories, and other health stories, that all helped me put together the fullest possible picture of Parkinson’s disease. Our modern system of medicine, in which a neurologist sees a PDer for a quick fifteen minutes, twice a year, almost guarantees that doctors will not be able to make the fascinating connections between seemingly unrelated symptoms: connections that led us to an in-depth understanding of Parkinson’s. A PD team member or I spent about an hour every week or every month with each patient. We got to know their medical histories and idiosyncrasies in great depth. This was how we came to spot similarities among PDers, such as the bladder situation, that was never even guessed at by doctors who were always pressed for time.

the morning. A few had trained themselves to urinate at regular intervals regardless of whether or not they felt any real urgency. Sometimes, they got home from work at the end of a long day and, as they noticed how much urine was passing, said to themselves, “Huh. Looks like I forgot to use the toilet when I was at work today. Again.”

In recovering PDers, as the bladder signal nerves began to revive from their partial dormancy, they began sending healthier, more vigorous signals. The brain, accustomed to a tiny nerve signal, misinterpreted these larger signals: it assumed that extreme bladder stretching was occurring. When the recovering PDer’s brain started receiving what seemed like massive, steady surges of bladder information, the brain’s response was to assume that the bladder was constantly, *painfully* full.

Eventually, the PDers’ brains recalibrated their responses to the healthy bladder signals.

But until then, while the brain was still recalibrating the meaning of vigorous, healthy nerve signals from the bladder, some recovering PDers felt the “need to urinate” signal getting triggered every few *minutes*. This was very frustrating, because the amount of urine passed each time in response to the panic call could be measured in teaspoons, or even droplets. Nevertheless, the brain signals were adamant.

The recalibration of the bladder urgency signals occurred over a matter of a few days, a few weeks, or a few months. Just as the increased sensitivity in a previously numb foot tapered off when the brain accommodated to it, the increased sensitivity to the bladder signals tapered off over time.

Going through hell

If this bladder urgency phase occurred at the same time as the extreme limpness in the legs phase, the recovering PDer found himself in a difficult situation. Some PDers needed help to stand up and get to the toilet. During the night, they quickly exhausted their spouses with their constant demands for help. Some PDers referred to this phase of recovery as “hell stage.”

Some PDers merely went through a mildly annoying period of increased bladder sensitivity. Others found the bladder sensitivity-limp legs combo to be the most trying part of the recovery process.

We suggested that, if the bladder urgency was severe and preventing one from getting sleep, a man might want to get an external (condom-type) catheter to wear at night. For women and men, we also suggested that there was no shame in wearing adult diapers at nighttime.

Bladder infection

We also warned that a bladder infection can cause symptoms of bladder urgency. It is reasonable to ask the doctor for a urine test (urinalysis) if one develops symptoms of bladder urgency. Many people develop bladder infections during times of stress. Recovery from Parkinson’s can be stressful.

Bladder infections are a serious illness, and must be treated. Bladder infections in younger people are usually associated with frequent urination, burning pain when urinating, dark colored urine and sometimes fever.

However, in older people a bladder infection may be nearly symptom-free, in terms of bladder-specific problems. In older people a bladder infection can be present with no fever, no pain, or not even noticeably frequent urination. But even if no fever is present, a bladder infection is still a serious health problem. In older people, a bladder infection can cause extreme weakness and mental instability, even if there are no obvious bladder symptoms whatsoever. If

there is any possibility that a recovering PDer might have a bladder infection, he should seek diagnosis and treatment.

Many PDers, having read about recovery symptoms, have wondered whether or not they should risk entering into a program that warns of such humiliation and inconvenience. Is recovery from Parkinson's disease worth it?

Everyone who has fully recovered has felt so grateful for the return of a healthy body that the recovery symptoms, in retrospect, were nothing to complain of.

“You look terrible!”

Very often, PDers started to look haggard during the phase of frequent urination and its corollary of poor sleep.

Part of this was due to the fatigue, but part of this was due to the greatly increased expressiveness of the face. If, prior to recovery, the PDer had a fairly inexpressive face, and his face was now projecting “I feel exhausted and discouraged, I can't get up out of chairs, I pee all the time, my feet hurt and I'm crying a lot,” his best friends often told him how bad he was looking.

This new development – frank concern from friends – was a completely new experience for some PDers.

When these comments arose, PDers were often deeply concerned. They were going through so many changes and they couldn't be certain that these changes were for the better. When someone said, “You look terrible,” the PDers usually incorrectly understood this to mean, “The Parkinson's is getting worse.”

Also, PDers often did not have the social skills to know how much information should be shared with concerned friends; PDers, in many cases, were not accustomed to being on the receiving end of sympathy.

Prior to recovery, most of the PDers we've worked with didn't mind *giving* sympathy – but they didn't like to receive it. Many did not even know how to deal with the *idea* of receiving sympathy. So when concerned friends started to speak up, it was an ego-challenging time. And then, the emotional discomfort and the fear that the Parkinson's was getting worse very often caused an increase in mood-based symptoms.

We had to wonder if one reason for this outpouring of compassion from friends and co-workers was that the PDer had finally ceased projecting supreme competence. Spouses of several PDers assured us that the PDer had, for decades, unconsciously projected the signal “I don't need your help.” Many projected an air of inapproachability, especially with regard to personal matters.

In the past, the PDer's facial immobility might have conveyed internal strength or lack of compassion. After recovering tone in the facial muscles, that immobility was gone. The PDer no longer had a poker face. As the PDer's face became more expressive, emotions such as sadness, fatigue, boredom or resentment became apparent.

Also, prior to recovery, most of our PD patients admitted that they had probably been adrenaline-dominant for a lone time. Adrenaline tends to push people away. In the past, a PDer's friends might not have felt safe about offering compassion, even if they thought the PDer could use some.

During recovery, as a PDer's face became expressive, his protective walls crumbled, and his adrenaline dropped, he often started to come across as a person who *could* be comforted. His wounded body and fatigue sent signals to his family and friends: "I'm tired and sick and I need help." Any sensitive human can pick up on these electrical, chemical, and body language signals. But many PDers had no experience at being on the receiving end of compassion.

Some family members were grateful that the PDer was becoming more "humanized." But sometimes, when a PDer started coming across as more vulnerable, family and friends were horrified by the PDer's changes. The PDer often had been "The Rock" or "The Capable One" of the family or social group. When the PDer became less heroic (less adrenaline-driven) and more "human," family members sometimes resented the changes in family dynamic and the new need to step up to the plate.

The change in facial expression

Many a PDer who assumed that his friendship was shown through his actions, who imagined that he was projecting sincerity and friendliness, and who prided himself on his never-changing look of stern intelligence was stunned when a "friend" or co-worker told him, during recovery, "I always assumed you didn't like me because you always frowned at me" or "...because you never smiled at me." Some PDers were outraged by these confessions. Others were thrilled by their deepening appreciation for how emotionally frozen they had become and by the "miracle" of their recovery.

If the PDer was still living in fear, negativity, or anxiety, he often projected his own fear- or cynicism-based motivations onto those who would help him or be concerned for him: he assumed that people were being condescending, scornful, or judgmental.

For those PDers who had tended to be aloof or "able to stand alone," we gently suggested that, during recovery, if family and friends started to express concern that the PDer looked exhausted, looked worried, or suddenly looked his age (instead of looking sort of frozen), he should celebrate. He was recovering from a long descent into lifelessness and separateness. He was coming back to life after decades of emotional separation. He was emotionally rejoining the living. And the living can be very loving and compassionate: they may express concern for what they perceive to be a person going through a challenging time.

Other PDers, those who had worked to keep their hearts open despite the body's steady decline in physical functionality, those whose mental/emotional blockage was directed only at the foot injury and not towards a generalized fear of emotional pain, did not need coaching as to the *bona fides* of their family and friends. They appreciated the family and friends' concern and the caring spirit in which it was offered.



CHAPTER TWENTY-TWO

RECOVERY SYMPTOMS: CHANGES IN SLEEP PATTERNS

One of the more pleasant, or more aggravating, symptoms of recovery was the change in sleep patterns.

Improved sleep

During recovery, PDers who had been plagued by insomnia found that they were suddenly sleeping very well.

“Too much” sleep

But some PDers found themselves wanting to go to bed early, wake up late, and take naps. They felt they were sleeping too much. Many complained, “I’m sleeping my life away.”

I reminded my patients that a person recovering from a serious injury or ravaging illness needs to sleep a lot. Since they were growing new nerve cells, muscle cells, and brain cells, they needed sleep: lots of it.

Some accepted this. Some were unable to accept it, and continued to feel chagrined, or even ashamed, about how much sleep they were getting.

Concerned about sleeping too much

Many PDers, by the time they were diagnosed, had started having a hard time sleeping through the night. In the beginning of recovery, this new ability to sleep well seemed like an answer to prayer. However, many recovering PDers soon had to pit their glorious new ability to sleep against their lifetime conviction that people who sleep a lot are lazy and bad.

Many PDers moaned to me that they weren’t getting “anything done” because they were sleeping so much. I had to point out to them that they were getting a lot done. They were growing new nerve connections, new muscles. And even if the foot injury had healed, the body might need to sleep a lot while healing other injuries that were finally accessible to the brain.

While this may not seem like a big problem to the reader, the extra hours of deep sleep was a real emotional challenge for many recovering PDers. Many PDers felt genuinely bad about themselves when they weren’t accomplishing a lot. This was a serious problem. It made recovery a time of guilt-induced anguish for some PDers.

Prior to their diagnosis with Parkinson’s, many PDers, unable to feel their own bodies and, in some cases, their own emotions, had valued themselves because they “got a lot done.” During recovery, many PDers avoided doing anything that wasn’t a stark necessity. One PDer said, “I plan my days around being able to get as much sleep as possible. If an activity isn’t absolutely necessary, I won’t do it.”

When the ability and *desire* to be a workaholic dropped away, some PDers felt ashamed and even depressed. As you can guess, depression is a condition in which the release of movement neurotransmitters is minimized.

A case study in “laziness”

I recall one recovering PDer who, like many people with Parkinson’s, worked three jobs. She was a math professor by day, a software designer in the evening, and a regularly performing professional musician on weekends. During the “increased need for sleep” phase of recovery, she found herself parking as close as possible to her classroom to minimize her walking distance, turning down music jobs, and unable to stay up at night to work on the software. She didn’t need the extra money; she was a successful single woman with no dependants, so the problem with working less was purely a self-worth issue.

While she was in the thick of this phase, she came into my office and exploded into tears. When she was finally able to talk, she told me about the above changes in her lifestyle. I asked why this was a problem. She replied, “I’m turning into the people I hate! But that’s not the problem. The problem is that I never knew I hated anyone!”

She was a very loving, kind and helpful human. She had always assumed that she loved everyone. But when she found herself minimizing her daily activities so that she could sleep more, her inner thoughts screamed at her, “You’re turning into the people you hate!” This realization, that she had subconsciously been judging – and finding lacking – those people who did *not* work three jobs and constantly exert themselves, made her deeply ashamed of herself.

She wasn’t sure what she was more ashamed of: her lack of interest in “doing it all” or her new realization that she had been looking down at people that were less active than herself.

The reader may think that patients like this were making a big deal out of nothing. But the increased need for sleep and the decrease in “getting things done,” or “making things happen,” was sometimes a devastating emotional experience for those people who had only valued themselves on the basis of what they were able to accomplish.

How long did the need for extra sleep last?

The need for extra sleep was not a straight line. Some people only needed a few extra hours a day, or a nap on weekend afternoons. Sometimes, this increased need lasted for months. In some cases, it lasted for years.

Very often, the “new” amount of sleep was actually a far healthier amount of sleep. Many of our patients had chronically shorted themselves on sleep. In some cases, the PDers never went back to their old patterns of getting by with less sleep than most people. As they recovered, they no longer felt the adrenaline-based drumbeat that forced them to sleep as little as possible. The new, “lazy person” sleep pattern was sometimes just a “healthy” sleep pattern.

Some PDers accepted happily the new amount of sleep that their bodies needed. Others slid into despair because they honestly felt that they were sleeping their lives away, with no end in sight.

In general, younger people needed much less “extra” sleep during recovery than older people. For example, one PDer in her thirties, who started our recovery program immediately upon diagnosis, only needed several days of staying home and sleeping all day. A retired PDer in his late sixties who had an advanced case of Parkinson’s found that, during recovery, he slept long and hard at night and then needed two naps in the daytime. However, there were no hard and fast rules, and there were variations and exceptions. To say nothing of the fact that some people in their thirties had an advanced case at the time of diagnosis and some people in their eighties had a mild case when they were diagnosed.

Every PDer’s symptoms are unique, etc.

Shifting sleep patterns

Some people noticed that their sleep patterns went through constant changes. For several months, a recovering PDer might have fallen asleep at nine at night and awakened at seven in the morning. A few months later, he consistently might not be able to fall asleep until three in the morning and then he would sleep until noon. And then, a few weeks or months later, the pattern changed again.

Anyone who has raised a child will recall that children go through periods when they need an enormous amount of sleep, and other periods when they don't even need to nap. These periods of increased sleep and altered sleep patterns correspond to periods during which the child does an enormous amount of mental, emotional, or physical growth.

In a PDer, these periods of increased sleep also corresponded to enormous amounts of healing and growth.

Two-hour intervals of deep stillness

A few PDers noticed that, even if they didn't feel tired, their bodies fell into heavy, drugged-like physical immobility for about two hours. These two hours of stillness sometimes occurred at the same time every day, and went on for several days or even for months. This very weird type of sleep seemed similar to the sleep induced by strong pain medications such as Demerol: the recovering PDer might be perfectly conscious, deeply relaxed, and yet unable to move any part of his body – appearing to be asleep even though he was conscious.

This type of “sleep,” or utter immobility while alert, usually only occurred, if it occurred at all, for about two hours at a stretch. After the two hours of feeling drugged was over, the recovering PDer quickly resumed whatever his normal movement ability happened to be. But the next day, at about the same time, he might, once again, have two hours of gentle, but utter, immobility. And again the next day. And the next.

Only a few patients had the two-hour, same time every day, limp paralysis. Of those that did, most of them only had it for a few days. One person had it for a week. One person had it for four months. The person who had it for four months also recovered from Parkinson's extremely quickly in every other respect: all of her obvious PD symptoms were gone within two months of the time she started treatment.

Channel theory

We suspect that these two-hour periods of extreme limpness and immobility corresponded to healing activity in specific electrical channels. As you will recall from chapter xxx, every primary channel has a specific two-hour period during the day when the channel is most active.

For example, the Stomach channel operates at its highest amperage level between 7 and 9 in the morning. (These times are approximate and are based on the sun and an individual's circadian rhythms. They are *not* based on Greenwich Mean Time.) If the body recognizes an area serviced by the Stomach channel that needs intensive repair, the body is most likely to do this repair work between 7 and 9 in the morning. During this channel's time, if the body is doing *enormous* amounts of healing work on any damaged places that are located on the Stomach channel, a person who is mentally relaxed may not be able to move any part of his body. He may feel limply, gently paralyzed, or very, very sleepy, from 7 to 9 in the morning – Stomach channel time. He would snap out of it when the Qi starts flowing more heavily in the next channel in the sequence, Spleen channel, from around 9 to 11.

When this type of two-hour paralysis event did occur, the person – who might have had nearly normal physical function during the rest of the day – had a bit of warning time: he felt himself slowly relaxing, over a period of about ten minutes, into the deeply calm stillness. Once he was comfortably ensconced in it, nothing short of an emergency was able to pull him out of it. And at the end of the two or so hours of gentle, “soft” paralysis, he found himself able to move perfectly normally again – until the same time the next day.

These bouts of non-moving were pleasant, even amusing, if the PDer could take them in stride. They might or might not occur on a daily basis until the area being worked on was healed. Also, after a person no longer had 7 to 9 in the morning (or whenever) soothing paralysis, he sometimes developed a new two- or so- hour stint of calm stillness at some other time of day.

For example, one person who needed a lot of repair work around his shoulder blades found himself unable to move from 1 to 3 in the afternoon for a few days while the shoulder blade area recovered more movement and feeling.

Again, it was *not* the rigid type of paralysis that a person might have following a stroke or polio. It was a time of softness, a deeply calm period during which a person might be awake or asleep, during which his ability to move was turned off. It is somewhat similar to the deep, relaxing rest that can come after a high fever has broken and a person slides into a deep, healing stillness during which he feels absolutely no interest in moving a single muscle.

This stillness did not occur if an emergency was ongoing. It happened in people who had made a decision to surrender to their bodies’ inclinations to whatever extent was humanly reasonable.

An example

The PDer who had morning “zone outs” for four months could get up any time in the morning, so long as it was before 7:00 a.m. Then, at 7:00 a.m., she would become limp, unable to talk, barely able to blink. At 9:00 a.m., she would “snap out of it.” Within fifteen minutes, she would be back on her feet, functioning normally.

She rescheduled her work around this daily period of inaccessibility by changing her start time from 9 a.m. to 10 a.m. No one she worked with ever knew that she spent two hours a day awake yet utterly unable to move.

This same PDer desperately wanted to attend a particular museum showing in Victoria, BC. This meant leaving the house at 5:30 a.m. to take the ferry from Seattle to Victoria. She had always loved being on the water. She never felt safer than when she was on a boat. She went limp at around 7 a.m., as she and her best friend were getting on the ferry. Her friend helped her, almost dragging her, to a seat in the ferry café. She sat, slumped, eyes half opened, body unmoving, for nearly two hours, while her friend chatted about what the kids were doing at school. Then, about twenty minutes before docking in Victoria, the ex-PDer started conversing with her friend in what had been, up until then, a monologue. Within a few minutes, she was talking and joking effortlessly.

Her friend was mildly alarmed. “You’re normal again! You told me about these two hour ‘quiet times,’ but I didn’t believe you. That’s the weirdest thing I’ve ever seen. I’ve been sitting here wondering how the heck I was going to drag you all over Victoria. I thought you were sick or something. I was getting ready to call for a doctor, and now you’re perfectly normal!”

Absolutely normal! It's the weirdest thing I've ever seen. And you've been doing this every morning for how long?"

Afraid of needing sleep

Many PDer's have decided not to enter the recovery program because they were frightened by the idea of needing lots of sleep. The number one reason that PDer's have given me for choosing not to enter a recovery program is that they cannot afford to miss any days of work. Some have also feared that, in order to explain to the boss or co-workers that they need more sleep, they might also have to confess the horrible fact of having been diagnosed with Parkinson's disease. They fear that either the increased need for sleep or the admission of having a physical illness will cost them their jobs.

Being afraid to miss work, or being afraid of appearing lazy (another popular reason for not wanting to attempt recovery) during the period of increased need for sleep, is a fear-based attitude that slams right up against what many PDer's think they need: accomplishments. As noted before, many PDer's value themselves only in terms of what they accomplish each day.

Seen in this light, it is understandable that many PDer's are *afraid* of anything that might rock the I Get Things Done boat – including recovery.

But, as noted above, in an emergency, PDer's *were* able to move. We observed that, if work was an emergency for a PDer, he was able to attend work. However, if this was the case, he also spent his days off in sleeping around the clock. Recovering PDer's seemed to be able to work around employment needs while still doing whatever recovery work had to be done – within reason.

Fear that recovery symptoms will expose the PDer's secret

Another reason often given for not wanting to enter a recovery program was the fear that, in order to explain any embarrassing recovery symptoms, the PDer might have to inform friends and co-workers that he had been diagnosed with Parkinson's. As opposed to those who merely feared momentarily looking like an idiot in public because of recovery dyskinesia, the possibility of missing some days of work due to increased sleep needs seemed to strike deeply at the hearts of those PDer's who were afraid of being "found out." Sad to say, this was *not* a rare fear.

"Cure me fast so no one will suspect I've had Parkinson's"

One PDer came to visit our program in Santa Cruz, and opened his first appointment by asking, "Can you cure me really quickly so that no one will ever know I had Parkinson's disease?"

I said that I couldn't promise anything, and asked why it mattered if people knew he'd had PD.

He replied that he was a physical fitness coach. "I make a good living because my clients think that I'm healthier than they are. If they knew I had a physical problem, they would reject me."

I told him that he might be wrong, and that if he was able to show his clients how he could confront and recover from a difficult illness, they might respect him even more. He was adamant that I did not understand, and that everyone would despise him and accuse him of being a liar if he promoted fitness when, in fact, he had Parkinson's.

I said that it seemed to me that if he truly felt that he was sick, and was pretending to be well so that he could continue to mislead his clients, then he *was* lying. He replied that he had to

lie, to be able to make a living. So I asked if maybe his problem wasn't so much that people might think he was a liar, but that they might find out that he actually *was* a liar. He agreed, and didn't seem to understand that the latter situation reflected the more poorly on him.

He then said that people would *hate* him if they knew that he was being a fitness coach while having Parkinson's disease. We went back and forth on this for some time. When I refused to guarantee that he could recovery quickly and in such a way that no one would ever know he'd been "unfit," he decided to not enter our program.

He was not the only one. Many PDers feared that, by divulging the fact of physical imperfection, they would make themselves vulnerable to scorn or even some sort of unnamed danger. After a while, we began to suspect that, in PDers with a severe mental/emotional blockage, this was actually an integral part of the Parkinson's pathology.

An injured animal, while still in the thick of the skirmish, does not want to show any signs of weakness to the enemy.

Many PDers, neurologically behaving like injured animals, or "walking wounded," feel that they are not yet in a place that is safe enough to allow them to let their guard down. Sadly, some have cultivated such a powerful attitude of wariness for so many decades that they cannot easily change. Worse still, some of them feel that any *change* in this attitude would be a sign of weakness, a sign of surrender. If I had to pick one word that many PDers dread more than any other, it would be the word "surrender."

Quitting the job

Faced with the idea of possibly needing to miss a few days of work because of needing sleep, many PDers decided to quit working. They usually stated that they could recover faster if they had time off to "focus" on their recovery. We now discourage this type of thinking.

We saw that PDers who continued working during their recovery fared much better. Isolation does not encourage learning a new mindset – it allows a person to wallow in his old ways. The regular presence of others was a boon to the PDers who had never known that other people can actually be extremely helpful – even if "other" people were usually thought to be slower, stupider, and lazier than the PDer. Learning to *expect* help from others is a part of learning to feel safe. And feeling safe releases dopamine. Dopamine release does *not* occur when a person is hiding from challenge.¹

Help in unexpected places

Those people who continued working and who even shared with co-workers the fact that they had Parkinson's and that they were trying to recover found an enormous weight lifted from their shoulders when they learned – often to their shock and amazement – that their co-workers are able to take their "failure to be perfect" in stride. Some even received unexpected support and strength from co-workers.

We now recommend that a person continue working or performing his normal activities of daily living to the best of his abilities, while allowing recovery symptoms, including his increased need for sleep, to manifest during his weekends and his "down time" in the evenings.

¹ Some recovering PDers really have a hard time understanding that dopamine release is *not* the result of feeling good. Dopamine release occurs when 1) a person feels safe and 2) a person *expects* to experience the joy of being alive *whether or not* he gets to do what he wants. Staying home from work to avoid doing something is the exact *opposite* expecting to feel the joy of being alive no matter what the circumstances.

Once in a while, when he really needs to make an exception and grab a few hours, or even a day or two of nothin' but sleep, fine. He needs to learn how to feel his body and respond to its needs – even sleep needs.



RECOVERY SYMPTOMS: BRAIN SHIFTS – AND MORE HYPOTHESES

Some of the most delightful symptoms of recovery, symptoms that could not possibly have been expected by anyone, were the intra-cranial brain reorientation movements.

These movements, which I named “brain shifts,” were fleeting events that felt as if the left and right brain halves were shifting position relative to each other.

There were three types: front to back, top to bottom, and side to side.

Front-to-back brain shift

In what we called a “front-to-back” brain shift, a person felt as if the front and back of his brain had moved further apart.

The shift often started with a feeling that the frontal bone (forehead) was gently pulling away from the rest of the cranium, moving forward a fraction of an inch. Just after the head felt very relaxed and light due to the decrease in internal pressure in the cranium, a sensation occurred as if the front lobe of the brain was gently floating upwards and forward. The back of the brain moved slightly down and back at the same time or a moment later.

The sensations passed very quickly, in the time that it takes a muscle to relax. The sensations were non-dramatic, and were followed by a faint awareness of decreased tension in the head.

Top-to-bottom brain shift

The top to bottom shift sometimes felt as if an enormous internal pressure was compressing the brain down onto the brain stem. This brain shift was described several times as feeling “as if a too-tight swim cap” was compressing the head. During this brain shift, people sometimes felt a strong urge to stomp the feet, almost as if they were trying to tamp their spines down into a better position while the head pushed down on the spine, as well. There were variations that didn’t include the “too tight swim cap,” but which seemed overall to fit the idea of a top-to-bottom brain shift.

For example, one person gave this description of the event: “I was sitting in the concert hall listening to a piano recital, and all of a sudden I turned my head and there was this deep piercing pain in the very center of my head. It went from the very top of my head down to the neck part of my spinal column. How can I describe it? I could say it’s like I pulled a muscle or something, but that wasn’t really the feeling. I was like a big train running through the center of my head down to my spine. Was it painful? Well, it didn’t really *hurt*. The big shock went away in a few minutes, but the aftershock stayed for about twenty minutes more.”

Another person said, “A massive weight was bearing down on my head, and it made me want to stomp the floor. My husband just stared at me with raised eyebrows as I vigorously raised alternating legs and brought them down hard on the floor. It felt so good. It seemed as if I stomped for ten minutes, but it might have been a much shorter time than I thought. When the pressure and tightness around the head stopped, I stopped stomping. And then I felt really relaxed inside my head.”

Side-to-side brain shift

The side-to-side adjustment was the most strange and glorious. It usually began on the healthier (less PD-affected) side of the brain, behind the ear, just above the mastoid process.

It started off as a low rumble, so slow as to be barely audible. At this stage, it was almost more of a feeling than a sound. As the vibration picked up speed and rose in frequency to an ever-higher pitch, it vibrated through the very center of the head. At this point, the PDer sometimes found himself thinking with awe and acceptance, “Wow. I’m about to die.” The vibration continued to increase in speed and rise higher in pitch until it made its way out through the opposite side of the brain, in the area near the temple. After this, a deep stillness was felt in the head.

After the vibration exited out the side of the head, the internal tremor (the drum master that sets the beat for the visible, external tremor) was sometimes truly over, or it was greatly diminished.

The sensation of internal stillness that followed the side-to-side shift was so profound, so peaceful, that some PDers assumed, for a moment, that they had died.

After experiencing a side-to-side brain shift, a PDer sometimes needed a few moments to ascertain that, *despite* the interior stillness, he was still alive. Sometimes, only after noticing that he was breathing, did the PDer realize that he hadn’t died.

Years before any symptoms of Parkinson’s disease had appeared, many PDers had long felt the presence of a constant internal tremor, a tremor that eventually drove the physical tremor. Others had not suspected that an internal tremor was firing off, driving the visible tremor. Either way, it felt unbelievably good when it stopped.

The side-to-side brain shift was usually a deeply significant moment in recovery. Some people slept right through it, and only realized that it must have happened because they woke in the morning with no ability to activate a tremor and with a sense that something unspeakable was gone.

Most of the PDers who were awake during the brain shift say that they assumed that they were dying. And when they decided that this death was going to be OK, even wonderful, that’s when the vibrating rose in pitch and finished its work. The word surrender comes to mind...

One person who experienced the head shift but who continued to tremor slightly told me that, even though she was still trembling, it was only a physical tremor and not nearly so annoying. She explained further, “There’s no tremor in my head any more. I have a shake in my arm when I’m standing, and it’s in my hip when I’m lying down, but it’s more just a shaking, not a tremor. It’s not nearly as annoying. Before, *I* was shaking; something inside of me was shaking. Now just my arm or my hip is shaking. It feels more like a muscle habit; I can laugh at it, now.” (As we were to discover, she still had yet another unhealed injury that was affecting her hip.)

In my office

I was fortunate enough to observe one person experience a side-to-side brain shift while she was in my office, while I was holding her shoulder, doing FSR.

Her eyes got huge. She brought her hands up to the sides of her head. Her mouth opened in a silent scream.¹ The whole thing lasted less than a minute. After several minutes of trying to describe for her husband and me what had just happened, she laughed and exclaimed, “I feel *good!*” She still had some recovery symptoms to go through, but she essentially felt, at her core, that she had suddenly, unmistakably, become “all better.”

After that, there was no way that she could go back to having idiopathic Parkinson’s. She couldn’t raise a tremor even when she tried: a few weeks later, her mother had to go to emergency room. The PDer felt guilty because “After all these years of tremoring when I was stressed, I wasn’t even able to get any tremor going when my own mother was in the emergency room.” She was never again able to get a tremor “going” for any reason. After the brain shift, she had no internal tremor; she never again experienced a visible tremor.²

One PDer’s description

The following is a partial transcript of a tape recording made in my office of a patient who had experienced the side-to-side head shift a mere four hours before coming to my office. “You’ll want to get this down,” he said, when he arrived for his weekly session. He was the first person to experience the side-to-side head shift. Since then, several others have shared similar stories.

“I had an awesome experience, just awesome...the upshot of it is that some life form, or some piece of me, some part of me, died during the night. And it may have been the Parkinson’s part...I had the feeling that something was coming to an end, it was as though I were dying, but I was aware that I didn’t think I was really dying...I wasn’t getting ready for my own real death, but as though some part of me or something in me was dying. And it was a totally unique experience. I went with it, breathing was fine, and whatever it was then moved to the point of dying, letting go...(long pause). And I still am not sure what are the best words to use. (His eyes filled with tears.) Time will get some perspective on that too. But it’s as though something was lifted. And I gave it plenty of time to go, and respectfully said goodbye to it. And then when I was sure it was gone, realizing that would be the end of whatever that was, I got up on the side of the bed. (Long pause.) At that point, I was aware, more keenly than before, that I was actually alive, that it was not a death experience, that I wasn’t getting ready to have a heart attack or die, but something was dead, something was gone, something was lifted and I had a strange experience of...lightness and ...smoothness (tears)...those two words were real clear. And I knew at that point that I was not dying, that I was not dead, that I was continuing to live...I was clear that this was an experience about me, and the interesting part was that I felt free of tremor, and it was unbelievable. But I’ve made a career out of being open to believing the unbelievable, so wait and see, time will tell, but clearly this was some kind of important experience.

“It was clear that I was tremor-free. I had a sense of balance and solidness that was new, that I didn’t really want to test, for fear that it wouldn’t really test out. So I started gently testing it. Oh. There was also a sense of symmetry that hadn’t been there before. So I kept testing it

¹ When another PDer emailed to me that he had experienced the side-to-side brain shift, he described it as being “a Silent Scream, like in the famous painting” by that name. The term fits so perfectly that I have used it often since then to describe the fleeting moment of seemingly altered consciousness that a person experiences when the brain hemispheres vibrate themselves back into their correct position.

² I write this in 2008. I saw her just a few months ago for a chest cold. She has had no Parkinson’s symptoms since her brain shift occurred in 1999.

putting my arms above my head, by putting them out in front (He gestured, with hands straight up, then out to the sides, then out in front and demonstrated the wrist movements.), looking for tremor, turning 'em, looking for cogwheel, not seeing anything, doing a bunch of touching, testing, touching the back of my head...and in the middle of this, I got up, twice, and tested my stability and balance, and ease of movement, and it was there!

“It was easy movement, it was as though I was without Parkinson’s...oh, then at one point, I said to myself, I wonder what my writing is like. So I have a pen, and a notepaper right by the bed, right by where I was sitting, to the left of where I was sitting, so I got that, held the little telephone note pad in my hand. So it’s not the steadiest thing, so I wrote something. What I wrote, interestingly enough, was ‘I am a renewing person. I am a renewing being’ and I (pause) looked at it and it was (tears again, choking a few times) luh, luh, large (choke) handwriting. (Crying.) Not micrographia! And it was a little scrawly, but then I reminded myself that my handwriting had always been scrawly, but it was just naturally as big as it used to be! (Gestures, thumb and forefinger showing an inch in height)...”

Other PDerS, after the side-to-side brain shift, also had a similar sense that Something Had Changed. Each PDer explained it differently.

Some PDerS experienced just one of the brain shift patterns. A few experienced all three.

Fleeting dizziness

Not everyone who recovered felt a distinct pattern of brain shift. Sometimes, recovering PDerS just had a moment of dizziness, as if the room was spinning, after which, they felt calmer inside and the tendency to tremor was gone.

One PDer told me that she’d been sitting on her living room couch next to her grandson, watching TV just prior to going to bed. When “the earthquake” hit, she dove to the floor and threw her arms over her head to protect herself from falling objects. When the shaking stopped, a few seconds later, she looked up at her husband and grandson who were staring at her in amazement. “Didn’t you feel the earthquake? It was huge! The whole room was spinning around.” They shook their heads. There had been no earthquake.

Another reported that he’d had a brain shift while he was relaxing, with eyes closed, during a plane flight. He felt the airplane dip one wing deeply to one side until the plane was almost sideways. Then the plane dipped deeply to the other side.

“Violent turbulence!” he said to himself. He grabbed onto his armrests, braced for anything, and opened his eyes. That’s when he saw that no one else around him was reacting. The flight attendant was calmly pouring a beverage. He looked at his own beverage. The liquid was not sloshing. The fleeting turbulence had been inside his head.

His tremor had been small and intermittent. After the “turbulence,” he never tremored again.

Various terms such as “room spinning,” “loop-de-loop” and “earthquake” have been offered to describe the fleeting perceptions that occurred while the brain was repositioning itself. These were painless, fascinating shifts that resulted in a decrease in internal tremor.

Again, many people never experienced these events while awake. However, they suspected that some distinct event, possibly one of the brain shifts, *had* happened to them at

night, because they woke up and felt unaccountably different: taller, calmer, and in some cases, permanently free from tremor.

A SPOT OF THEORY

If you'll forgive a bit of theory right here, a left-right brain hemisphere misalignment may be a perfectly normal shift that occurs when the Stomach channel makes its short circuit into the Gall Bladder channel at the corner of the forehead. Certainly, a sustained, asymmetrical (one side of the head only) surge of "severe injury: go to sleep" current can cause a slight shift in the left and right side *electrical* symmetry of the larger brain currents. Over years, this asymmetrical surge of "injury: go to sleep" current may be enough to cause a slight physical misalignment. This misalignment seems to be the source of the electrical "rattle" in the head: the internal tremor.

During the side-to-side brain shift, the sensation was sometimes described as a feeling that the two sides of the brain were vibrating themselves back into alignment. And when the alignment was correct, the internal rattling stopped. There was no more internal tremor. Period.

As mentioned many chapters earlier, many PDer's remember a moment, very often in their late teens or early twenties, when they experienced a fleeting, one-time sensation of buzzing, whirring, or spinning near the temple or even inside the head. They sometimes said it felt as if the room was spinning, or the inside of the brain was spinning. A PDer's pathological cerebral shifts and, very often, the first faint drumbeats of the internal tremor, may stem from that moment.

Very possibly, a pathological shift in brain hemisphere orientation is a healthy event that follows on the heels of a severe injury that has not been addressed even after a long time has passed. In an emotionally healthy person, this pathological brain shift creates an internal discomfort that should cause that person to actively seek comfort and healing for his nearly forgotten injury. After the injured person lets himself be comforted, healed, and is feeling safe, the pathological brain shifts have done their job. The hemispheres can then relax back into their correct orientation. In an emotionally healthy person, the tremor may serve as a reminder signal that the injury hasn't yet been addressed. Seen in this light, the tremor is helpful and healthy, not pathological.

During recovery from Parkinson's, the brain-shifting experiences were very possibly healthy, corrective movements in which those injury-based cranial shifts were being reversed: the brain parts were able to slide back to their correct positions. It certainly felt, during and after the head shifts, as if the muscles around the cranium were relaxing *and* the various parts of the brain were realigning themselves.

After experiencing the side-to-side brain shift and going on to become fully recovered, some PDer's suspected that, even in the *decades* prior to the external tremor becoming obvious, the left-right misalignment of the brain hemispheres had been the cause of their faint, internal tremors. These vibrations had been such a part of their lives for so long and had been so constant that, when they stopped, the PDer's could not remember having experienced such internal stillness: many of them thought they must have died. Certainly, the internal vibration long preceded the diagnosis of Parkinson's disease.

We now suspect that, not only does the brain misalignment cause an internal tremor that might lurk for years prior to the onset of Parkinson's disease, but it also causes a person to be more likely to launch into an external, visible tremor: setting the stage, as it were.

Thanks to the tremor-stopping, side-to-side brain shifts that we saw in or were described to us by recovering PDer's, we now have a much better basis for hypotheses about what used to be the most baffling symptom of Parkinson's disease: tremor.

If we can conclude, based on the tremor stopping, side-to-side brain shifts, that a hemispheric misalignment is at the root of the Parkinson's tremor, we can draw up the following hypotheses about the apparent variations in Parkinson's tremor.

Tremor hypotheses

Resting tremor

As the PDer's brain hemispheric misalignment worsens through the years, the internal vibration finally becomes large enough that it becomes apparent in those body parts that have lost their connection with the motor control area of the brain.

For example, in classic idiopathic Parkinson's disease, the muscle between the thumb and forefinger, the muscle against which the shaft of the pen rests when a person writes, very often becomes atrophied. When the internal tremor first manifests visibly, it is often in this atrophied, mentally-disconnected region of the hand. This is a resting tremor: it occurs when the PDer is not using his hands. If he does use his hands, the activity in the other muscle sweeps the atrophied area along in the movements, and so the tremor appears to stop. But when the PDer is sitting still, the disconnected bit of muscle in the hand vibrates in time with the only signal it can still perceive from the brain: the internal tremor.

Over time, as more and more body parts become disconnected from conscious control, the number of areas in which tremor can manifest increases. Instead of just a small, index finger vibration, a PDer may eventually end up with resting tremor down the entire length of the arm(s), and/or the leg(s) and/or the chin. As the Parkinson's worsens, the resting tremor often increases in amplitude and in the number of areas that are affected.

In addition to the increase in the number of disconnected body parts, the brain's hemispheric misalignment, which causes excess electrical activity in the brain, may also become more pronounced over the years. As it worsens, the amplitude of the tremor (the *size* of the back and forth motion) increases.

Fear-based tremor

At some point, the resting tremor may feel as if it has been joined by a tremor that is activated by stress or any negative, anxious, or worried thought. The negative thought may be as seemingly benign as the waking realization, "Today's a work day," or the self-conscious, "My hair looks lousy."

The stress- or fear-based tremor can seem like the exact opposite of the resting tremor, which appears when the PDer is sitting around calmly.

However, the resting tremor and the fear-based tremors are related. When a PDer happens to feel a small amount of fear, enough to cause a faint, internal trembling, the trembling resonates with the internal tremor, enhancing it. This enhancement of the internal tremor by what would ordinarily be a faint, invisible, internal shaking from fear causes amplification of the internal tremor and consequently, amplification of any external tremors.

Again, fear-based trembling, a normal, brain-based event, very possibly resonates with the PDer's internal tremor and amplifies it.

If the PDer becomes increasingly inclined to anxiety or wariness, he will also be inclined towards increased and amplified tremoring. At some point, it may seem to the PDer as if he is *always* trembling: when he is at rest, the tremor is milder. When stress or anxiety is added, the tremor increases in strength. But in general, the tremor no longer goes away.

As untreated PDers' conditions worsen, they usually become far more susceptible to this type of fear-induced tremor. And because their brains' hemispheres are increasingly misaligned, the PDer is increasingly likely to launch into amplified tremor from the least little addition of fear or stress.

Resting tremor as opposed to active tremor

When the tremor becomes somewhat constant, the PDer may no longer consider himself to have a resting tremor. However, inasmuch as the tremor may decrease slightly or even cease temporarily when a limb is in action, it is still a "resting" tremor from the perspective of medical terminology. In this case, the term "resting" tremor is used to differentiate from *active* tremor: a type of tremoring that occurs in some people, *not* PDers, in which the limbs do *not* tremor when a person is resting, but *do* tremor faintly when the limbs are overstretched, fully extended, or doing work.

Eating-based tremor

Yet another factor that can worsen the tremor for a short time is eating. When a PDer eats – or tries to eat – the energy level in his Stomach channel is automatically increased. The Stomach channel is the one that is damaged. The extra energy in the channel that comes from eating or trying to eat joins the backwards-flowing Qi in this channel. If the push of backwards-flowing Qi is what causes the hemispheric misalignment, an increase in this pattern can increase the amount of the misalignment, increasing the stress. This can lead to an increase in the amplitude of the tremor.

This increase in tremor can be stressful: the PDer's tremor can increase in amplitude yet again because of the *stress* of having an eating-based tremor. The combination of stress and the increased backwards Qi from eating, added to the constant, internal tremor, can cause a veritable explosion of external tremoring at meal times.

As an aside, the increased tremor, pain, or electrical shock that occurs when an acupuncturist ignores the traditional warnings and inserts an acupuncture needle into any channel that is flowing backwards is similar to the alarm that is triggered by trying to eat while the Stomach channel happens to be flowing backwards.

Healthy tremor

When a *healthy* person (a non-PDer) experiences a shift into predominantly sympathetic mode, he often tremors when the activating event is over. For example, a person exiting a scary movie may be surprised to find himself trembling a bit as he leaves the theater. He instinctively gives a little shudder, or as it commonly expressed, he "shakes it off." The intentional shake or shudder causes his brain and heart to shift back into parasympathetic mode. Likewise, a person may shiver violently for a few seconds after a swim in a cold mountain lake. Even before the swimmer gets to his warm towel, he gives himself a small shudder or shake, and his body relaxes

back to parasympathetic mode. A frightened child will tremble in his mother's arms until he feels safe again, at which point he will give a little shiver to reset his body's heart and brain orientations to those that are characteristic of parasympathetic mode. A person who was in shock often finds himself trembling until he physically "snaps" himself out of it with a little shake when he realizes that he is OK.¹

A steady shiver, a slight tremor, even a body-wide tremor, can be a healthy, perfectly normal event. It usually occurs after a person has had a strong startle, a bad chill, or moment of fear: anything that triggers a large sympathetic system response. The tremor stops when a person thinks to himself, "I'm safe." These thoughts may take the form of "It was only a movie; I'm OK," or "That was intense, but I'm OK now," but these are just variations on the idea "I'm safe again," or "I'm safe after all." The thought, "I'm safe," is the first step that a person needs to start the relaxation process so that he can shift back into parasympathetic mode.

Sadly, many PDer's have a very hard time feeling safe. After having been diagnosed with Parkinson's, many PDer's respond to the slightest manifestation of tremor, not by affirming that they are actually safe, but by invoking the thought, "Oh no! My Parkinson's is getting worse!" This affirmation of doom usually serves to amplify the push towards the sympathetic nervous system and *increase* the intensity of the tremor, if anything.

TREMOR INHIBITION

Temporary relief from tremor

Many PDer's work at achieving the almost-asleep stillness that temporarily quiets the fear-induced portion of the tremor. PDer's are often keen to describe to me in excessive detail their "discovery" of some type of massage or some meditation tape that miraculously makes the tremor "go away" for awhile. They do not realize that tremor *always* ebbs when a PDer slides towards the unconsciousness of sleep. These "discoveries," these short-term relaxation tricks, have *nothing* to do with healing and actual recovery. As soon as the meditation or deep breathing is over, the PDer is just as susceptible to tremor as he was before.²

Practicing being nearly asleep or numb does nothing to get rid of the underlying problem: the brain hemisphere misalignment and a predisposition to thinking that one is not inherently safe.

In fact, PDer's who work hard at techniques to *temporarily* calm themselves are missing the point: they need to practice affirming that they are *always* safe, that they have *never* really

¹ The old myth that shivering is a mechanism to warm the body after a chill, a myth that is still taught in medical schools today as "recognized fact," is incorrect. A person can "shiver in fear" even if the day is warm. For that matter, shivering from cold, unless a person recognizes the shiver as a warning and takes steps to warm himself, makes a person feel colder, not warmer. All of the various events that can provoke shiver and/or tremor relate to the phase that *follows* powerful activation and use of the sympathetic nervous system, prior to switching back into parasympathetic mode. The healthy shudder that occurs in response to the shiver may be a part of the switching mechanism.

² Very possibly, since the whole purpose of tremor is to convey a message to the consciousness to "Remember you are safe, after all: shake off the feeling that you are stuck in fear," the tremor ebbs when the consciousness ebbs during deep relaxation and pre-sleep: no point in sending a message if there's no one on the receiving end. It is curious to note that some PDer's *do* tremor while they are dreaming – when they are relatively more conscious than when they are in dreamless sleep.

been in danger, after all. This “always safe” or “safe after all” mindset is the one that healthy humans use to turn off the trembling that occurs after a sharp shock or fear.

Many PDer's who have gotten stuck in partial recovery have told me that, having been diagnosed with Parkinson's, they cannot avail themselves of this attitude. Because they have Parkinson's, they will never be “safe” again: a self-fulfilling prophesy.

Unless the PDer gets rid of the root cause of tremor, it doesn't matter how many times he *temporarily* stills the tremor by turning off his full awareness; the tremor, if any, will reappear every time that he has a return of full awareness combined with negativity.

Inhibiting the PD tremor by using adrenaline-based activity

Sometimes, the PDer's tremor can be inhibited by activity. By actively *using* adrenaline, by physically acting as if he is in a condition of genuine emergency and taking action, the PDer may be able to control the tremor in those body parts that are still under mental command.

After all, tremor in healthy people does not occur during an actual emergency, when the adrenaline is flowing. Tremor begins when the life-saving run for safety, the heart-pounding swim in the icy lake, or the intense psychodrama of the movie comes to an *end*.

Tremor is a gentle signal that the brain and heart are still in fear mode even though the emergency is over. Tremor is a signal to the body that it's time to shake off the fear and get back to relaxed, contented mode.

PDer's, because of the internal injury and the long-standing internal tremor, cannot get “back” to the relaxed mode: their basic situation is one of injury and brain misalignment – conditions that signal “Get to where it's safe and then relax, so that you can heal.”

Many PDer's have no idea how to relax. Many do not actually know what relaxation even feels like. For them, tremor is not a gentle reminder to go back to feeling safe or feeling relaxed. For them, tremor is a baffling manifestation of the fact that they are subconsciously still hiding an injury *and* that they are *not* safe. (The subconscious hiding of the injury can be what makes it so baffling.)

In these people, tremor – a manifestation of the dreaded Parkinson's – reminds them that they do have *cause* for *conscious* fear. An increase in fear increases the tremor: a PDer's tremor, if he notices it or merely *thinks* about it, often leads to greater tremor: tremor over a larger area or tremor of greater amplitude.

Inhibiting the tremor by performing dopamine-based activity

Some PDer's can slide, now and then, into normal, tremor-free movement. This occurs when the PDer is doing an activity that he has decided is safe. What constitutes “safe” is completely random, and varies from one PDer to another. We've seen “safe” activities that range from singing to doing the crossword puzzle.

When this happens, the PDer is using dopamine. The internal tremor, though still present, does not manifest as an external tremor during these times. The dopamine-based feeling of safety is able to override, and possibly even sedate somewhat, the internal tremor. However, as soon as the PDer returns to his wariness, the tremor returns.

As an aside, this temporary cessation of tremor and movement inhibition symptoms during safe activities should have gone a long way to inhibiting the spread of the grossly inaccurate and inadequate “dead dopamine cell” theory, but it has not. Almost seems as if the battle cry of grant-dependent researchers is: “When the theory doesn't fit the facts, ignore the facts!”

Nevertheless, if a PDer's tremor goes away when he does laundry or when he does some other activity that gives him quiet pleasure, the logical PDer should take this as proof that his *mindset*, not "dead dopamine cells," is contributing to his tremor situation. Realizing that the tremor calms down temporarily – even during full wakefulness – during activities or moments that the PDer considers "safe," should help the PDer realize the importance of affirming that he is *ever* safe.

During recovery, if a PDer was healing or healed from his foot injury *and* was able to perceive the physical sensations of expansion in the chest that occur when one feels truly safe – no matter *what* his life circumstances happen to be – *that* was when the brain shift was most likely to occur. The brain shift happened when the PDer was able to *feel* (not think, but *feel*) the way a person feels when he is safe. The PDer who could *feel* safe – even if he thought he was about to *die* – could surrender enough to allow his brain to drift back to the healthy position. When this happened, the tremor was gone for good.

Fear of the brain shift

As you have no doubt already guessed, some PDers have told me that they dare not enter into a recovery program because they might experience a brain shift while driving. And by now you can probably provide the same reply that I gave them: brain shifts have only occurred when the PDer felt deeply peaceful. These events have not occurred during times when alertness or adrenaline was called for.

PDers experienced their brain shifting events while resting, daydreaming, or while half-awake. Others concluded that a head shift must have occurred during sleep because, upon awakening, the head felt different or else the internal tremor was gone or they felt a new sensation inside: profound stillness.

No one, in our experience, underwent a head shift while driving, while bustling about, or while highly alert.

CLOSING THIS CHAPTER

Healthy people respond to a post-sympathetic mode tremor or shivering by remembering that they are no longer at risk. They reinstitute the safe feeling in the chest, give themselves a little shake or *frisson*, and the tremor goes away.

Most of our PD patients responded to tremor or even the thought of tremor by worrying about what the tremor might signify, or worrying about what other people might think if they saw the tremor. These responses caused amplification of the tremor.

The most paradoxical aspect of the recovery brain shifts was that a PDer had to feel truly safe in order to experience them. Safe. Not calm, not sleepy, not restful, but safe.

After brain shifts occurred, PDers felt profoundly peaceful: safe. Especially after the side-to-side brain shift, many PDers noticed that no stressor was able to induce a tremor again even if they found themselves in a stressful situation.

In other words, a brain shift allowed PDers to feel deeply at peace – you might say it allowed them to feel utterly safe. But in order to *initiate* the brain shift...they had to feel safe.

The answer to the paradox appeared to be *surrender*. The word "surrender" came up often with fully recovered PDers. When they spoke about surrendering, they meant it in the sense of surrendering from their posture of perpetual wariness, cleverness, defensiveness, or

heightened alertness. The “surrender” was simply the admission that, no matter what, *even if they did nothing* in self-defense or self-maintenance, they were actually safe. And when they decided that they were actually safe “after all,” and let themselves *feel* safe (not think, but *feel*), their bodies responded with relaxation and a brain shift that brought about an end to their tremors – and an even fuller feeling of safety.

And yet, how many PDers have responded with hostility to my suggestion of surrender! I can picture several of them: trembling violently, hunched over, rigid, with an expressionless face and barely able to get the words, out, proudly exclaiming, “It’s my refusal to surrender that’s got me where I am today!”

They have no idea why I smile and say, “Yes.”



CHAPTER TWENTY-FOUR

RECOVERY SYMPTOMS: CHANGES IN PERSONALITY AND ATTITUDE

Introduction

The previous chapters focused on those recovery changes that were related to muscles, nerves, sinews, and even brain hemispheres. Those were physiological changes, changes that had a physical basis.

This chapter describes a few of the recovery symptoms that were more mental than physical: mood and personality changes. Some of these changes, in turn, led to seemingly physical changes: improved attitude and mood often caused a clear improvement in movement initiation, speed and fluidity of movement, and tremor.

In many PDers, the physical changes were harbingers of mental and emotional changes. In other PDers, the mental and emotional changes seemed to come first, leading the way for the physical changes. It was often hard to tell which came first. And truly, it didn't matter. What did matter was that the patients who became stuck in partial recovery were the ones who experienced many of the physical changes but few, if any, of the emotional changes.

In order for PDers who were veering towards partial recovery to experience the floods of new perceptions or attitudes that led to ease of movement and cessation of tremor, some of them had to learn how to access their own sensory experiences. Others had to work at shutting down their inner "Voice of Doom."

Still others were outraged at our suggestions that they "surrender" to emotional vulnerability or take charge of their own thoughts in order to vanquish their chronic wariness or pessimism. Despite having experienced many physical symptoms of recovery and even periods during which they moved perfectly normally until they remembered that they couldn't, many of these people decided that Parkinson's was incurable and dropped out of the program.

For those who were able to automatically experience the recovery symptoms that altered personality and attitude, and for those who worked hard at it and finally attained "feeling safe," these were some of the most satisfying recovery symptoms of all.

Crying

At some point after the foot injury began to heal, recovering PDers often found themselves bursting into tears at the least little thing. Various PDers told me, prior to recovery, that they rarely cried, or that they *never* cried, or even that they *couldn't* cry. During recovery, for a few weeks or months, they often found themselves crying at almost anything.

Typical reports included, "I'm turning into a sap! I saw a little child walking a puppy on a leash, and it was so cute, I burst into tears," and "I caught myself crying at *Oprah*, for God's sake," and "I read the headlines on the newspaper and I was so touched, I started crying!"

Usually, the onset of easy tears was a wonderful feeling, accompanied by a feeling of openness in the heart and an end of a long-time fear that tears would lead to being condemned as a sap, a weakling, or stupid.

Then again, the onset of tears could be very painful.

One PDer who did not recall ever crying since she was six years old started crying one day after a session in which we'd worked on her imagining that her physical body and her "imaginary body, the body that contained her heart" bridged the several-foot gap that usually separated them and merged into one person.

She started crying in my office, saying, "Why are people so mean to one another?...gee. I'm crying." And then she started crying harder. She was so violently wracked with sobs that I suggested she not return to work, that she go straight home and call in sick for the rest of the day.

She assured me that she wouldn't have any problem at work: she had always been able to stay in control of her emotions.

She called me the next day. She had returned to work, started sobbing hysterically at her desk, and actually fell on the floor and was unable to stand up. A co-worker drove her home. She stayed home from work the next day and cried. She was unable to staunch the tears, but more importantly, she was unable to stop the flood of emotions that surged in her breast. She was feeling emotions for the first time since she was six years old. She felt emotionally drained by all the feelings and the crying, but she figured that it was all to the good. Then the worst happened.

Her best friends, a couple that she had introduced to each other, died in a car crash just three days after she'd called me from home. Her friends had been on their way back from Disneyland. Their infant daughter also died. The eight-year old child was injured, but survived.

This PDer, who had not cried *or felt any emotion* since she was six years old, was hit with the full force of real-time emotional loss. She told me, the next week, that when she got the news of her friends' deaths, she cried so hard that her chest hurt, her eyes hurt, her face hurt. Her arms hurt, her skin hurt. At some point, she was in so much pain that she couldn't tell if she was crying because she was in such physical pain or if she was in such physical pain because she was crying. She feared that she might go crazy from the emotional pain, the physical pain, and the crying.

A week later, she told me that, for several days, she questioned whether or not she had been better off back when she was unable to cry, back when she had attended family deathbeds and funerals and experienced no feelings *whatsoever*.

I asked her if she now regretted the changes that she'd been going through, if she wished we hadn't opened that door.

I will never forget her answer.

She looked me right in the eye and said, "You saved my life. I was dying and you brought me back."

She had no regrets.

Nearly all PDers *enjoyed* the sensations of increased feelings and tears. Very often the tears were tears of joy, of connectedness with others. Even in the above case, she was grateful to be able to feel and express the anguish and pain that she was feeling.

The ability to cry is very important. I cannot think of any person who has recovered from Parkinson's who retained his *inability* to cry. Curiously, some of those who recovered easily had actually worked on learning to cry, *long* before they were ever diagnosed with Parkinson's. I highly suspect that their self-taught ability to experience their own heart feelings was one of the main reasons that they recovered so easily.

Learning to cry in high school

Lynne, whose foot got slammed in a car door when she was five, learned to cry when she was seventeen. It happened after one of her teachers referred her to the school psychologist for “the usual senior-year counseling session.” (Lynne only realized decades later that her ferociously stoic demeanor had probably prompted her favorite teacher to set up the unusual session. When Lynne’s own children were graduating from high school, Lynne suddenly realized that none of her old high-school classmates had ever been sent to a “usual senior-year counseling session.”)

During the session, the counselor asked Lynne a few pointed questions about her home life. He remarked on how Lynne seemed to freeze up when asked about her mother. Lynne coolly replied that, “All children should love their mothers. My mother always reminds me that even the worst criminals love their mothers.” The counselor countered, “Not all mothers deserved to be loved. Some mothers chop up their children and flush them down the toilet.”

Lynne was shocked and relieved by the thought that some mothers didn’t merit blind devotion. The counselor then asked Lynne if she ever cried. Lynne replied, “No one likes a person who cries.” As a very young child, Lynne had learned that crying was one of the behaviors that made her mother insane with rage, and always led to a brutal beating with a leather strap. It wasn’t until several years after recovering from Parkinson’s that Lynne realized that her mother had actually manifested orgasmic breathing and body movements while using the strap on Lynne. At any rate, Lynne had learned very quickly never to cry.

The counselor contradicted her, “Everyone cries. Crying is normal. Everyone knows that tears just mean sadness. Maybe your mother doesn’t understand about crying, but healthy people do. If you walked all over school crying, not one person would be angry with you or dislike you; people would most likely just say to themselves ‘she must be sad about something’ and continue on their way.”

Lynne was astonished at this new thought, and told the counselor, “I’ve got to get back to class. But I’ll do an experiment. I will cry and see if anyone gets mad at me.”

She started crying on her way back to class. She had not cried since she was five years old, so it took a while to get started; she pretended that she was an actress who could cry on command, and that got a few tears rolling. Once she started, she couldn’t stop. She did not take her usual seat at the front of the class that day. She sat at the back and cried. To her amazement, no one, not even the teacher, got mad at her. For that matter, she was given a wide berth. Other than that, no one seemed to even notice her. (This had been back in the pre-hug 1960s. If she did the same experiment today, probably many people would have come forth with hugs.) Certainly, no one made fun of her or scorned her. She enjoyed the sensation so much that she sat at the back of the room in her next class, as well, and cried for another forty minutes.

Shortly after that, she left home. From then on, she had made a point of letting herself cry, sing, dance, and emote whenever she felt an emotion welling up in her. Prior to her diagnosis with Parkinson’s disease, she had noticed that it had become increasingly difficult for her to notice her own feelings. She found her self with an increasingly flat affect and mood – but unlike many of my PD patients, she had been very aware of this decline. She fought it vigorously by forcing herself to sing songs that always made her feel better, and which, in turn, temporarily improved her movements.

She recovered from Parkinson’s disease very quickly; most of her symptoms were gone in a few months.

Several of our PD patients described similar stories of learning or teaching themselves how to cry, feel vulnerable, or focus on the feelings their own bodies. Those PDers who had worked on these skills tended to recover quickly. Oppositely, those PDers who were not willing to learn to cry or willing to feel pain were almost certain to become stuck in partial recovery. Again, no PDer who has fully recovered retained his inability to cry.

Punctuality

One distinct symptom that accompanied the change *away* from adrenaline and fear was a decrease in punctuality. I was always thrilled when a previously punctual PDer called me on the phone to say something like, “Ha ha! I guess you figured out that I’m not at my appointment. I’m at the beach watching the birds and I decided not to come to your office. I’ve just realized that I’ve never really sat and watched birds before. It’s great! See next week.”

I loved it when a PDer who had previously been chronically punctual started to recover – and then showed up fifteen minutes late for his next appointment...and laughed it off! It was always a sign of emotional recovery.¹

Panic attacks

Many people experienced a panic attacks after succeeding in letting go of their anxiety or negativity. These attacks occurred in situations with highly specific parameters: 1) the situation was always supremely *non-important*; 2) a decision had to be made with regard to a *new situation*; 3) because the situation was new, *no precedent* could be applied.

The panic attack was usually full-blown, complete with pounding heart and sheer terror with no way out. A common description was “my mind seemed to be going down a black hole.”

After interviewing the recoverers who had panic attacks – and they only had one apiece, I noticed the following generalities.

The attacks occurred after a person started feeling very good, very comfortable. He could feel himself being more relaxed and less likely to use adrenaline. He may have even noticed that his heart was more calm and that he was sleeping better than he used to.

Then, along came a situation in which he had to make an innocuous decision: a decision that he had *never* had to make before. Some examples of decisions that prompted panic attacks are: Where to put the cat dish for the new kitty’s food? Where to set the knick-knacks that sit on the bookcase when the carpet man moves the bookcase to take up the old carpet? “How do I install a brand new computer game program?” (This was back in the days when every new computer program had its own unique set of installation instructions.)

In every case of panic attack, the person was confronting a *new* situation that was not actually very important. When he calmly tried to think of what to do in this situation, the brain presented an utterly blank screen: no thoughts appeared. He then wondered what was the matter with his brain.

In the past, these PDers had been adrenaline-driven, accustomed to making lightening fast decisions. Now, their brains had gone literally blank. As they tried harder to find some mental thread to grasp, in an attempt to figure out how to do the new task, they realized that there were no mental threads. The task itself became less important. The mental focus was redirected

¹ I had *one* PD patient who was not compulsively on time. She was consistently, considerably late for everything. Every PDer is unique. There are exceptions to every generality. She was also a musician, and one of the PDers who recovered *very* rapidly.

to the realization that there were NO mental threads. It felt as if all thought processes have been turned off, as if the brain itself was missing.

What was probably happening, based on detailed descriptions that we've heard, is that the person was trying – for the first time in decades or maybe for the first time in his life – to make a decision using dopamine instead of adrenaline.

A recovering PDer may have no remembered experience of making a decision while using dopamine. When he tried to make a decision while being dopamine dominant, the mental “thought screen” showed up blank. This led to the next thought, “My brain is missing!” and from there, into a black hole of terror and a full blown panic attack. At some point, the PDer’s panic forced him to drop back into sympathetic mode – a mental condition that he knew very, very well. Once he slipped into fear mode, he was able to snap out of his panic.

Happily, these attacks only happened once. After that, the recoverer seemed to know how to make decisions without going into sympathetic mode.

We have never known of anyone who couldn't figure out where to set the new knick-knacks after having had his one panic attack.

These panic attacks were very real. The woman who slid into a black hellhole of panic while trying to decide where to put the kitty dish was a NASA researcher, a brilliant woman. She found herself standing in the center of the living room screaming, “Help me! Help me!” at full volume. She said that, even as she was doing it, a part of her brain was thinking, “This must be a panic attack! Cool. I must be recovering!” But even so, the sense of impending doom, helplessness, panic, of needing someone to take over and take care of her, was physiologically *real*. The panic did not have to do with the kitty dish, per se. The panic began when her brain registered “Empty” when she tried to think of how to think about where to put the new dish.

One previously intrepid, world-traveling PDer had a panic attack when his wife proposed that they try the new restaurant in town. He went into a full-blown panic and was soon screaming for help as his wife stood by in amazement. The reason? He didn't know what they would do after they got into the parking lot because he didn't know what they would need to do to find the front door of the restaurant. And when he tried to *think* about how a person might go about finding the front door on a building he hadn't been to before, he couldn't figure out how to think. It was the inability to think, and the feeling that his brain was literally empty, that triggered the panic.

As an aside, our patients with Parkinson's seem to have relied on the emergency form of decision making for most of their lives. This is why they often came across as stronger, smarter, and faster than their peers. They might not have been stronger, smarter and faster than their playmates and peers if they had all been on the same neurological footing – using the parasympathetic mode instead of sympathetic. We are pretty certain of this because, after recovering, PDers were often amazed to find themselves becoming more average in terms of strength, quickness of mind, and speed. They did not become stupid or sloth-like. However, they are only *pleasantly* above average, not super-duper, not driven – and they were OK with it.

Because of their own chronic use of adrenaline and their amped up, emergency-induced thought and physical processes, many PDers had actively resented their “slow” fellow students and coworkers, had dismissed them as “underachievers.” Even since grade school, the mere presence of these “slower and stupider” people had been a real irritant for many of our PD

patients, who, deep down inside, had felt that life was an ongoing emergency, a state of perpetual risk. (More on this subject in appendix xxx, The adrenaline-dopamine relationship.)

Getting back to panic attacks: when the recovering PDer needed to make an innocuous decision, one that was simply too mundane to activate his sympathetic nervous system, he found he had no way to access the decision-making part of his brain and be calm at the same time. He actually got a blank slate when he tried to make a simple decision while under the influence of dopamine. It was always the blank slate that scared him, not the implications of the decision.

As you have correctly guessed, a few PDers have told me that they shouldn't enter into a recovery program because they might have a panic attack while driving the car. You know exactly what my reply was: the panic attacks occur while doing something perfectly dull in a very safe setting. No one in our experience has had a panic attack while in a risk-laden situation.

Then again, one recovering PDer did have a panic attack while driving. He had taken the weekend off and had flown back east to visit his daughter at college. The visit was purely a lark. It was the first time he'd taken off just for fun in a long time. As he was enjoying himself driving along the freeway, he missed the exit. He took the next exit instead and then realized he didn't know what to do next. He couldn't even think of what to do next, so he pulled over and stopped the car. Then, in this perfectly safe, non-emergency situation, in which, on some level, he was actually enjoying himself, he tried to think about what he should do next. He couldn't think. Within seconds, he was in a complete panic because he couldn't think. His heart was pounding, he was sure he was going to die. After what seemed like several minutes but what was probably several seconds, his brain kicked in. He thought of something to do: he called his wife, long-distance. She agreed that he'd just had a panic attack, and what did he think he should do. He said that he needed to pull a U-turn, get back on the freeway, and go back the way he'd come. Duh. He knew what to do.

But he had been thrown into a panic by the fear that had tackled him when his brain had failed to respond in the usual manner. He did get to where he was going, and he never had another panic attack.

The interesting thing about this particular style of panic attack is this: after the person truly begins to panic, the sympathetic nervous system does kick in. He is then able to think in the manner that he has always thought in the past. And so the panic attack ends.

In these and in other cases, it has seemed as if the ex-PDer, in the joyous throes of steady dopamine release, has been reluctant to slide back into using the old familiar sympathetic mode. But decisions can only be made in one of two ways: by using the mind while in predominantly sympathetic mode, or by feeling the preference of the heart while in predominantly parasympathetic mode. The sympathetic system is guided by mind. The parasympathetic is guided by physical heart sensations.

The first time a person recovering from Parkinson's tries to make an unimportant decision using his *mind* (as per his lifetime habit) while he is in predominantly *parasympathetic* mode, he can't come up with a solution. In parasympathetic mode, the heart instructs the mind. If one tries to find a solution to a problem using only the mind pathways, while staying in predominantly parasympathetic mode, he will not get an answer: the mind may present a blank.

In recovering PDer's, the mind system hasn't yet been trained to work as a subordinate during parasympathetic, heart-led decision making.¹ Hence, the panic attack. Happily, our recovered PDer's instinctively learned, after one panic attack, how to think and make decisions while in parasympathetic mode.

Guilt or boredom from lack of tremoring

Another emotional symptom of recovery was guilt. I have already written about the PDer who never tremored again after a side-to-side- head shift and who felt terribly guilty when, shortly after, her mother was in the emergency room at hospital. For years, her hand had tremored during the most mild of crises. As the emergency room clock ticked away and she failed to tremor, she thought to herself, "I must not care about my mother." I've tremored from all kinds of stupid, unimportant things, and now my own mother is in danger and I'm not even tremoring."

She told me about her shame the next time I saw her. I had to assure her that tremoring in a crisis is not necessarily normal.

Some PDer's were ashamed of themselves when, upon recovering, they realized the extent to which their Parkinson's symptoms had been the result of their own mental and emotional blockages. I recall one PDer repeatedly slamming his open hand onto his forehead while saying, "I've been doing this to myself! What an idiot I've been. I've done all this to myself!"

Another type of guilt that sometimes descended on a recovering PDer was the realization that, for a large part of his life, he had increasingly been an unpleasant, demanding perfectionist, know it all, or whatever personality form his fear and adrenaline had taken.

As a healthy level of humility began to take root and thrive, the temptation existed for the PDer to indulge in guilt for having been such a fear-driven, pride-driven (or whatever) in the past. My advice was that indulging in guilt and shame is a variation on pride. Be humble enough to know that *everyone* makes errors. Forgive yourself and get on with your life. No doubt you were nearly always doing what you thought was best.

Another post-tremor weirdness was the feeling of emptiness. As one person expressed it, "I miss the tremor. I know that's weird, but I always felt some sort of internal pressure to look alert. As long as I was tremoring inside, I felt like I was always doing something. Now that it's gone and I can sit motionless, I feel as if I'm not *doing* anything when I'm just sitting around. And I sort of miss that old intensity that wouldn't let me sit still for very long. I'm going to have to find a new motivator."

Loss of self-identity

As mentioned in an earlier chapter xxx, many recovering PDer's have found themselves asking the question that might be more characteristic of an adolescent: "Who am I?"

This may be because, in part, the PDer never really experienced a calm period during adolescence in which he was able to dwell on that question.

¹ This reminds me of a story about Karl Jung, the famous philosopher/psychologist/ explorer. Jung asked Ochwaiy Bianco, chief of the Pueblo Indians, for his opinion of the white man. The chief said white men must be crazy because they think with their heads, and it is well known that only crazy people do that. Jung asked how the Indians thought. The chief replied that, naturally, they thought with their hearts. (From *The Sun*; "Sunbeams (Letters to the editor);" Laurens van der Post; Sept 2007; p. 48)

Or, the PDer may have known exactly who he was so long as he was “on top of his game” and running on adrenaline, but he might have had no idea who he was when he found himself able to relax deeply, feel music, and not caring about the order in which someone else stacked the dishes into the dishwasher.

Who was he when he suddenly realized that most of what he’d been worrying about for years didn’t really matter? Who was he if he was starting to live in harmony with his own feelings, his own heart?

Many PDers valued themselves in terms of what they were able to accomplish, and because they were stronger, faster, or smarter than most everyone else. What they needed to learn was that no one is loved for his accomplishments, and no one is loved because he happens to be strong, fast, clever, or “always right.”

People are loved to the degree that they allow their hearts to resonate. A person with Parkinson’s *may* have a heart that can resonate with others, send love to others. It may resonate well with dogs or small children.

But what PDers also often have is an inability to feel the resonance between his own heart and his own body, or between his own heart and the love that others are directing towards him.

Because of this lack of resonance and ever-increasing reliance on adrenaline, he may have been steadily building protective walls around his own heart until he got to the point that he no longer knew who he really was.

When, during recovery from Parkinson’s, those walls begin to crumble, PDers were often surprised to learn that they were not who or what they thought they were. But all the recoverers were certain that they loved their new self far more than they’d been able to love their old, PDish self.

Prior to recovery, very possibly the hardest thing for many PDers to understand was this: in the past, they were loved, not because of who they were and what they did, but *in spite* of it.

Overflowing with gratitude

A common symptom after recovery is a feeling of contentment and gratitude.

Gratitude is a feeling that *cannot* be easily accessed while locked into sympathetic mode. The gratitude that a person felt when he realized that he was safe and that he had always been taken care of – whether he’d known it or not – was very often the trigger for the feeling of safety that set in motion a brain shift or the first obvious surge of recovery-type (not activity-dependent) dopamine.

Many fully recovered PDers were certain that immersion in “gratitude for having always been safe” was the thing that initiated their new lightness of movement that was characteristic of lasting recovery and dopamine-based thinking.

Many PDers who got stuck in partial recovery assumed that this was impossible: *if, and only if*, they could move easily again or they could have concrete proof that they had dopamine flowing again, would they then be able to feel gratitude.

Many have told me that they have nothing to be grateful *for* because they have Parkinson’s. If and when they recover, *then* they will have plenty to be grateful for.

This sulky stance was common in many of the people who became stuck in partial recovery.

I often tried to explain that, if a person needs a “cause” for gratitude, or if he thinks that having an illness makes gratitude impossible, then he doesn’t understand what gratitude really is. Gratitude is a *feeling*. Gratitude is not a thought process. Gratitude is *not* a logical payment that one makes after having received a favor: that’s a payment – that’s not gratitude.

When a person gazes at a stupendous sunrise over a mountain lake, his heart resonates with the vibrations of color and shape in that sunrise. His chest fills with an indescribable emotion. “Gratitude” is an apt name for that indescribable emotion. The word “gratitude” is related to the word “grace.” Grace is defined in my oldest dictionary as “unearned blessings.” The glorious feelings in the heart that can occur from resonance with a sunrise have not been *earned*. Those feelings simply *are*. We can call those feelings “love.” We can also call them “gratitude.”

Learning about gratitude

The gratitude exercise is one of the mind-retraining techniques that we offer. One of our long-distance patients had chosen to practice the gratitude exercise every night. He saw Chris again after six months and reported that, every evening, he thought of five things to feel grateful for. Chris asked him *how* this made him feel.

“Feel?”

“Yes. When you feel grateful, how do you feel?”

“Feel?”

“Right. How would you describe the feeling of gratitude?”

“Feeling? I just make a list of five things, say the five things, and go to bed. What am I supposed to be *feeling*?”

Chris stopped the duologue, and slowly led the PDer through the gratitude exercise. The very wealthy PDer picked, for the exercise, one of the things that he’d been grateful for every evening: his toothbrush. Chris asked him to just imagine his toothbrush for several minutes. After several minutes had passed, Chris asked him to mentally give wordless thanks to the toothbrush, and to do so for several minutes. After about three minutes had passed, the PDer sighed deeply and said something like, “Oh.” He had felt his heart open up while thanking the toothbrush.

Gratitude has nothing to do with logic. Gratitude is a feeling. And the feeling of gratitude has been one of the most direct keys for unlocking the brain’s trove of dopamine.

“No one can understand”

During and after recovery, a PDer who is overflowing with gratitude may find that there is “no one can understand what I’ve been through.”

Once, when we asked a recently recovered PDer to talk with a PDer who was still struggling with negativity, she spoke with him for fewer than two minutes before standing up and saying to us, “There’s nothing I can say to him. He’s not ready to understand. I know that I wouldn’t have been, either. I had to learn for myself.”

I’m just mentioning it here in this chapter on mental and emotional symptoms of recovery so that the recovering PDer who reads this will know that he is not the only one who feels that there is no way to describe what recovery is like, that “no one can understand what I’ve been through.”

Why talk about it?

This actually brings us to the subject of why so few people who have recovered from Parkinson's have stepped forward. In the first place, in our limited experience, neurologists have been pretty adamant that anyone who recovers from Parkinson's never really had Parkinson's. He was either misdiagnosed or else he was crazy. This makes it hard for a recovered PDer to flaunt his recovery.

But secondly, and perhaps more importantly, people who recover from Parkinson's often say that recovery is not the "victory" that they expected. They can once again move easily because they have become humble, not because they have returned to glory. They have learned to love themselves for the sheer joy of being instead of for how strong or steady they used to think they were. They tend to be sheepish about the way they behaved and thought, prior to recovery. There is no way that they would ever want to be that person again. People who recover often speak of having lived two different lives, with two personalities, all in one lifetime. PDers are smart enough to know that this is not the sort of talk that makes a lot of sense, particularly to anyone who is still stuck in a Parkinson's Personality.

Most ex-PDers admit that there is *nothing* that they can say to a person who still has Parkinson's that would help that PDer recover.

As one PDer put it, "I know that, prior to recovery, I would have dismissed all of the things that have turned out to be most important because I would not have understood what you were saying.

"If you had talked to me about gratitude, I would have assured you that I knew what gratitude was. But I didn't. If you had talked to me about heart, I would have assured you that I knew what love and heart are. But I didn't. And there would have been nothing that you could have said that would have made me understand.

"I had to lay down my weapons of self-protection. I had to surrender to the good that exists in the universe and in me. And surrender was the most hated word in my vocabulary. There was nothing you could have *said* that I could have understood."¹

And this "Nothing you could have said" can actually be one of the challenges of recovering from Parkinson's: There are no *words* for the feeling that initiates a surge of activity in the substantia and the release of dopamine. The problem is that, prior to recovery, the PDer may increasingly have lived in a world of *words*.

During recovery, he will return to a world in which the heart expansions of joy and silent instructions from the heart will resume their proper place. *Words* will once again be used correctly: as servants that express the feelings of the reigning heart. Even the world's greatest poets work hard to create word portraits of this world of feelings. Most recovering PDers don't even come close to having the skill set that would allow them to use *words* to explain their return to life. But the longer they have Parkinson's, the more likely it is that they can *only* interface with themselves and their world via words.

¹ Also, those who have recovered *easily* may not have much to offer on the subject. They were never emotionally blocked. They may not understand the heart denial that is characteristic of people stuck in partial recovery. If a person recovers easily, he probably was not highly dissociated. He may not even go through any period of *learning* to reconnect his heart and mind: the reconnection may be automatic. If an "easy recoverer" is asked what advice he has for someone stuck in partial recovery, he may just say, "Huh? Fix the foot! It's easy." The "easy" or "fast" recoverer may not understand what the big deal is all about. Fortunately for this project, several of the pioneers were easy recoverers. If I'd only had partially-recovered PDers to work with in the earliest days of our research, I would soon have abandoned the Little Project.

No fear of criticism

Many recovering PDers noticed a dropping away of their defensiveness.

Of course, not all PDers had been afraid of making mistakes or being laughed at, but many had been. Their symptoms ranged from a protective emotional flatness to abject fear of “what others might think” to the point that they lived every moment as if an invisible critic was offering running commentary. When the inner critic died away, the relief was enormous.

Return of the heart

During the visualization exercises that accompanied the foot treatments, many PDers were surprised to realize that they could not visualize at all, let alone imagine themselves having a radiantly healthy heart in their chest. During recovery, they “got their heart back.” They could imagine themselves having a heart, and they could *feel* the expansion feelings and/or hesitations of the heart in response to their thoughts and activities. They were able to sit quietly and *feel* pleasure in watching the sunset as opposed to *thinking* about the sunset. They were able to enjoy themselves while just sitting on the beach or while just watching trees move in the wind. They were able to stay calm while watching a child attempt some new skill even if the child wasn’t doing it the most efficient way. They found themselves enjoying the experience of being wrong and admitting it or laughing at themselves and having others laugh with them.

These enormous changes suggested a reversal of some of the core psychological processes that are at work in Parkinson’s disease: an end of dissociation and a return to heart sensitivity.

Cycles of change

I will close these chapters on recovery symptoms with one of our most bizarre observations. People with idiopathic Parkinson’s disease have a twenty-three day cycle of movement ability, during which movement goes from good, to bad, and back to good again. A PDer will go from a very good day or days, during which he has a great attitude and feels at the top of his game, and slowly slide, over the next eleven days, down into a pit in which he may be certain that he has never been so rigid (or negative, or tremory, or whatever his current, worst problem is). Then gradually, over another eleven days, he will cruise back up to the top again. The complete cycle lasts twenty-three days. And then it starts again.

The most uncanny thing about this cycle was that all PDers were on the same twenty-three day cycle. All PDers in my experience had their best days on the same days, and eleven or twelve days later, they all had their worst days, and then, by day twenty-three, they all had good days again.

The incredible thing was that this cycle did not just happen to my patients in my office, in my hometown. It happened to all the PDers around the world that had ever contacted me.

Before I realized that there was a cycle, I was often remarking to my family, “Everyone is much worse today, I don’t know what I’m doing wrong. Maybe I’m a fool for trying.” And then a week and a half later, I would be rapturous with glee because “*everyone* is doing so much better.”

My son finally pointed out that I’d been doing this up and down cycle for over a year, and that it was impossible that “everyone” was doing poorly or that “everyone” was doing well. Surely I was exaggerating.

So I went through my charts. I went through the emails that had been sent to me from people I knew *and* from people that I had never met. Sure enough, there was a consistent, twenty-three day cycle in PDers from around the world, even if I’d never met with or emailed

with them before. In fact, I suddenly saw that I tended to get the most emails at the apex and nadir of the cycles.

This was unbiased reporting: the emailers who had never met any of us on the team were *not* being influenced by me or by anyone in the project.

The other thing that was quite strange was that, since starting this project, there were a few times that the low in the cycle was extremely low or the high was extremely high.

I recall that all my patients experienced a *severe* low in the first week of August of 1998. Another one occurred towards the end of January in 2000. I have not tracked enough of these extremely powerful lows and highs to detect a pattern. I did not share this information with PDers at first, because I feared “contamination” of my research data. But once I had several years of data, I did present this information about the twenty-three day cycle to patients so that they could keep their chins up when they found themselves having a rough week or so.¹

The influence of whatever it is that drives this cycle ceased when a person recovered from Parkinson’s disease. The *physical* symptoms of Parkinson’s and the emotional symptoms of Parkinson’s (if any), the negativity, wariness, self-pity and/or fear or shame of self-pity, and anxiety, were influenced by the twenty-three day cycle.

However, the physical and emotional symptoms of recovery were not influenced by the twenty-three day cycle. The pain and tingling in injuries that were healing, the ability to taste and smell, to sleep long and deep, to cry, the changes in self-awareness and the new found ability to feel one’s own heart’s responses were *not* influenced by the twenty-three day cycle.

SUMMARY

The people who recovered most easily were those who had never lost their ability to cry or guide themselves using the physical sensations in the chest, or those who had lost these abilities at some point in their lives and had worked to resume them in the years long before the Parkinson’s was diagnosed.

Those people who were able to let go of their selectively rigid emotional habits enough to cry, to stop caring about what others, including the Internal Critic or the Voice of Doom, might think, and to no longer fear the potential emotional pain that comes when the heart is vulnerable were able to recovery *somewhat* easily, even if they were stunned by the flood of long-absent feelings that they experienced when their injuries healed.

Those who were unable to even understand what I meant by the word “feeling of expansion in the chest,” those who remained convinced that the point of life is to avoid physical and/or emotional pain, and those who remained constantly wary of how they are being judged by others usually became stuck, for some period of time, in partial recovery. Those who became stuck in partial recovery could nevertheless recover – but they had to work at overcoming their mental and emotional inhibitions. Techniques for helping change these patterns are included in the section on treatment techniques.



¹ Several friends of the project have tried to research the probable cause of this worldwide cycle. The only astronomical event with a twenty-three day cycle that might possibly be driving the pattern is a star in our galaxy that emits a strong radio wave in a twenty-three day cycle. This is not a biorhythm pattern. Biorhythms begin on the day a person is born. Therefore, there are 365 potential starting dates for the biorhythm cycles. But all PDers seem to be stuck in the same cycle. It may be that, when they shut down their heart, they fall out of their native biorhythm and into a sort of default cycle that can be influenced by strong atmospheric phenomena.

PART III

RELATED RESEARCH

INCLUDING

THE DISCOVERIES AND A CHRONOLOGY OF THE RECOVERY PROJECT

“The mind can make a heaven out of hell or a hell out of heaven.”

- John Milton

CHAPTER TWENTY-FIVE

PLACEBOS AND PARKINSON'S DISEASE

The placebo effect

A placebo is an inert or neutral substance or event that makes a person feel better. The western understanding of this phenomenon is that a placebo works via the power of suggestion.¹

The placebo effect has been researched in many highly respected, rigorous, double-blind, scientifically conducted studies.

Placebos do seem to work for some types of illnesses, and do not work in others. The most recent research suggests that the determining factor in whether or not a placebo will work in a particular illness is this: whether or not dopamine plays a role in the illness.² If, due to a placebo treatment, a person *anticipates* that he will feel better, dopamine is released. The release of dopamine is the trigger that causes beneficial changes in the person's condition.

Placebos work very well in people with Parkinson's disease.

Negative-placebo effect

Oppositely, fear or the expectation of trouble can behave like a “negative placebo.” In the case of a negative placebo, the expectation of *problems* can set in motion actual physiological changes, such as increasing the body's tilt towards the sympathetic system (and adrenaline) and the simultaneous tilt *away* from the parasympathetic (and dopamine). In other words, negative placebos can inhibit dopamine release. This sympathetic nervous response, if severe enough, can even set in motion instantaneous development of Parkinson's-like symptoms, even in people who do not have idiopathic Parkinson's disease.³

¹ The word “placebo” comes from Latin and means “I shall please.” The word placebo these days usually refers to the medical use of dummy (sugar) pills or pretend treatments that make the person feel better even though the pills or treatments have no (known) healing mechanism. Placebos are sometimes used in medical trials when testing new drugs to determine if the benefit of the drug is coming from a physiological interaction or is merely coming from psychological suggestion.

² Illnesses such as insomnia, pain disorders, allergies, depression, digestive disorders, and even susceptibility to illness are immediately affected by dopamine levels. Illnesses such as broken bones and cancer are *not* immediately affected by dopamine levels. Then again, the *pain* associated with a broken bone or cancer is a dopamine-related problem.

³ If the symptoms of fear or stress are severe enough to produce symptoms that actually resemble Parkinson's disease *and* the condition lasts for a significant period of time, the condition is referred to as “psychogenic parkinsonism.” But symptoms that resemble those of Parkinson's can also arise in a matter of minutes, and linger for a short period of time, such as an hour or so. I'll give a few familiar examples:

Consider a person with the beginnings of hypothermia (extreme chill) or the mind-altering stage of a severe flu during the alternating-fevers-and-chills phase. The shifts in body language brought on by these events can occur relatively quickly. What do they have in common with parkinsonism?

A person who is on the verge of severe hypothermia will be bent forward, his arms bent at the elbow and held close to the body, and his head pulled forward. His face may be nearly expressionless, his stride will be small, maybe even shuffling. His teeth may chatter and he may be trembling in his limbs. His speech will be very slow,

As for people who *do* have idiopathic Parkinson's disease, a negative placebo, an expectation of worsening problems, can cause a rapid acceleration of the symptoms of Parkinson's disease. This effect can even occur in a person who has physically recovered from Parkinson's *if* he imagines that he still has Parkinson's. His tremor and movement initiation symptoms may remain or might rapidly worsen even if his body structures are obviously healing.

In our experience, most unmedicated PDers *are* highly susceptible to negative placebos.

Examples of PDer placebo studies

Moving with sugar water

In a placebo study conducted in the early 2000s, placebo researchers injected PDers with sugar water. These were PDers who were accustomed to taking L-dopa based drugs and whose condition had advanced to the point where they were having On-Off behaviors in response to the medication.¹ Due to familiarity with drug effects, these people already knew how the drugs would make them feel: safe enough to move.² Thus, they had developed a specific expectation for drug-enhanced movement in response to their usual dose.

These PDers, while in an Off (rigid) phase, were told, falsely, that the sugar water was a dissolved form of L-dopa, and that the study merely wanted to see if the solution worked faster or at the same rate as the pill form of the drugs.

What happened after the PDers drank the sugar water? They uniformly responded to the placebo by experiencing ease of movement initiation in the exact same timing and behavior as if they had been given their usual oral dose of L-dopa. I repeat: they *thought* they had been given L-dopa, so they were able to move in the manner that their drugs usually allowed, even though

and at low volume. It may be extremely difficult for him to perform any movements that require him to open out his body and stretch languorously. He may have trouble initiating *any* movement.

In the case of hypothermia, availability of dopamine appears to drop dramatically – even to the point where a person can be immobilized. This condition can come on very quickly.

Based on extremely rapid changes in condition in our PD patients following a severe chill or overheating, a change that can last for several days even if the chill is quickly remedied, we hypothesize that dopamine is heavily drawn on, rapidly depleted, in conditions requiring a severe temperature regulation effort.

Just as Parkinson's-like symptoms can be brought on very quickly in a person who is accidentally locked into an industrial freezer, these same symptoms can quickly be brought on by intense anticipation of pain or fear *if* there is not a concomitant increase in adrenaline release.

These examples are induced by physical factors. The same symptoms can occur rapidly, almost instantaneously, in response to devastating emotional news.

These examples are meant to show that manifestations of poverty of movement, rigidity, and tremor can come on quickly, if the physical or mental state calls for them.

¹ The On-Off behaviors that occur from addiction to antiparkinson's medications are discussed at great length in my book *Medications of Parkinson's or Once Upon a Pill*, available for free download at www.pdrecovery.org.

² I need to mention this: many PDers vigorously insist that dopamine-enhancing antiparkinson's drugs do *not* make them feel happy. Nevertheless, increasing the joy signals in the brain *is* the mechanism by which the drugs work. Dopamine is the neurotransmitter of pleasure and joy. When the motor area feels joyful enough, a person can execute uninhibited movement. Whether or not the conscious mind of the fear-oriented PDer is able or willing to cognize joy may determine if the drugged PDer can feel an overt mood lift from the dopamine. Typically, by the time the drug use and the Parkinson's is advanced, even the PDers who deny feeling "good" from their medications state that they feel "bad" when the medications wear off.

they had actually only been given sugar water. Based on what we now know from PET scan studies, this result occurred because the PDers' brains released dopamine in response to an expectation of feeling good.

The placebo effect in this study lasted just as long as each PDer's typical duration of benefit from a dose of his usual drug.¹

The same type of study using sugar pills obtained the same type of results: PDers, when they think, mistakenly, that they have been given their medication, respond as if they had actually taken the medication.

Many variations on the above PD placebo study have been performed with similar results. Research abounds in this field. And nearly every study I have read that examines the placebo effect in a generalized way makes mention of the way in which PDers in particular respond to placebos.

Another different type of Parkinson's disease placebo study, described below, concluded that, possibly, the more dramatic the placebo action, the longer-lasting the placebo effect.

Placebo holes in the head

In the April, 2004, issue of *Archives of General Psychiatry*, an extreme placebo effect was described: 39 people with advanced Parkinson's had holes drilled in their heads. Half of the subjects had embryonic brain cells transplanted into their brains, the other half had "sham surgery:" holes, but no embryonic tissues. Neither the patients nor their regular doctors knew who had gotten the tissue transplants and who had the placebos (sham surgeries). Thirty of the patients agreed to participate in a long-term follow-up study. These people were asked whether or not they thought they had received the transplants.

"Those who thought they received the transplant at 12 months [after surgery] reported better quality of life than those who thought they received the sham surgery, regardless of which surgery they actually received," according to the write-up. It continued, "Some of the placebo patients made striking improvements. One patient said she had not been physically active for several years before surgery. After surgery, she was able to hike and ice skate. She eventually learned that she'd had sham surgery."²

As an aside, I have to wonder if the shock to the body caused by drilling holes in the head and brain was sufficient to amp up the sagging adrenaline levels a bit; after all, these people were able to sustain the placebo effects for such an extended period.

¹ I have lost the citation for this study. A quick look at the Internet, trying to find it again, brought up a reference to a similar study done by the University of British Columbia in Vancouver. This study used placebo "injections of a harmless saline solution" (salt water). PDers were told that they were receiving L-dopa (a dopamine precursor). Using PET scans that measure dopamine receptor activity in the brain, researchers were able to see a boost in dopamine activity levels in the PDers' brains – a boost to the same levels as produced by "the [dopamine-enhancing] drug commonly used to treat the disease." From "Science File: Healing Body by Fakery," *Times* staff writer, Robert Lee Hotz, Feb 18, 2002.

² Danial DeNoon, reviewed by Michael Smith, MD, in "Strong Placebo, Strong Parkinson's Effect," *WebMD Medical News*, April 14, 2004.

Maybe the adrenaline response to having a hole drilled in the head, combined with the dopamine-releasing positive-expectation (placebo) effect, might account for a placebo benefit that was longer-lasting than expected.

Also, the psychological benefit from *imagining* oneself to be one of the lucky ones, one of the ones who got the actual transplant (even though, in fact, they might *not* have gotten the transplant) may actually work to open the heart of the PDer just a bit – just enough to turn down the sympathetic nervous system and thereby be able to trigger neurotransmitter release.

A brain-implant placebo study

One of my favorite PD placebo studies, done in 2005, involved PDers who had received deep-brain stimulating implants.¹ I like this one because, in addition to obvious (visible to the naked eye) changes in movement initiation and cessation of tremor, the brain scans showed clear proof of inner brain electrical changes: these changes corresponded, not to the treatments, but to expectations induced by spoken words.

The deep-brain stimulating implants work by distracting the little electrical anxiety signals in the brain that contribute to immobility and tremor. The implants perform this distraction via the method of sending a much larger, more focused electrical shock signal into the brain. The brain is thus able to shift from an attitude of “uh oh” to one of “*Omigosh!!!*”

The adrenaline-boosting shift from the implants enables the late-stage PDer, whose heart to brain communication has become so reduced that he can no longer raise his adrenaline levels up to Functional Level anymore, to suddenly rise to the occasion of this new, significant alarm: electrical jolts into the brain! The sympathetic nervous system is stimulated; adrenaline levels get an upward nudge. The lower-level anxiety static in the brain gets overridden by the wire-and-battery induced stimulation which provides a sense of electrical trauma in the brain. Thus, the PDer once again has the release of sufficient mental and motor neurotransmitter (adrenaline): he can move easily.²

Getting back to the implant/placebo study: PDers had the implants inserted, *but* the battery pack that activates the inserts was not yet turned on. Then, to study the effect of placebo on these patients, half of them were told, falsely, that their implants *had* been turned on and that they *should* experience rapid improvement in their symptoms. They were also told that their

¹ L. Neergaard, “Expectations Can Help Healing,” AP, *Yahoo News*, Mon, Nov. 28, 2005.

² Although some uninformed clinical neurologists and other MDs who are out of the loop do imagine that the deep-brain stimulation (DBS) must somehow work by stimulating dopamine, the manufacturers of the product and researchers in the field know that dopamine increase has *never* resulted from DBS. The 1960 to 1995 preoccupation with dopamine in regard to Parkinson’s disease, and the subsequent assumption by the uninformed that “implants, if they work, must necessarily increase dopamine,” is outdated thinking.

The people who are actually doing current research in Parkinson’s know that the implants work by delivering a mild shock. The shock most likely encourages the release of adrenaline; it certainly does not cause the release of dopamine. The research that led to the approval of brain implants was done on lab animals in which parkinsonism had been induced. The stimulation from the implants allowed the lab animals to move, but their brains did not show an increase in dopamine.

In humans, unlike in the lab animals, the implants may have two mechanisms. They stimulate adrenaline, thus overriding the electrical disarray in the brain, but the surgery itself may also provide an *expectation* of feeling better. An expectation of feeling better makes a person feel safe, cared for. Thus, in some PDers, some amount of expectation-based dopamine release may be psychologically activated, especially immediately following the surgery.

tremors would stop almost immediately. These PDers rapidly began to experience normal movement initiation. Their tremoring instantly stopped. Brain scans of these PDers showed that the large number of tremor-inducing electrical firings in the “firestorm” area of the brain had calmed down.

Again, the PDers in this group were falsely told that their implants had been turned on. Their brain scans showed a calming effect. Their movement became easy and their tremors stopped *even though the implants had not been turned on*.

The other half of this group of PDers in the study was told, truthfully, that *their* implants had *not* yet been turned on. They were also told that they *should not* expect any immediate improvement until the implants were turned on later in the week. Subsequently, this latter group did not have any immediate improvement in tremor or movement, nor did their brain scans show any improvement; these people were waiting for the implants to be turned on so that they could begin to feel better. After the implants were turned on *and* the subjects were informed of the fact, this group could then also move easily. Only *after they were told* that they *should* feel better did their brains scans show a calming of the “firestorm” area.¹

But getting back to the main point, this placebo study, like all the others, provides further support for the idea that PDers’ movement-initiation problems and their tremors are expectation dependant: movement becomes easy, and measurable amounts (measured with PET scans) of dopamine are released if the PDer *expects* that he will be able to move easily.

Using people with Parkinson’s disease in placebo research

A person who follows the research on placebos might notice the frequency with which Parkinson’s disease is used in placebo research. There are two reasons for this. The first is, when looking for a placebo-induced change in behavior, the difference between “it works” and “it doesn’t work” is, in the case of Parkinson’s, visible from across the room. The PDers used in these studies nearly always are medicated. Very often, they are in the “on-off” stage of advanced Parkinson’s disease: they can move when their drugs kick in and they cannot move very well when the drugs wear off. This visible shift in movement when “On” or “Off” makes these people good subjects for placebo studies.

When doing scientific studies, the researcher finds it easier to work with subjects in whom the results are easily seen, glaringly obvious. If measuring the results of a study requires extracting molecules and measuring ever-changing brain waves, the results will be difficult

¹ I suspect this study was originally inspired by the fact that many PDers, including three in my experience, could move more easily immediately following the surgery, *prior* to the implants being turned on. Since the implants aren’t “supposed” to work this way – and do not work this way in lab animals – I imagine that a curious researcher decided to do this quick, easy, and very telling experiment. After all, if patients received benefit from simply having the implants positioned, what need is there for the battery pack and the electric stimulation?

I suspect that the adrenaline surge brought on by having holes drilled in the head and wires placed inside the brain is the source of the immediate, “pre-turn on” improvement. This adrenaline increase from surgery may be accompanied, in PDers, by an increase in dopamine due to expectation of improvement.

In light of the fact that many PDers do have an instantaneous improvement, the study group that was told to *not* expect any benefit until the wires were turned on was also being given a placebo – a negative placebo: they felt no benefit because they had been specifically told that they would not have a benefit. As for whether one group or both groups were influenced by what they were told is clear; all subjects had brain scans and motor function that corresponded to their expectations based on what they had been told.

and/or expensive to read. People with advanced Parkinson's, on the other hand, are very easy to "read:" either they can move or they cannot. For this reason, PDers are frequently used in placebo studies.

The other reason that PDers are so often used in placebo research is that PDers are highly susceptible to suggestion. The daily, even hourly variability of PDer's movement ability is increasingly recognized (among placebo researchers and even by observant PDers and their loved ones) as being highly mood and/or expectation dependent. Placebos are a method for stimulating the positive mood and expectation.

The physiological basis of the placebo response

Researchers used to think that PDers responded to placebo through some purely psychological influence. New research shows that the reason for PDers' response to placebo is physiological: placebos trigger the release of ample dopamine – even in people with Parkinson's.

Also, since the late 1990s, researchers have used PET scans to measure dopamine-receptor activity in the brain.¹ In PET scan research studies that measure the activity of dopamine receptors in PDers, the PDers who have received a verbal or physical placebo show an increase in dopamine receptor activity – the result of an increase in one's ability to use the dopamine system.

Again, though I risk redundancy, this increase in dopamine receptor activity occurs when PDers think, mistakenly, that they have been given some treatment that increases dopamine levels.

When a PDer is given sugar pills or sugar water and told that the pills or water contain his usual dose of dopamine-enhancing medication, he can feel the onset of easy, dopamine-style movement in the time frame that he expects from his medication. When the time comes when he expects his medication to wear off, he starts to move slowly again.

PDers who are given placebos can easily initiate movement. Under the influence of the placebo, they move and feel confident in exactly the same way that they move and feel when they take their antiparkinson's drugs. This effect lasts until the time arrives when they expect their placebo to wear off. The expectation of the placebo wearing off is usually based on the PDer's experience with his medications' wear-off timing.

¹ PET scans measure activity of dopamine receptors. In the PET scan process, radioactive dopamine-like chemicals are injected into the body. These radioactive molecules migrate into the brain and attach to dopamine receptors. If a significant number of dopamine receptors are become dormant through the decades (as they have in a person with idiopathic Parkinson's disease), the typical (healthy) number of radioactive molecules will not be able to attach. In PDers, the scan *may* show a dopamine receptor response that is lower than that of a healthy person.

In people who have rapid-onset symptoms of parkinsonism, as occurs in hypothermia or trauma, the dopamine receptors are still healthy. Therefore, the PET scan may show normal receptor activity with the dopamine-like radioactive tracers even though dopamine is not being released by the patient.

This is the theory, at any rate. However, as noted elsewhere, the medical jury is still out on whether or not these scans are an accurate diagnostic tool for Parkinson's disease.

SPECT scans are a new variation on PET scans. SPECT scans are currently used only for research, and are not available for the general public.

These movement-observation and brain-scan placebo studies make it clear that dopamine *levels* in people with idiopathic Parkinson's are sufficient. The problem in Parkinson's disease is insufficient dopamine *release*, and not dopamine insufficiency, per se.

The expectation-dependant symptoms of Parkinson's, movement initiation and, sometimes, tremor, are due to an *inability to create the mental state* necessary to trigger dopamine *release*. As placebo studies make clear, these symptoms of Parkinson's are *not* due to *insufficiency* of dopamine.

The PD symptoms that do not respond to placebos

In response to placebos, PDer's *channel*-related symptoms are not improved. The PD symptoms mentioned in chapter seven that are located on the Stomach channel, symptoms such as hardening of anteriolateral leg muscles and numbness in the toes, will not be altered in response to a placebo. These problems are due to injury and electrical distortion, not inhibition of dopamine release. A PDer who has lost his sense of taste and whose foot and "smile" muscles are physically distorted will still have no sense of taste and will still have his foot and facial distortion problems even if he has a good movement-initiation response to drugs or placebo.

Only the PDer's movement initiation, speed-of-movement problems, and in some cases, the relatively more severe, fear-based portion of his array of tremor problems – the problems that are dopamine related – will respond to the placebo.

Negative placebos

In the brain implant study, the people who had no benefit from the deep-brain stimulating implant until they were told that they would be victims of a negative suggestion, a negative placebo. They were told that they should not feel better, and so they did not – even though many PDer's do have an immediate result from the implant process, even before the battery is turned on. Possibly this placebo benefit occurs in people whose doctors forget to tell them that the battery will not be turned on for a few days after the surgery.

In the placebo study, by *telling* the PDer's in advance that they would feel no benefit from the surgery until the batteries were turned on, the researchers were using a "negative placebo," a negative suggestion.

Almost all PDer's, even those who have never taken medication, respond strongly to negative placebos, to suggestions that the PDer should feel worse. The following two case studies will look at the role of negative expectation in the rapid worsening of Parkinson's disease symptoms in two PDer's in our recovery program.

A mind game example: self-induced parkinsonism

For the first case, I will quote directly from an email that we received. Extra information that I add to help the reader will be in brackets [].

"Dear Chris and or JJ,

"You may remember me. I've made three weeklong trips from Colorado to see you guys in the last 3 years. A few months after I saw you last I began manifesting...the half-healed state. The tremor intensified dramatically and I became profoundly fatigued and weak.

"This went on month after month and I became one of those people who panicked and went to a neurologist who put me on a very low dose of Sinemet [L-dopa]. I had a very violent

[excessive movement] reaction after the first dose and I vowed to never take it again. The fact that I would take it at all gives you some idea of how defeated I was.

“As luck or fate would have it, the new edition of your book became available for download the day after I took the Sinemet. I immersed myself in your writings (again) and gradually began to get some hope and energy back.

[The older edition did not have the material on the adrenaline-dopamine relationship, or information about fear and negative thinking, so there was much new material for this reader.]

“Eight months after the fatigue started, it began to lift until, by July 1 [2005], I was back to 95%. Particularly as the later chapters on “Fear,” “Negative Thinking,” and “Mind Games” became available and I integrated their ideas into my daily practice, my symptoms markedly improved. Progress was happening and the future was opening up again. Then your addendum of October 2005 was released.

[This addendum to our website was a warning: based on our findings, people who had taken dopamine-enhancing drugs (L-dopa, dopamine agonists, or MAO inhibitors) prior to entering our program had developed difficulties (symptoms that corresponded with those of drug-induced parkinsonism) that seemed to, in most cases, prevent full recovery. Worse yet, their recovery usually stalled in highly traumatic manifestations.

Because people who have even partly recovered are usually not able to tolerate the medications any longer, the people who were part-way recovered but who were suffering from symptoms of drug-induced parkinsonism were in a very difficult position. Therefore, with heavy heart, we made the decision that we would no longer include in our research project people whose experiences with these drugs had, on top of their idiopathic Parkinson’s, also set in motion drug-induced parkinsonism (semi-permanent brain damage). The writer is referring to this disheartening warning, a warning about these specific types of drugs, which was dated October 2005. The writer continues:]

“My intention is not to shoot the messenger, instead it’s to let you know how personally devastating [this addendum’s] effect was on me...Its effect has been to send me into a tailspin of negative thinking, hopelessness and depression with the *resultant worsening of symptoms*. [Italics are mine.] So the reason for this letter is to get clarity and guidance. I was on Artane for almost 2 years from 2000 to 2002 but never took a full dose...what are my options? Please let me know. – SP”

I will paraphrase our reply:

“Dear SP,

“Your tailspin is an example of the role of expectation on dopamine release and mood regulation. The problems with brain damage, as described in our October addendum, are set in motion – as we clearly noted – by the dopamine-enhancing drugs. The drug that you took, Artane, is not a dopamine-enhancing drug. Artane is an anticholinergic. Anticholinergics are a completely different family of drug. The book *Medications of Parkinson’s* explains very clearly that Artane is not a dopamine-enhancing drug.

The drug that you took is a mild muscle sedative. It is not a brain stimulant. It does not cause brain damage. Therefore, your symptomatic tailspin, which occurred because you thought you would not be able to recover – despite your own positive changes – was entirely the product of your imagination. We have seen *no* problems, no impediments to recovery, in those PDers who took Artane.

“It sounds like you’ve really experienced the influence of positive and negative thinking. Your improvement back up to 95% really happened. There is no need for you to listen to anyone or anything that tries to tell you that what you have done/are doing is impossible when you have clearly seen for yourself that you can do it. Yes, you can do it. You did do it.

“You might choose to view your recent tailspin objectively (having already fully experienced it subjectively) simply as the example of what happens when one does the opposite of what you had been doing throughout those several months of amelioration and improvement that you recently enjoyed. You might also choose to note that the difference between darkness or light is often as simple as the flick of a switch or a drug warning that, as it turns out, didn’t actually apply to your case.”

SP wrote back to us:

“Your words had the effect of loosening the choke-hold Devil Doubt (aka the fear-based mind) had on me. What had happened the last few months [SP’s gradual improvement] was undeniable and yet the frightened little guy in me was particularly vulnerable to [the addendum]...

“Thank you again. – SP”

Another example of attitude-induced parkinsonism

I received a detailed email from The Netherlands. It was several pages long, so I will paraphrase it:

“My young husband (early 40’s) was diagnosed in June, 2005, with early Parkinson’s disease. His symptoms were still very mild at that time. I am Chinese, an acupuncturist, and so we were looking for alternatives to the drugs offered by the doctor.

“However, by mid-September, four months after his diagnosis, he was no longer able to pick up our two young children, and had difficulty dressing himself or performing other activities of daily living. Although I gave him some herbs that – interestingly, in retrospect – support the Stomach channel, his Parkinson’s symptoms did not improve.¹ We were alarmed at his very fast rate of decline and were considering going back to the neurologist.

“Near the end of September, I discovered your website. I then contacted some people in Amsterdam who have been in your program. They were very helpful and positive about their experiences with your program. So I downloaded your book [the first 20 chapters] and gave it to my husband. He stayed up all night reading it; he finished it in just two days.

“Since he finished your book a few weeks ago, his condition has completely reverted to what it was back in June: very mild. We have not yet started doing any treatments, but he is now in approximately the same physical condition that he was in when he went to see the neurologist four months ago: he now has, once again, very mild symptoms of early Parkinson’s disease – symptoms that do *not* impede his ability to perform activities of daily living.”

In case the reader is wondering why the man improved without being treated, the most likely explanation is that his rapidly worsening symptoms were due to his *expectations* of rigidity and immobility. When he found that his illness was *not* incurable, the mood shift allowed him to revert to his previous condition, in which he was once again manifesting his actual, physical

¹ Herbs that amplify Stomach channel Qi will not reverse Parkinson’s disease. The rules for treating Rebellious Qi are very straightforward, and include this warning: *never* tonify (strengthen) a condition of Rebellious Qi.

symptoms (which happened to be quite mild), without the extra onus of the psychological weight of his diagnosis.

The power of the diagnosis

The above case study makes a very powerful point: the “curse” of being diagnosed with an incurable illness can accelerate the problems of the illness, especially when the illness has to do with mood and expectation-dependent neurotransmitters.¹

As noted in previous chapters, I have seen many PDers who told me that their symptoms went into a tailspin when they received their diagnosis. On the other hand, I have treated many people who never suspected that their shuffling feet, slight tremor, cogwheeling wrists and ankles, expressionless face, non-swinging arms and postural stoop signified anything other than a passing “muscular thing.” These undiagnosed people all recovered easily and completely.

I have also had PDers (who had been told by a neurologist that they had PD) undeniably recover *but* then go into a terrible tailspin, complete with tremor and rigidity lasting for a few days, after tripping over an unexpected sleeping cat or some other darned thing. They’ve wailed at me something like: “I almost fell down! The Parkinson’s must have returned!” No amount of reassurance that *everyone* stumbles once in a while can shift their certainty.

It can then take a few days or a week before these “fallen” people accidentally perform some unexpectedly agile motor activities. These activities suggest to them that they have, once again, “recovered,” after which the recent PD-like symptoms evaporate.

This ends the section on placebo *research* in PDers. The next section in this chapter discusses psychogenic parkinsonism. In a sense, this form of parkinsonisms is similar to that generated by a negative placebo, inasmuch as it does not have a physiological basis, but is induced by the mind.

Psychogenic parkinsonism

In the words of Michael S. Okun, MD, in Askthedoctor@forum.parkinson.org, Dec 27, 2005: “These are folks who have slowness of movement and a lot of features that look like PD, but have another reason for the symptoms...this reason may be psychogenic – stress, anxiety, depression, affective disorder, rape, trauma, other event...or unknown... and if caught early they can be treated to complete resolution in many cases. They are not as rare as one may think!”

¹ Back when I was still in medical school, we were told that, in China, it was against the law to tell a patient that he had cancer. A patient, if informed that he had cancer, might easily die within two weeks because of the powerful grip that the word “cancer” has on the Chinese consciousness.

On the other hand, if a person in China with incurable cancer was only told that he merely had a “deficiency” and that strong tonics might help, he would usually take the tonics (and pain-reducing medications, if necessary) and go on to comfortably live, in some cases, several more years (sometimes even ten years) before the cancer actually moved into a quick-moving lethal phase. This political issue, in which a patient’s “right to know” may conflict with the doctor’s knowledge that the patient will be better off if he doesn’t know, can be argued either way. I won’t even begin to go into it here, but you can see the ramifications that it has for the subject of Parkinson’s disease.

I know that these cases are not rare. I have seen some. Roma rapidly developed psychogenic parkinsonism. Roma's case was mentioned briefly in an earlier chapter: she was the patient who didn't tremor while eating desserts because "desserts don't matter."

Roma, or "Desserts don't matter!"

Roma came to see me after her neurologist diagnosed her with Parkinson's disease and her Ayurvedic doctor confirmed the diagnosis.

Roma walked gingerly, carefully, into my office and greeted me with a rapid vocal patter. Her posture was hunched and she held her arms tightly by her side. But after sitting down on the couch in my office, her spine relaxed, her left shoulder relaxed, and she leaned back easily into a comfortable position. She effortlessly crossed one leg easily over the other, and, while talking, gestured quickly with her hands to emphasize her points. Her very faint tremor came and went in her right hand. After observing her for several minutes, I said, "You don't have Parkinson's disease. What's going on?"

Roma was stunned and asked why I was so certain. I then had her perform a series of exercises including the cogwheeling tests, the balance tests, the "reach upwards and take a deep breath test," and so on. She had no Parkinson's-like responses to any of these tests. What she did have was a right arm that was stiff due to tightness at the shoulder and a right hand that tremored a little, now and then. Also, though she took small, slow, careful steps, carried her arms in a bent position and was slightly hunched over when *walking*, her body language always relaxed into a position of ease as soon as she sat down. Sitting, she gestured quickly with her arms and hands and moved her head, neck, and torso easily.

The Qi in her legs was working just fine, so I ended up working on her stiff arm, but my parting words to her were that I didn't think she had Parkinson's.

She called me a week later. She had revisited her Ayurvedic doctor and told him what I'd said. He checked her out thoroughly and then said he was chagrined that he had "missed" the diagnosis. He agreed that she certainly did not have Parkinson's.

So what did she have?

Roma came to see me again and I grilled her with lots of questions. Here were some of the answers.

Her tremor was the worst when she was eating. While this by itself is not unusual for a person with Parkinson's, note this: she never tremored when she was eating desserts.

It turns out, Roma had a PhD in nutrition. She was proud to have studied with one of the top nutritionists of the century. To her mind, food "mattered." Food mattered a lot. I came to suspect, later on, that she had put such faith in nutrition that she imagined "correct eating," whatever that is, might be able to prevent cancer, slow aging, keep the bones strong, etc.

So that filled in one part of the puzzle. She tremored when she ate nutritious food because she was anxious about nutrition. Since dessert "didn't matter," she didn't tremor during dessert.

But why had she started trembling to begin with, and why, when walking, did she take small steps and carry herself in that semi-rigid, PD-like hunched posture?

I asked her when the symptoms had first appeared. She said she wasn't sure, but she would ask her daughter.

Roma's dread of osteoporosis

The next time I saw Roma, she gave me the answer to the puzzle.

Her daughter had told her, “I know exactly when you starting walking in that weird way and trembling. It was the day the doctor told you that you had osteoporosis. You called me and said that you had osteoporosis, and when I came over to see you, you were moving all hunched over all of a sudden. You’ve moved that way ever since.”

I asked Roma what she had to say about that.

“Of course, since I have osteoporosis, I’m careful now when I walk. I have to worry about falling down. I need to be extra careful, take small slow steps, so that I don’t break any bones. I’m not trying to look like I have Parkinson’s, I just need to walk this way to be sure that I don’t lose my balance and break something.”

In fact, this was not true. Roma was incapable of walking with large steps or swinging her arms while walking. She wasn’t just “being careful.” When she walked, she was terrified of falling, and she moved as if she was nearly immobilized with fear. She took tiny, shuffling, PD-like steps.

I asked Roma how she had felt when the doctor told her that she had osteoporosis. She said that she remembered exactly. “I felt decrepit. That’s the word that flashed in my head. Decrepit. ‘You’re decrepit,’ I told myself.”

I asked Roma what it meant to her to be decrepit. She told me that it meant that she was disgusting. She was a failure. She hated being decrepit, it was the worst thing in the world.

I realized now why Roma was walking the way she did: she was deathly afraid of “decrepit.” I could also hypothesize about her horribly mixed feelings about eating. All of her nutritional studies had let her down. Her careful eating had not prevented her from becoming decrepit, the worst thing in the world. Possibly, subconsciously, she decided that she needed to be more vigilant about getting enough nutrition. Meals may have become, to some part of her mind, her only hope. Thus, the enormous emotional strain she felt when eating. Of course, desserts didn’t count; desserts, she assured me, were just for fun.

Roma’s symptoms all began on the day she was told that she had osteoporosis. One day she was fine, the next day she had full-blown rigidity and poverty of movement, and a small tremor – if she was walking. She had psychogenic parkinsonism.

Three years after she met me, Roma recalled the time she first consciously dissociated from her feelings; when she was nine years old she was sent off to summer camp because her mother was sick. When she returned from summer camp, her mother was dead and buried. She understood that she was not supposed to ask questions or “make a fuss.”

Her relatively recent diagnosis of osteoporosis was, for Roma, another horrible thing from which she needed to dissociate. Her body was decrepit: she dissociated from her body. She rapidly developed psychogenic parkinsonism. She did not have any of the classic physical changes of Parkinson’s disease, the symptoms that might show up in a photograph. She only had those symptoms that related to movement initiation and tremor: the mind/emotion related symptoms.

Over the next two years, she developed painful rigidities and movement initiation problems. Her movement problems became almost constant.

Not until I understood the role of the dissociation response in psychogenic parkinsonism was I able to help Roma in any way. I did not work on Roma’s feet. I taught her how to turn off the dissociation response. After she *temporarily* mastered it, she was once again, in her words, “flying high.” But she knew that when she left our program in Santa Cruz and went back to her home on the east coast, her symptoms would return and worsen. And they did.

The case of Roma is a powerful demonstration of how negative feelings and negative expectations can rapidly create symptoms that resemble Parkinson's disease, complete with tremor, rigidity and slowness.

Interestingly, after three years, Roma had a PET scan done which showed a decrease in dopamine receptor activity that was similar to the patterns seen in people with idiopathic Parkinson's disease. As mentioned earlier, doctors have seen that PET scans do not necessarily confirm Parkinson's disease. Many people who are diagnosed with PD have PET scans that are perfectly normal. Roma's case was clearly psychogenic: she who went into a rapid emotional decline after being told that she had Parkinson's; as mentioned in an earlier chapter, she developed highly specific PD symptoms in response to my specific suggestions; and recalled shutting off her heart when her mother died. These are all symptoms that suggest an utterly psychogenic cause for her symptoms. And Roma had a PET scan consistent that showed inhibition of dopamine receptors.

Wrapping up this chapter on placebos, I wish to emphasize one point. Placebos do not allow PDer's to move simply because PDer's imagine that they are able to move. The placebo studies that measure actual brain neurochemistry have found that PDer's have actual shifts in brain chemistry – an increase in dopamine release – in response to placebos. In response to positive placebos, PDer's release dopamine. The dopamine change is measurable.

In other words, PDer's do have sufficient dopamine to operate their mental and motor systems. What PDer's also have is an inability to *release* dopamine. Again, the decline, over decades, of the dopamine-producing cells in the midbrain, is due to the lack of *use* of the dopamine-producing cells. However, even with the decline in dopamine-cell numbers, the remaining cells are *absolutely able* to produce as much dopamine as is needed.

The brain, extremely plastic, increases development of those brain areas that are highly used. The brain can also render dormant (reverting even to an undifferentiated state) those cells that are not called on.

The next chapter will expand on this concept of inhibited dopamine *release*. Dopamine release is utterly, completely dependent on expectation and feeling safe. This concept is crucial.

A PDer must appreciate that his emotional posture and his ability to have positive feelings about his own body and mind are the determining factors in whether or not he can initiate movement or move easily. If the PDer does not understand this, then, following the healing of his foot injury, he may passively wait for his mobility to miraculously return. He may imagine that merely fixing his foot injury should allow him to move normally once again. But he will be wrong; fixing the foot merely allows the brain to be *capable* of releasing dopamine at whatever levels the heart calls for. The PDer is still responsible for instructing his heart to feel safe, and to interpret the sensory experience of having a body as positive instead of threatening.

If, however, his brain processes have become habituated to negative attitude or if he remains in the mental state induced by the conscious cultivation of the dissociation response, he will not be able to feel safe. He will not be able to activate the release of dopamine in the midbrain. Though he once again has the *capability* to release dopamine, he will remain locked in dopamine deficiency and dopamine deficient behaviors. Although his face may be more symmetrical, his circulation improved and his injury healed, he may still have difficulty with

movement initiation, slowness of movement, and tremor: he may look like a person who's been accidentally locked in an industrial freezer for ten minutes.



“His heart was one of those which most enamour us, - Wax to receive, and marble to retain.”

from Lord Byron's *Beppo*

CHAPTER TWENTY-SIX

MATTERS OF THE HEART

Research in the fairly new field of neurocardiology has direct implications for our Parkinson's research. This chapter will introduce the physiology of the heart-brain relationship and its significance to Parkinson's disease.

The heart is a ball of nerves

Most twentieth century anatomists considered the heart to be a hollow mass of muscle fibers whose job was to pump the blood. It turns out that the heart is actually about sixty percent nerve tissue.¹ I refer to these nerves as heart-nerves. They communicate emotional information to and from the brain.²

The heart-nerves are *not* the same as the heart-*muscle* nerves used in operating the heart pump.

The heart-nerves communicate with the brain in two ways. First, the heart connects to the brain through a pair of nerves (one on the left, one on the right) that goes to and from the heart via the spinal cord: up the spine into the brain. Second, the heart connects to the brain through the pair of left and right vagus nerves, which travel to and from the brain stem out through the tissues of the neck, and then down through the torso.³

Information from the heart via these heart-nerves tells the brain the manner in which to interpret incoming sensory and thought information: whether ongoing events are good or not, and how much. These interpretive instructions are based on the feelings of the heart, and not on

¹ “There are at least forty thousand nerve cells in the heart – as many as are found in various subcortical centers in the brain.” *The Heartmath Solution*, Childre and Martin, HarperSanFrancisco, 1999, p. 10.

The Heartmath Solution describes some of the work of the Heartmath Institute, a highly respected organization dedicated to sharing information about the heart's physical relationship with emotions and thoughts, and to teaching techniques that harmonize heart and brain electromagnetic patterns. Because *The Heartmath Solution* is found several times in the footnotes of this chapter, the reader might want to know if any “hard science” publications support the findings of the Heartmath Institute. Yes; *The Heartmath Solution* is heavily endnoted in the best scientific tradition with references to highly respected science journals. For example, the above quoted sentence is endnoted to this reference: Armour, J. and Ardell, J., eds. *Neurocardiology*, New York, Oxford University Press, 1984.

² In this book, I use the hyphenated term “heart-nerves,” to differentiate these brain-connecting heart-nerves from the nerves that regulate the beating of the heart. This hyphenated format is not standardized. Although neurocardiology is a growing field, I have not yet learned of a distinct nomenclature for these nerves that differentiates them from the nerve triggers that regulate the heartbeat.

³ The lengthy vagus nerve touches more than just the heart. It traverses the torso, touching the stomach, the intestines, and most other innards that are activated in times of mental harmony and somewhat inhibited during times of stress. The vagus nerve plays a large role in the sequences described in this chapter, but I'm not going to discuss it. It would be too much information for someone whose goal is merely understanding Parkinson's disease. Believe it or not, I'm trying to keep the length of this book under control.

brain-based thought patterns. The heart feelings are formed by the heart's electromagnetic resonance with outer and inner experiences.¹ These heart feelings are then communicated to various parts of the brain via the heart-nerves.²

An example: the melody line recognition area

For example, one area that receives information from the heart-nerves is located in the brain's frontal lobe. This area is immediately adjacent to the place in the frontal lobe that is activated when one follows a line of melody. It has been proposed by western brain researchers that the intimate proximity of these two areas is the reason that music can quickly evoke a mood or emotion. An old favorite song often evokes the mood and energy level – the heart feeling – that a person had “back in the day,” when he first learned the song.³

¹ This idea conforms with Vedic (5,000 year old Hindu philosophy) teachings. In the 20th century, Paramahansa Yogananda, an international authority on the science of yoga and interpretation of the Vedic classics, often used the analogy of radio wave reception and tuning to explain the mechanism by which the heart can tune in and resonate with various wavelengths. (I imagine that explaining the concept of heart wave resonance must have been very difficult during the dark ages, before the days of radio, TV, the Internet and global positioning. In the 21st century, using an electrical system to tune in with invisible waves is a fact of life.)

² *The Heartmath Solution* explains that the heart behaves as if it has a “brain” of its own. It also points out that the brain responds to commands sent by the heart, and that, oppositely, very often the brain sends commands to the heart which the heart may or may not comply with. The book endnotes this information to Lacey, J. and Lacey, B. “Some autonomic-central nervous system interrelationships.” And Black, P., *Physiological Correlates of Emotion*, New York, Academic Press, 1970:205-227.

Heart instructions are supposed to be dominant over brain commands: brain instructions are supposed to be subordinate and can be overridden by the heart. In PDer's, we find that this excellent chain of command system has been over-ruled. Because PDer's dissociation from their hearts, the brain is in charge by default. What caused the dissociation? The brain. Essentially, by “getting rid of the heart,” the brain that has usurped the leadership role of the heart. This was the condition against which St. Teresa of Avila warns us when she says “Pay no more attention to the brain than you would to the ravings of an idiot.”

“The heart produces and releases a major hormone, ANF (atrial natriuretic factor), which profoundly effects every operation in the limbic structure (the emotional brain, also known as the primitive brain, or the “lizard brain”). The limbic area, in addition to regulating non-reason-based responses, also has an effect on memory, learning, and the hormone centers,” says heart expert Joseph Pearce.

He continues, “Approximately half of the ANF released by the heart helps to integrate the rest of the body, allowing its parts to perform as a whole.”

The other half works with the brain; Joseph Pearce further says “it can carry on a twenty-four-hour-a-day dialogue between the heart and the brain The heart is also a very powerful electromagnetic generator. It creates an electromagnetic field that encompasses the whole body and extends out anywhere from eight to twelve feet away from it. It is so powerful that you can take an electrocardiogram reading from as far as three feet away from the body . . . this electromagnetic field affects the brain. All indications are that it furnishes the whole radio wave spectrum from which the brain draws its material to create our internal experience of the world. The radio spectrum of the heart is profoundly affected by our emotional response to the world. Our emotional response changes the heart's spectrum, which is what the brain feeds on.” (From Chris Mercogliano, Kim Debus, “Does the Heart Have a Brain? An interview with Joseph Chilton Pearce,” *Self-Realization*, Summer 2000, pp. 42-44. For more details on the research papers supporting this work, please visit the website of the Heartmath Institute: www.heartmath.org.)

³ Music, for centuries, has been anecdotally connected with the heart. Research done at Dartmouth College in 2002 actually pinned down the two brain areas that form the connection: one stores emotional information from the heart and the other tracks melody lines. The two areas are adjacent to each other in the brain's frontal lobe. See: Cédric Bihl, “Un air de déjà-entendu . . .” *National Geographic France*, août 2003, p. 19.

I want to share a story of a patient of mine (not a PDer) who experienced the connection between heart feeling and music. He had been emotionally shut down since he was three years old, since the traumatic time that he

The processes involved here begin with the feelings that the heart was experiencing at the time a given song was first heard. The heart-nerves send to the brain the information about what the heart is experiencing. An imprint of these feelings, and a note as to the quality and quantity of feeling evoked at the time, is stored in the brain. *And* the feeling information is linked to the melody information!

Years later, if the ears receive the sound of that music, the melody-line tracking area in the frontal lobe recognizes the song. This recognition triggers the link to the stored information about the original heart feeling. The heart then replicates, to some extent, that original feeling.

The heart's role in emotions

Asian medical theory holds that the heart, as it resonates – or not – with the electromagnetic fields of inner and outer experiences, is the initial determinant of feeling and emotion.¹

In modern times, the general public has been taught that all thoughts and feelings are based in the brain; the body below the neck is merely a machine that transports the head around. But the general public is not up to date. Research in modern neurocardiology is starting to support the idea that the heart is the original source of feeling and emotion, a concept that's been a core precept in nearly every culture, ancient and modern.

When a person listens to a beautiful symphony or beholds a magnificent sunset, he might feel expansion in the chest. This feeling of expansion results from an increase in amplitude of the electromagnetic waves of the heart. This increase in amplitude is due to the heart's resonance with the energy patterns in the music or the sky. These heart-feelings are *not* based on the brain's

became deaf. In response to treatment, he began to experience heart feelings – and started singing a song that he'd learned in pre-school. When he felt a wave of expansion in his chest for the first time since age three, he was in his living room. I paraphrase his report: "I felt affection for the first time in twenty years. I happened to be staring at the sofa; I felt such affection for that sofa! And when I felt that affection, I suddenly found myself singing 'The Itsy Bitsy Spider.'"

Going off on a tangent, Paramahansa Yogananda, the great 20th century teacher of meditation, used to say that constant inner chanting is as important as meditation in the battle for Self-control of the mind and heart. I forget the exact wording, but it was something along the lines of "Chanting is half the battle." The significance of keeping an inner song running through the head at all times – a spiritually uplifting song – suddenly came home to me with a bang when I understood how the music-association area of the brain can be used to stimulate the nerves that open the door to heart-feelings.

¹ In this sentence, "Asian medical theory" refers to theory based on the Hindu philosophic tradition. In Great Britain, "Asian" usually refers to things Indian. Confusingly, in Pacific Rim countries, including the United States, the word "Asian" usually means Chinese, Japanese, and Korean. In nearly all instances in this book, the term "Asian medicine" is used in the latter sense. That is why I clarify here.

Ultimately, the teachings of both India and the Far East are similar when it comes to discussing the role played by the heart. However, in the United States, there are far more practitioners of Chinese medicine than of Ayurvedic medicine. Therefore, I am explaining most of my arguments along the lines of the well-known Chinese principles.

Even so, although I am an acupuncturist and I shore up my arguments about Parkinson's disease with principles of Chinese medicine, it was through rigorous study and application of ancient Indian philosophy and science that the meanings of the (often poorly translated, even baffling) ancient Chinese teachings came to light for me.

interpretation of the music or the colors in the sky at sunset. These feelings precede any brain involvement.

These feelings are caused by changes in the electromagnetic wave *patterns* of the heart and changes in the *amount* of energy in the electromagnetic signals produced by the heart. Resonance or conflict can increase or decrease the amplitude, the size, of waves. In the heart, this resonance – or lack of – translates into increased or decreased amount of various heart feelings.

Many people are surprised to learn that the electrical activity of the heart creates electromagnetic patterns: heart waves. However, if they recall that the brain's electrical activity creates measurable brain waves, they will understand that the heart's electrical activity creates measurable heart waves.

The heart's electrical field is quite large; it can be detected and charted from several feet away. The heart's electrical signals are holographic (the same in all directions, whether or not they are measured from the front, back, top, or side).¹

HEART BRAIN ENTRAINMENT

When a person feels content or calm, his brain-wave patterns entrain with his heart-rate-variability patterns.² A measurable synchronicity between the heart rate and brain waves occurs. The heart, not the brain, sets the pace.

When a person becomes fearful, this synchronicity is broken off. The heart rate variability patterns become jagged and disordered, but more significantly, the brain wave patterns become unrelated to the heart rate patterns. I repeat, when fearful or under stress, brain waves cease to be entrained with the heart-rate variability patterns.

When the fear is over, the brain's wave patterns can again become entrained with the heart's wave patterns.³

¹ "The heart's electromagnetic field is the most powerful produced by the body; it's approximately five thousand times greater in strength than the field produced by the brain, for example. The heart's field not only permeates every cell in the body but also radiates outside of us; it can be measured up to eight to ten feet away with sensitive detectors called *magnetometers*... Scientists at [various centers] have found that the electrical information patterns generated by the heart are detectable in our brain waves via a test known as an electroencephalogram (EEG)... A series of experiments by Gary Schwartz and his colleagues at the University of Arizona found that the complex patterns of *cardiac* (italics are mine) activity in our brain waves could not be fully explained by neurological or other established communication pathways. Their data provides evidence that there's a direct energetic interaction between the electromagnetic field produced by the heart and that produced by the brain... When we focus attention on our hearts, the synchronization between our hearts and brains increases." From *The Heartmath Solution*, Childre and Martin, HarperSanFrancisco, 1999, p. 33-34.

² The term "heart rate variability" might want some explaining. Heart rate variability is defined as the beat-to-beat changes in the heart rate. Heart beats are not regularly spaced. When the doctor measures someone's pulse, he is noting the average heart rate, not a fixed rate. The heart rate changes with every heart beat even when we're sleeping.

As recently as the 1960s, it was assumed that a rock steady heart rate must be a good thing. We now know that a heart that *does* maintain a steady, unvarying rate is a heart at risk, a heart that has lost its ability to respond to outer circumstances. Heart rate variability declines with aging. (Possibly, the reason that beta-blockers and pacemakers can cause emotional emptiness in some people is that they inhibit the full range of heart rate variabilities.)

³ *Heartmath Solution*, Ibid, uses this example: "Because the heart is the strongest biological oscillator in the human system – the equivalent of the strongest pendulum in a collection of clocks [the principle of entrainment was

Understanding the disruption of heart-brain entrainment

The benefit of disruption of heart-brain entrainment during times of stress or emergency can be easily understood. Consider the example of an injured person running from a hungry lion. At such a time of crisis, a person does not want access to his feelings or information about his own physical and emotional pain: he does not want full access to his heart's ability to resonate or not with inner and outer experiences.

The lion example

A person with a broken leg can run for miles on a broken leg if his life is in imminent peril. When running from the lion, a person does not want to be distracted by the fact that his leg is broken. He does not want to be distracted by the fact that his best friend rejected him two days ago. The perceptions of physical and emotional pain are dangerous distractions when one is running from danger. It may be that this long-recognized ability to detach from one's own feelings of physical and emotional pain during times of stress or fear is due to the non-entrainment of the heart and brain waves at these times.

During times of perceived danger, the brain wave patterns temporarily become independent from the heart wave patterns. It may be that, with brain waves thus disconnected from the heart, going off in their own direction, one's brain cannot fully cognize one's own sensory feelings, including one's own physical and emotional pain: interpretation of sensory nerve activity is primarily guided by the heart.¹

Other capabilities that may be inhibited during this heart-brain disconnect are the abilities to indulge in positive visualization and mental imagery. Playful imagination or fantasizing positive outcomes during times of emergency might well be dangerous distractions from the job at hand. For that matter, pleasant feelings can also be dangerous distractions; the moment of fleeing a wild lion is *not* the time to think about one's upcoming art project or to marvel at the ecstatic purples and golds of the sunset overhead. Positive sensory feelings and emotions, as well as negative ones, are inhibited during an emergency – when the brain wave patterns are disconnected from the patterns of the heart.²

A chemical shift

Heart signals sent to the brain via the vagus nerve activate the brain's dopamine-based mental and motor processes, and stimulate the parasympathetic (feeling contented) nerve system. Heart signals sent to the brain via the heart's spinal nerve activate the brain's adrenaline-based mental and motor processes, and stimulate the sympathetic (feeling fearful) system.

first realized from studying a collection of pendulum clocks] – the rest of the body's systems can be pulled into entrainment with the heart's rhythms. As an example, when we're in a state of deep love or appreciation, the brain synchronizes – comes into harmony – with the heart's harmonious rhythms.”

¹ It appears that the level of non-perception is on a sliding scale: the greater the emergency, the less one is able to perceive his own feelings. In spite of the heart-brain patterns being synchronous or non-synchronous, a seemingly black or white, all-or-nothing condition, we do see a sliding scale of emergency-response emotional shut-out. This sliding scale may be due to the continuous flow of heart-nerve information between the heart and the brain. Even when the heart and brain wave variability patterns become non-synchronous, the heart-nerve continue to send information about the *degree* of problem, and the *extent* to which thoughts become adrenaline-dominant.

² Therefore, when people consciously shut off their hearts to prevent awareness of a negative experience, they inadvertently also shut themselves off to positive experiences.

During times of contentment, the heart uses the vagus nerves more and the spinal nerves less. During times of stress or emergency, when the heart and brain waves become non-entrained, the heart uses the spinal nerve more and the vagus nerve less. Both nerves sets are always somewhat in use. Even when a person is feeling primarily contented, a small amount of energy may be flowing in the sympathetic nerves.

The extent to which the brain is informed of heart information via the spinal nerve determines the extent to which the brain use adrenaline-based commands to activate motor and mental function of the sympathetic nervous system. Thus, a nerve and neurotransmitter shift towards adrenaline and the sympathetic nervous system accompanies the electromagnetic change that occurs during heart-brain wave *non*-entrainment.

In an emergency, as adrenaline is increasingly released, the release of dopamine is increasingly inhibited.

When the emergency is over, the heart rate (the average beat rate) slows down, and the heart rate variability patterns become more coherent. Brain wave patterns may again become resonant with heart patterns. Adrenaline levels climb down. Dopamine can be released accordingly.

When the stress or the emergency comes to a close, perceptions of physical and emotional pain, if any, become once again available. These perceptions are accessed via dopamine. An emotionally healthy person resumes, via dopamine, the ability to playfully imagine and visualize, and to anticipate purely happy outcomes. His ability to *feel* physical and emotional input regarding one's own sensory experiences, either negative or positive, a *heart-based ability*, returns.

However, while a person is emotionally inhibited to the extent that he is selectively dissociated from his heart, he will not be able to access dopamine.

Even when *wave patterns are not in sync*, the *nerves remain connected*

During times of fear or stress, the heart-*nerves*, either via the spine or vagus nerves, remain connected to the brain – unlike the heart-brain *wave* entrainment, which disconnects. Whether scared *or* happy, waking or sleeping, these nerve signals continue to tell the brain how the heart is feeling (resonant or not), and how much.

We have not yet discussed the *quantity*, the *size*, of the heart signals. Briefly, the *amount* of signal getting to the brain from the heart-nerves appears to determine the *degree* to which neurotransmitters are released.

We hypothesize that the size of these heart-nerve signals (the quantity, the “how much”) that the heart continues to send to the brain indicates the *level* of emotional energy that is available at the moment.

Whether the brain is using dopamine or adrenaline, whether the brain and heart waves are entrained or not, the amount, the size, the “how much” of the electrical signals that travel from the heart-nerves to the brain seems to determine how *much* of a response the brain can muster: how *much* adrenaline or dopamine can be put into play.

Based on SPECT scans that show the decline in heart-nerve receptor activity in PDers, the ongoing research in neurocardiology, and the perceptible heart changes that occur in people who recover from Parkinson's disease, the rest of this chapter hypothesizes a new understanding

of heart-nerve connectivity and heart-brain non-entrainment that is consistent with the changes that occur during Parkinson's disease and during recovery from Parkinson's disease.¹

The decision to disconnect the wave patterns is made by the brain: a hypothesis

At a certain level of danger, negative thinking, anxiety – or in the situations that are met with a dissociation response – the brain wave patterns disconnect from their entrainment with the heart wave patterns. Based on our own research, this wave pattern disconnect is a brain-based decision, and not a heart-based phenomenon.²

However, with regard to the heart-nerve's sympathetic and vagus nervous system signals, the heart is ever sending electrical signals to the brain.

In an emotionally healthy person, the heart-nerve's signals to the brain are never turned off. The heart cheerfully sends information and energy to the brain, whether the brain is bouncing around in a panic or calmly enjoying the situation. If the heart is *not* electromagnetically resonating with inner and outer experiences, it favors the spinal nerves, the ones that stimulate the sympathetic nervous system. If the heart is resonating with ongoing events, it favors the vagus nerve, the parasympathetic connection.

Notice that I said the *emotionally healthy* heart continues to send nerve information to the brain even when the brain works itself into a dither and disconnects its wave patterns from those of the heart.

¹ The following articles discuss the discovery that the dopamine receptors in the heart have significantly diminished activity in people with Parkinson's disease: Goldstein et al, "Cardiac Sympathetic Denervation in Parkinson's Disease," *Annals of Internal Medicine*, Vol. 133, No. 5, Sept 5, 2000, pp. 338-347 and Kaufman, Horatio. "Primary Autonomic Failure: Three Clinical Presentations of One Disease?" *Annals of Internal Medicine*, Vol. 133, No. 5, 2000, pp. 382-384.

² The researchers at the Heartmath Institute have conjectured that heart-brain entrainment automatically occurs anytime the heart rate variability is somewhat calm and coherent, because they see this in a majority – but not all – of their subjects. Our research on PDers conflicts with this hypothesis.

Most of our PD patients do *not* experience the type of contentment that is associated with heart-brain entrainment even if they have devoted their lives to meditation and inner calm. We have worked with PDers who have, for decades, practiced daily meditation, including breath and heart-rate control. They can create in themselves conditions of extremely slow heart and breathing rate. However, they cannot register the positive feelings associated with heart-brain entrainment. For that matter, most of our partially recovered PDers could not perform, and in many cases could not even comprehend, the very very simple exercises that the Heartmath Institute has developed to induce heart-brain entrainment.

When PDers use *mental* games described later in this book, games in which they *mentally* pretend that their hearts are *extremely* blocked, and utterly *incapable* of *any* sensory feeling, their sensory perceptions seem "normal." When, immediately following, they pretend to remove the extreme blockages from the heart, they often perceive a flood healthy sensory perception. They are usually surprised at the extent to which their PDish idea of "normal" was actually highly inhibited. Even more telling, when they do this experiment, they often feel, instantaneously, a joy and contentment that has been long absent from their lives. The condition of joy is fleeting; it does not last: they have not yet permanently removed the "dissociate from the heart" command from their brains. However, this exercise can be helpful. It demonstrates to the stubborn PDer that, in fact, his "normal" condition is grossly inhibited and joyless, and that it can be changed. Even so, many PDers respond to the discoveries of this exercise with a "So what?" and no determination to change the dissociated status quo.

Therefore, we suspect that decisions made by the *brain*, and not merely a lack of agitation in the heart, can determine whether or not heart-brain entrainment can actually occur. Of course, in emotionally healthy people, the brain is quickly subservient to the heart; the heart's energetic field is much larger than that of the brain. But in people with mental/emotional blockages such as a long-running selective dissociation from the heart, the rogue brain may be operating under its own commands, commands that specifically deny access to the heart.

The emotionally healthy heart is like the loving mother who humors her child with unconditional love and support even when the brilliant child indulges in unnecessary panics over upcoming college-entrance board exams. The heart's love is always sending nerve signals to the brain, humoring the brain, enjoying its little eccentricities. It is the brain, the home of the ego – the source of fear – that disconnects its *wave* properties from the heart *wave* patterns when the going gets tough. The emotionally healthy heart, via *nerve signals*, remains ever true.¹

Decrease in the amount of heart-nerve signal

The *amount* of the heart's nerve signals to the brain may begin to diminish at some point. This decline may occur when overall health of the body is decreasing or when the heart has begun to lose interest in life. Sometimes, when the sheer joy of living decreases abruptly, as can happen, for example, when a long-term spouse dies, the signals from the heart may abruptly become significantly diminished. The remaining spouse may soon die.

Based on Asian medical theory, when the *amount* of heart-nerve signals declines, when the joy of living decreases, the *capacity* for life also declines.² When the amount of heart nerve

¹ In a fearless person, one who has utterly surrendered his life over to the Love, Wisdom and Fairness that permeates the cosmos, the heart and brain wave disconnect does not occur.

The saints who play, childlike, with cobras or tigers, or who go graciously, fearlessly to their death, *never* permit their minds to dash off into a condition of adrenaline-dominance. The men and women that have conquered control of the mind are able to remain always in a condition of heart and brain resonance, with the heart guiding the brain. Only those whose minds are still governed by their egos are susceptible to the disconnection with the heart waves that the brain initiates when it imagines itself to be in danger.

The soul is never in danger: as the Vedas put it, “No fire can burn it; no wave can drown it.” The soul needs no adrenaline-releasing brain-wave disconnect in order to feel safe. The ego, being a temporary, false construct, is always on the lookout for anything that threatens its position. The ego-led mind may eagerly descend into panic at the first sign of irregularity. When it does, it disconnects from the heart and thus gets to be “in charge” for a while. In PDers, the brain is almost *always* in charge.

Most PDers we've known honestly do not believe that the universe is “Fair.” Essentially, because they cannot see all the subtle workings of the laws of cause and effect, they do not believe in cause and effect: Fairness. In this sense, these PDers are the supreme irrationalists. And ironically, so many of them imagine themselves to be more rational than most.

² The joy of the *soul* never decreases. However, the joy associated with *living* a particular role in a particular body may wane in response to circumstances, such as aging or emotional loss. According to Asian medical theory, the Heart is the source of joy. According to Asian (Vedic) theory, after subtly vibrating Divine Energy enters the body at the back of the neck (the “Mouth of God”), it goes in two directions: the brain and the heart. Energy is *stored* in the brain, and as much as is needed at any given moment *manifests* physically (converts into the heavier, “denser” waves of electromagnetic energy that becomes the electrical functions of the heart). The *physical* heart is formed by further condensations or “densifications” of the heart's electrical energy as it plays against the DNA of the heart cells. The heart is the first “manifestation” of the extremely subtle waves of Love that energize the body. After energy manifests as heart, the heart then directs the energy throughout the rest of the body based on the vibrations received by the heart. Hence, Heart is the most direct “source” for joy in a living system.

This understanding of living systems is perfectly aligned with the findings of modern physics. When the underlying *philosophies* of Eastern and Western (“modern”) medicine are compared, Western medicine, with its emphasis on the crudest results of energy (instead of on the source of energy and the influences that direct that energy), comes across as archaic. To a student of modern physics, the so-called “researchers” of Western medicine, the majority of whom fail to integrate modern chemistry and physics into their work, often resemble the people living in Plato's dark cave.

In Asian medicine, anxiety is the result of Heart insufficiency. When energy levels decline in the heart, the strength of function of other organs, including the kidney and its adjacent adrenal gland (a major source of adrenaline) also declines.

signals decline, the potential levels of release of the two main neurotransmitters, dopamine and adrenaline, diminish. Diminished release of dopamine or adrenaline results in physical and emotional slowness, depression and/or anxiety.

Depression from an insufficient heart-nerve signal

As the *amount* of heart-nerve signals declines, so that the *amount* of dopamine release declines, depression can ensue. In this case, the heart and brain waves *may be* in sync, but because of a diminished amount of heart-nerve signals to the brain, there is not *enough* dopamine release to trigger responses to sensory and thought stimuli. A person in this condition may look at the bright blue sky or the beauty of a rose and have a minimal or not detectable response.

When the heart's electromagnetic field is diminished, the amount of heart-nerve signal going to the brain is diminished, and so the amount of dopamine released by the brain is diminished. The emotional capacity for response is diminished.¹

Anxiety from an insufficient heart-nerve signal

Even if the *amount* of heart-nerve signals declines, fear-inducing situations *can still* cause a loss of entrainment between the heart and brain wave patterns. However, if the size of the heart-nerve signals is diminished, the brain has a correspondingly diminished capacity for mounting its adrenaline response even though the heart and brain waves patterns become disconnected. If the amount of heart nerve signals decline, then when the brain shifts to sympathetic (fear) mode, the mind may only be able to create an impotent anxiety response because of an insufficient *level* of adrenaline to rally the body to action.

In anxiety, negativity and fear-based thinking dominate the brain; the heart and brain wave patterns are not in sync: the brain is disconnected from heart feelings. But in some cases of anxiety, the *level* of adrenaline release is diminished. This insufficiency of adrenaline may occur if the amount of heart signal is insufficient.

The lowered level of adrenaline release is not large enough to stir the body to action. The fear whirls pointlessly around in the head, but no actions are taken to battle the source of the fear.

¹ Keep in mind that there are two problematic facets of the mental/emotional blockage of Parkinson's. The first, the dissociation of the heart that dis-entrains the heart and brain, is set in motion at the time of the fear event that precipitates the heart disconnect. This event may snowball in the brain to include increasing numbers of mental arenas from which the heart must be dissociated. The decrease in heart-nerve signals, however, is a slow development. Like the dopamine-producing cells in the brain that become dormant from minimal use, the heart-nerve dormancy most likely also develops slowly, in response to minimal use. This can explain why the personality of Parkinson's might emerge at a very young age: at the time of the initial heart-brain disconnect. But the mood-related symptoms of Parkinson's, the steadily worsening anxiety and depression, and the increasing inability to rouse oneself to adrenaline-based movement, appear slowly, over time, as a result of the gradual decline in numbers of active heart-nerve.

Again, not all PDers have the same level of mental/emotional blockage. Some PDers *do* seem to have the capacity for heart-brain resonance. Some actively cultivate it even though their body is actively inhibiting dopamine because of their injury. Their emotional decline during Parkinson's heads more towards depression than towards anxiety. In our limited experience, these "open-hearted" PDers whose emotions are merely becoming flattened recover more easily than those who are consciously wary and those who want to justify their wariness.

Anxiety is the name of this condition, in which fear dominates the mind *but* the body is not able to mount a big enough response to either challenge the threat or rein in the negative thinking.¹

The *amount* of heart-nerve signal determines the *quantity* of mental and chemical response that the body can produce. The mind, while able to produce a fear or a happiness campaign by being either disconnected or connected, respectively, to the heart's wave patterns, does not ultimately control the amount of energy available to that campaign. The *amount* of *heart* involvement, sent via the heart nerves, may be the key determinant for how *much* of a response the body can produce.

Dopamine and the heart

The heart is always fine-tuning its dopamine/adrenaline balance. Both adrenaline and dopamine are always in use in the heart. Every microsecond, in response to thoughts and to internal and external sensory perceptions, the heart is moving slightly more towards one nerve set and its neurotransmitter or towards the other. The degree and manner of heart wave resonance with thoughts and with internal and external sensory perception determines the moment-to-moment balance between adrenaline and dopamine. If the heart is more resonant, the neurotransmitter balance shifts more towards dopamine. If the heart is less resonant or emotionally shut down, the neurotransmitter blend shifts more towards adrenaline.

The ratio of adrenaline to dopamine at any given second determines *how* the brain will interpret the incoming sensory information at that moment, and the manner in which the brain will respond.

Up until now, I've only mentioned dopamine as a paired neurotransmitter with adrenaline. In fact, dopamine is not just the "opposite" of adrenaline. Dopamine is the main driver of the heart. If the brain perceives a reason to be fearful, the heart's dopamine triggers adrenaline and a tilt towards the *sympathetic* nervous system's connection to the brain. If the brain is not fearful, the heart's basic dopamine supply triggers more dopamine and a tilt towards the *parasympathetic* nervous system's connection to the brain.²

Dopamine is the primary activator of the heart. Dopamine levels in the heart determine the vigor of the neural signals to the brain. Dopamine levels in the heart are determined by the amount of joy and the amount of resonance that the heart is feeling.

The sheer joy of being alive is the energy that allows the heart to resonate and initiate the primary dopamine release for the heart.

Dopamine does not cause joy. Joy causes the release of dopamine. The greater the joy, the greater the level of primary dopamine in the heart. Whether a person is happy or sad, he can always resonate with the sheer joy of being alive. Whether a person is in the midst of battle or in solitude, the sheer joy of living can be present behind his fear or his tranquility. Joy and the heart's ability to resonate are very nearly the same. The former is more purely energetic, the other is the more physical manifestation of the joy energy.

¹ In Asian medicine, insufficiency of Heart Qi has long been considered the cause of anxiety. It's fun for me to see how modern research is starting to confirm the ancient sciences.

² Humans need to always maintain some level of fear: this minimal level of fear is needed to stimulate breathing and a heartbeat. Of course, an advanced soul who is utterly fearless may choose to still his heart and lungs.

Just like light, which has a wave pattern and a photon, human joy has a purely vibratory component and a more tangible component. Just as the astral form of light does not require a photon, the vibratory component of joy exists whether the body exists or not.

For example, light has two components: the light “wave” and the photon. The wave and the photon are considered to be equal and simultaneous, in terms of energy, but the photon is the denser, more tangible, more “crude” half of the combo. Like light’s relatively more tangible half, the photon, the electromagnetic wave of the resonating heart is the denser, more crude, more tangible component of joy.

When the heart is resonating with the joy of being alive, it releases dopamine to itself. That dopamine then energizes the other heart responses. This underlying source of dopamine is what powers the heart’s balancing act between the dopamine and adrenaline that flows to the brain.

The core dopamine in the heart drives the dopamine and adrenaline systems in the rest of the body. The dopamine stashes in the head, in the substantia area and other parts of the brain, are merely satellite supplies of dopamine. They are activated and dopamine is released into various parts of the brain, when the heart instructs the brain to respond to sensory events with joy. The core level of dopamine prepares a person, in body and brain, to be a feeling, sentient being.

The heart and the dissociation response

This core level of heart dopamine is only diminished when a person ceases to feel the sheer joy of being alive or while dissociating from his heart. Also, the core level of dopamine diminishes when a person prepares to die.

The dissociation response shuts down the ability to *feel*. The dissociation response prepares an animal for death. In PDers, heart SPECT scans show that dopamine receptor activity is significantly diminished. PDers have trouble feeling. We might say that some PDers have spent their life fending off death or getting ready to die.¹

¹ By thinking about the dissociation response as a preparation for death, a curiosity of recovery suddenly made sense to me. We noticed from the earliest days of the project that, shortly after recovering, many recovered PDers used the unlikely phrase: “So what? It’s not like anyone is going to die.”

The first time I heard the phrase, a recovered PDer was telling me why she decided to stay at the beach instead of coming to her acupuncture appointment. She had never before chosen to brush off a responsibility. But as she had sat on the beach enjoying the sea gulls, it suddenly occurred to her, for the first time in her life, that she could choose to be irresponsible for once. “So what?” she had asked herself. “It’s not like anyone is going to die if I miss the appointment.”

Another time, a recently recovered PDer told me that she’d had the astonishing realization that, as director of a show, she need only take responsibility for her own job. If any of the actors, singers or musicians failed to do their personal best, the show might be a little less good, but “So what? It’s not like anyone is going to die.”

Other PDers also felt the enormous weight of the world sliding off their shoulders as they suddenly saw their life roles in realistic perspectives for the first time. A not uncommon way for recovered PDers to express this new wisdom was “So what? It’s not like anyone’s going to die!”

It seemed as if these easily-recovered PDers were suddenly able to stop using their mind in a manner that suggested everything they did had a life or death consequence. When their foot injuries healed, they noticed shifts in perceptions and behaviors. To me, the most curious commonality was the vocalized realization: “So what? No one is going to die!” Only after I learned that the dissociation response is a preparation for death did the “No one is going to die!” epiphanies begin to make sense. These PDers, prior to recovery, had dissociated from themselves or parts of themselves. Therefore, beyond all logic, they necessarily had thought patterns and heart-mind separation patterns characteristic of those of a person facing imminent death.



*Since I left You, mine eye is in my mind; And that which governs me to go about
Doth part his function, and is partly blind, seems seeing, but effectually is out; For
it no form delivers to the heart...*

Shakespeare's Sonnet CXIII

CHAPTER TWENTY-SEVEN

MORE HEART MATTERS

DEFINING "FEELING"

The word "feeling" has many meanings. To continue in the discussion of "Heart," I must make it clear which one I'm using: when I say "feeling," "feelings," and "felt," I'm referring to sensory and heart-wave input to the brain. For example, input from the nerves of smell, taste, vision, hearing, or touch brings sensory input to the brain. This input is *felt*. The five types of sensory nerves impart *feelings* to the brain.

The sixth type of feeling, or "sixth sense," is emotional and intuitional feeling. Examples of this type of feeling are the expansion or tightening in the chest in response to an increase or decrease, respectively, in resonance with sensory or thought experiences. The names we give to these emotions are love, compassion, joy, grief, and fearlessness, to name a few. These are purely sensory *feelings*, not thought processes. They are not related to ego.

Emotionalism

Ego-based thought processes can roil in the mind to create conditions of jealousy, greed, self-pity, negative criticism, disappointment, anxiety, and all the other types of negative thinking. These are types of emotionalism. I am *not* using the word "feeling" to refer to emotionalism, even though that can be one of the standard English meanings of the word "feeling."

Emotionalism *begins* in the mind – it does not originate directly from sensory input. All of these thought-based conditions come from the ego. As ego combines with various thoughts of satisfaction or dissatisfaction of one sort or another, emotionalism arises. Emotionalism does not arise directly from the six senses. Emotionalism is ego-based *interpretations* of the brain's information: the *results* of mental manipulations.

Emotionalism *can* be related to sensory feeling, inasmuch as emotionalism can be triggered when sensory feeling is combined with ego-based thinking. For example, *seeing* the expensive car of a business competitor can trigger jealousy. In this case, the sensory input of vision goes to the brain where the *ego* processes the visual information about the car and creates jealousy.

In another example, the smell of a remembered perfume can combine with the ego and trigger thoughts of bitterness (Why did she leave me for that jerk?) or lust (My sexual desire is being stimulated!). Jealousy, lust and bitterness are examples of emotionalism, not basic *feeling*.

The smell of familiar perfume is an olfactory *feeling*. A heart-based response to the perfume will resonate with the perfume smell and accept it for what it is: a flowery smell, a musky smell, or a spicy smell. The heart-based response can simply enjoy, or not, the feeling of the smell.

A mind-based response to the smell will reflect the desires of the ego: "I'm still angry at her," or "I'm wild with desire!"

Emotionalism is not sensory feeling. Emotionalism is ego and thought habits *responding* to feeling, responding to sensory information or to a train of thought.

In this book, as I use the word “feeling,” be aware that I am talking about the incoming sensations from the six senses. I am *not* talking about emotionalism.

The sense of touch

The sense of touch is the one that is extremely inhibited during automatic dissociation. Some people are understandably misguided by the use of the word “touch” in describing this sense. They imagine that it refers only to the sensations that occur when a person’s reaches out to “touch” another surface. But the sense of touch refers to much more than that. A better way of describing the sense of “touch” might be to call it the sense of “everything that isn’t a smell, taste, sight, or sound.” But that would be too wordy. The sense of touch includes the ability to know where a body part is even if the eyes are closed and cannot see the body part (proprioception). The sense of touch includes the ability to feel pain, warmth, or vitality inside the body or on the surface of the body (internal and external physical awareness).

The sixth sense

The “sense” of emotion can register the sensory *feeling* in the heart that arises from vibrations of love, compassion, gratitude, contentment, joy and intuitive knowledge and “hunches.” When we “feel” loving thoughts being directed to us from across the room and the chest expands a bit from that “feeling,” we are using the sixth sense: pure heart resonance.

PDers and the inhibition of feeling

When I write about PDers being numb, I am referring primarily to their senses of touch and their sixth sense, even though their sense of sight, sound, taste and smell are also altered and somewhat inhibited. When I speak of feeling, in general, I am referring to all six forms of sensory feeling. All of these forms of feeling are conveyed to the brain from the five types of sensory nerves and the heart-nerves. The brain receives the input. The brain *interprets* the information based on the *amount* of signals from the heart-nerves (which is based on the amount of joy-of-living that the heart is feeling at that moment), *and* on whether or not the heart is using predominantly the sympathetic nerves or the parasympathetic nerves, *and* the extent to which brain wave patterns are *entrained* with the heart wave patterns at that moment.

After the first experience of a particular sensory input, the brain *may* interpret subsequent experiences of the same sensory input based on ongoing heart patterns, but it may also interpret the experiences based on previous experience: habit. For example, in the music-recognition pattern described earlier in the chapter, the sound of an old favorite song may generate a heart response based on previous heart experiences that were occurring at the time the song was first heard.

I am not actually wandering from the point of Parkinson’s. The manner in which the brain uses habit to build on fear-based experiences, until, in some people, including some PDers, the entire brain is linked up to a habit of fear, is related to this process.¹

¹ Post Traumatic Stress Disorder is related to this phenomenon, in which a terrible fear links up with more and more brain regions, and the habit of visiting these fears becomes increasingly entrenched.

HEART-NERVES AND HEART RESONANCE

I am proposing, based on Asian medicine principles, Vedic teachings, and our experiences with PDer's during recovery, the following: in times of safety, the which heart-nerve system is used, the vagus nerve or the spinal nerve, is the primary determinant of whether the incoming information is perceived as good (resonant) or not, and to what degree.

If a person feels safe, he primarily uses the vagus nerve to send and receive signals to and from the brain. If a person is wary, frightened, or dissociated, he uses primarily the heart's spinal nerve to send and receive signals to and from the brain.

When a person feels safe

If the brain is in sync with the heart, the *vagus* nerve signals from the heart influence the brain to *interpret* and *act on* all other incoming sensory information at that moment with a positive response and memory. The information from the vagus nerves shares information about both the *type* of heart response (on a 3-D sliding scale from glorious to mundane, based on resonance) and the *quantity* of heart response (a lot or a little).

The brain notes whether the heart is declaring "highly resonant," "not resonant" or somewhere in between, and also notes "how much." The brain then responds to internal and external sensory information accordingly.

In times of safety, the heart-nerves tell the brain what is going on, and the brain interprets sensations and responds to them in a positive manner.

When a person doesn't feel safe

When a person perceives danger and/or he experiences automatic dissociation or selective dissociation from the heart, the brain disrupts the resonance of heart and brain wave patterns. The heart and brain roles are reversed: the brain tells the heart what to do.

In times of potential danger, difficulty, or dissociation, the brain sends signals to and from the heart using, predominantly, the spinal nerves (instead of the vagus nerves). The brain then interprets sensory information directly from the five senses – instead of running all sensory information past the heart's "interpretive" center. The brain interprets sensory information using adrenaline-based thinking.

This sympathetic nervous system type of response to sensory input is extremely fast. It is very efficient. Of more concern to the person with Parkinson's, adrenaline-based motor responses and adrenaline-based *thinking* do not register or involve the full spectrum of *feeling* or emotion, nor does it allow for the full spectrum of imagination or visualization. In particular, the ability to feel sensory awareness of the insides of one's body is inhibited when a person is sympathetic mode dominant.

(As noted earlier, most people are usually using a *blend* of sympathetic and parasympathetic responses. All breathing requires use of the sympathetic system. Digestion and relaxation require the use of the parasympathetic. Most people are always somewhere in between pure sympathetic and pure parasympathetic. Based on a person's thoughts or feelings at any given moment, people move closer to one side or the other of the continuum between these two conditions.)

As mentioned earlier in the "chased by a ravening lion" example, *feelings* of physical or emotional pain get in the way when a person is in danger. Conveniently, these distracting types of sensory perception are not accessible when adrenaline-based thinking is dominant. When the brain decides that danger is dominant, the heart and brain waves patterns are not entrained and a

person can't feel the insides of his own body or his sixth sense. And as noted already, the areas of the brain that allow for playful imagination, visualization, and creating positive outcome scenarios are also not accessible when adrenaline-based thinking is ongoing. In times of high danger, a person thinks. He doesn't feel his own body very well, nor does he feel the *any* of the senses via his heart's resonance.

An example: feeling the sea lions

Let me share a quick story that might demonstrate the differences that occur in sensory awareness, in *feeling*, when one is adrenaline-dominant (heart and brain wave patterns disconnected) and when one is dopamine-dominant (heart and brain wave patterns in sync, the parasympathetic system dominating the sympathetic system).

A deeply thoughtful and considerate PD patient, who felt himself to be a person of great sensitivity to others and therefore full of feelings, told me (I paraphrase), "I'm finally willing to admit that maybe I am a bit disconnected from my feelings.

"Last night I couldn't sleep, so my wife suggested that I remember how I'd felt earlier that day at the ocean-side cliffs, as we watched the sea lions out on their rock. So I thought about what I'd noticed about the sea lions: how many there were; how they used flippers like legs as they climbed up out of the water; how the bigger ones bullied the smaller ones for position; how the ocean, the sea lions, the rock and the clouds formed a harmonious picture. None of these things seemed to bring me closer to dozing, so I asked my wife what she had noticed about the sea lions that would be helpful in falling asleep.

"She replied that she could remember the gentle rolling sensation flowing back and forth in her chest in time with the ocean waves, the letting-go of her worries. As she *felt* what the sea lions must have felt as they surrendered their weight to the perfect support of the ocean swells, she felt a sense of surrender to the universe. She felt an expansion in her heart from the warmth and relaxation they projected as they basked in the sun. She felt, in sync with the sea lions, a swelling in the chest from their sheer joy-of-being as they slapped fins and roared at each other.

The PDer continued, "Everything that I remembered had to do with numbers, colors, shapes or actions: things that I could think about, in words. Everything that my wife remembered had to do with how her heart and body had felt the sensations of the sun, the ocean's support, and the joy projected by the sea lions. We'd spent half an hour in the same place and we had no actual experiences in common."

This PDer's "feelings" from his day at the beach are typical of those of many a PDer; and they aren't actually feelings: they are thoughts. His wife's feelings were dopamine-based – and they were, in fact, feelings: not processed emotions and thoughts, but *feelings*.¹

¹ Flying completely off the point, I'll mention that, as in this example, most of my married PD patients have spouses who are temperamental opposites. I suspect that only rarely would two adrenaline-dominant people be able to find a lasting marriage formula. However, I do know of one couple in which both parties developed Parkinson's disease. They were still temperamental opposites: he was a "slow mover," artistic, and he was drifting into worsening depression. She was wary, hyper-alert and always on the go, with crisp math and organization skills, and she was drifting into increased anxiety. He recovered very easily, and experienced no surprising changes as his heart became more resonant, except for an increased interest in physical fitness. She also recovered easily, but noticed many personality changes, including an "opening" of the heart, changes in sensory awareness, a new ability to experience calm, and a letting go of most of her exacting mental priorities.

Another example: hearing the birds

One PDer told me, prior to his recovery, that he no longer knew the meaning of the word “joy.” Recently, several years after fully recovering, he told me that during a morning meditation on his balcony, with the late-winter sun streaming down on him, a returning flock of birds in a nearby tree suddenly burst into celebratory song. “I felt my heart expanding.” He gestured with his arms making a big circle, his hands a good foot out in front of his chest. He continued, “The feeling was so intense, I almost cried.”

His “heart expanding” was able to occur because he once again had a functional heart-mind relationship. His heart was capable of feeling, and was capable of transmitting awareness of feelings to his mind.

Just to be on the safe side, I’ll be redundant yet again: when I speak of using the heart to feel, I am talking about the way the heart expands or not in response to internal or external sensory and thought input. After the initial event heart resonance, the heart response, or lack of it, then influences subsequent thoughts, moods, emotions, and motor functionality.

In this example, the feeling of joy he experienced from hearing the birds was not based on thoughts: just the reverse; he heard the birds and his heart resonated with the sound; his subsequent thoughts were flavored by his heart’s experience of joy, the heart’s resonance with something beautiful or harmonious.¹

The “closed heart”

A person whose “heart is closed” to his own physical and emotional pain does not perceive his other feelings as well, either. For example, many PDers report that colors seem brighter after recovery. Environmental sounds are more pleasing, less irritating, after recovery. The recovered PDer spontaneously sees changing playful images in floating clouds and faces in the leaves of trees, even though he may have lived decades, or even most of his life, without having been *able* to behold these most basic imaginings.

After recovery, a PDer responds to sensory experiences as if his brain wave patterns were entrained with his heart wave pattern. And these sensory experiences are clearly different, much richer and fuller, than what he’d experienced in the past. Therefore, we can conclude that, while having Parkinson’s (and in many cases, for decades prior to his diagnosis), his brain wave patterns were probably *not* entrained with his heart wave patterns: he had been primarily in sympathetic, or even dissociated, mode.

¹ We say that the heart is “heavy” when sad, “stuck in the throat” when fearful, “jumps for joy” when excited, “breaks in two” when sundered by fate from a loved one, “palpitates” with worry, or “swells” with joy or justifiable pride. It is “closed off” when sullen feelings of resentment, anger, self-pity, or fear predominate. These heart conditions, or heart “feelings,” affect the information being processed by the brain at that time.

An emotionally healthy person will sometimes shut down the nattering mind and float along with his imagination or simply enjoy his feelings. His heart is *always* wide open. Conversely, a person with advancing Parkinson’s typically keeps his heart completely or partially shut down. If he tends towards depression, he may feel as if he is becoming lifeless. If he tends towards anxiety, he rarely, if ever, shuts down his restless or anxious inner monologue.

TWO TYPES OF MOVEMENT: DOPAMINE-BASED AND ADRENALINE-BASED

In case the PDer is wondering where all this is going, I'm about to bring it back to the processes that most PDers are worried about: motor function and neurotransmitters.

Dopamine-based movement

Spontaneous, easy motor function actually *combines* motor and mental processes. Playful pretending, unselfconscious dancing, eating relaxedly and *all* forms of dopamine-based movement are activities in which a person effortlessly transforms his *idea* of movement, his *mental image* of movement, into the manifestation of movement. The *idea* is triggered by dopamine. Acetylcholine, another neurotransmitter, then activates the rest of the nerves in the nerve chain, including the nerves that go into the muscles, causing healthy muscle contraction.¹

In the split second before a healthy person activates a dopamine-based movement, he imagines himself performing the activity. The imagination then activates the motor function. A healthy ability to visualize is driven by dopamine. The ability to *visualize* or *imagine* oneself moving is a crucial step in dopamine-based motor function.

In healthy people, the transition from mental image to motor response is so quick that most people never know they are doing it. Some people, such as dancers and athletes, who need to move even “more so” than most people, know very well that what they are trying to do is use their bodies in such a way as to most perfectly express the *idea* of movements that they are mentally picturing or feeling.²

Dopamine-based movement is relatively effortless, and it originates in the positive imagination. This imagination is only available when the heart and brain wave patterns are in sync.³

¹ Neurologists know perfectly well that acetylcholine, and not dopamine, activates the muscles. However, they often tell people with Parkinson's that their *muscles* don't work because of a dopamine deficiency. Evidently, some doctors conveniently ignore everything they've learned about muscle function when confronted with the evidence that PDers can move easily when under the influence of dopamine – a powerful *mind*-altering drug. They then tell their patients that dopamine causes movement – even though most doctors learned in *high school* that acetylcholine is actually the neurotransmitter that activates the muscles.

² Trainers for top athletes and dancers have found that a performer can improve his game or art *more* by spending hours *imagining* himself moving better than he can by actually *practicing* the physical movements. They have learned that the two most important factors in improving performance are improving the ability to imagine the movement and refining the imagined movement; the importance of training of the muscles runs a distant third.

³ A person needn't always imagine every detail of movement. He *may* imagine a specific, single movement, or he may use a habit “shortcut” that accesses a motion-integration sequence that has been stashed in the “complex learned-movements” sector at the back of the brain (the cerebellum). Even shortcuts are either adrenaline activated or dopamine activated. Complex learned-movements created while using dopamine can only be activated by using the dopamine-based shortcut. Adrenaline-based complex learned movements are activated by an adrenaline-based shortcut.

Children who merrily practice emergency drills may have no ability to access those drill skills during an actual emergency; habits learned while using dopamine may not be accessible to the mind when the brain is in adrenaline mode.

For example, playing the violin is an activity that uses habits of highly complex, highly integrated movements. The following vignette suggests that the violinist with Parkinson's had two complete sets of integrated movements stored in her cerebellum. One was used for playing violin with adrenaline, one was used for playing with dopamine.

Secondarily, as a person moves, his body feels the internal sensations generated by movement. These feelings, when perceived by a brain that is in sync with the heart, are gratifying, enjoyable.

Consider the movements that a cat makes when he wakes up from his nap. He languorously stretches one muscle after another, enjoying the sensations generated by the use of his body. He does this because it feels good; the sensations generated thereby are pleasing.

This type of self-awareness of movement is dopamine-based. This type of self-awareness and feeling is only accessible to the brain when the heart and brain are entrained, and when adequate amounts of heart-nerve signals are making their way to the brain.

Adrenaline-based movement

Adrenaline-based movement is command-based movement. A person using adrenaline to run *or* to stand stock-still is mentally *telling* himself what to do. Whether the command is coming from an instinct center or from rational consciousness, adrenaline-based motor function is generated by brain commands to perform specific actions.

These mental commands can allow a person to “keep going when the going gets tough.” As one PDer told me, “I just force myself to keep putting one foot in front of the other.”

Another example of adrenaline-based movement is the instinctive behavior that allows the tiny prairie dog mother to defend her pup by attacking a large coyote. When using this type of instinctive movement, the “command form,” though non-verbal, is used to execute motor function. Fear-driven, adrenaline-based motor function, even when instinctive, uses a motor command system that *tells* the body what to do. This is somewhat the “opposite” of the dopamine-based system, in which movement is a form of self-expression, the expression of an imagined movement.

Adrenaline is the neurotransmitter that most people use in situations that require the “mind over matter” principle. A person in this situation tells himself, commands himself, consciously or from habit or instinct, to perform a certain activity, and the motor function obeys.¹

This violinist, a recovered PDer, taking up the violin again after not having played for nearly a decade, was astonished at the abrupt change in her playing that occurred after about two weeks of practice. She wrote the following: “Last night, I suddenly played the way I used to play when I was twenty, not the way that I played in my thirties and forties. The effortless expression of musical ideas and the absence of mental determination reminded me of how I played when I was in college. My bowing movements, in particular, were led by the music, instead of by me. There is no way I could have mentally directed myself to play that way. My adult son, with a degree in music and an excellent ear, was startled. He said lovingly, if somewhat tactlessly, “You actually *did* use to play the violin!” Although he had heard me playing professionally when I was in my forties, he had never heard me play when I played with my heart instead of my mind. I could feel the difference; he could hear it.”

Both of her styles of playing used effective cerebellar (complex learned-habit) movements that performed the correct motions. But the set that was activated by dopamine used different emotional motivations and slightly different, but *audibly* noticeable, movements.

¹ This usage is actually a perversion of the excellent “Mind over matter” principle. PDers use this principle with a grim determination, convinced that they can perform anything if they put their mind to it firmly enough. The steadily increasing paralysis of Parkinson’s, *despite* their increasingly frantic efforts to mentally command themselves to move, should drive home to them that their fear-based mind *cannot* conquer the physical body, or “matter.” The actual meaning of this principle is that matter, being created by Divine thought, or Mind, is therefore subordinate to Mind. Attunement with Divine Mind can therefore conquer any material problem because all of the universe, all of creation, is a construct of that Divine Mind.

In the adrenaline-based mind, the mind that is not entrained with the heart, self-awareness of movement results in emotionalism rather than feeling. For example, after an adrenaline-dominant athlete runs a race, he may feel a surge of pride in the accomplishments of his body. He may be excited by the motor stimulation, he may be exhilarated by his motor prowess. Or similarly, after completing his yoga practice, he may feel good because he has virtuously accomplished what he set out to do. But these emotions are based on his ego-based thoughts of self-approval or the thrill of increased oxygen. He may feel vitalized by his motor activities, but he is not necessarily *feeling* his motor sensations. When I speak of feeling one's motor actions, I am thinking of the way that the cat feels pleasure in his own heart's resonance with his physical stretching movements. When a person is mentally or egoistically stimulated by his use of motor functions, this is not necessarily *sensory* feeling of the body – this is stimulation of the ego.

A quick aside to consider waking up in the morning

Although many PDers cannot believe it, some people wake up in the morning and languorously stretch, just like a cat. As these people come to consciousness after a night's sleep, they go through a progression of mental states. First, they realize that they *are*; then they realize *who* they are. Next, they notice the sensations of the body. They may notice the feeling of the sheets' texture on their feet. They may notice the feeling of the cool morning air on the face. As they slowly check in with all their body parts, enjoying the sensations of being alive and awake, they tentatively move and stretch to create and enjoy the feeling of life returning to the relaxed limbs. As the limbs move, the contented heart notices the pleasant feelings generated thereby. The heart swells as it resonates with the sensations being generated by movement of the body. It sends this pleasant information to the brain.

Twenty minutes later, while going through his morning toiletries, this person may wonder what day of the week it is, and start to think about his calendar for the day.

Compare this with the way that most of my PD patients describe their long-time wake-up routine. They return to wakeful consciousness and realize *who* they are. Next, they become aware of *where* they are. Their next thought often jolts the PDer into full alertness. The mental wording of the thought may be something along the lines of “*Uh oh! What day is it? And what am I supposed to be doing?!*”

Adrenaline-based sensory awareness

As another example of the difference in sensory perception when the brain waves are disconnected from the heart, many PDers have complained to me that they never felt any of the advertised happiness or joy from doing hatha (physical) yoga, Tai Ji Chuan, or Qi Gong exercises. This is because they were mentally focused on the positions and competency of their

Fear, according to eastern thought, is the child of material creation, not the parent. If one is focused on matter, one fears the changes and dissolution of matter that are inevitable in the temporary, illusory constructs of time and the atom. (Twenty-first century physics confirms that atoms are not “stable building blocks.” Their subatomic bits are constantly moving in and out of existence, changing from energy into matter and then back again to pure energy.)

Oppositely, attunement with timelessness and unchanging Truth, Love, and Divine Mind allows one to be fearless. Divine Mind can rule over matter. When one is locked into fear-based thinking, one cannot attain attunement with peace, joy, or Divine understanding. One *cannot* employ the *true* principle of Mind over matter if one is the slave of fear: when ruled by fear, one's mind is attuned with ego, and not Truth.

movements. They were unable to actually *feel* the pleasant increase in the heart's electromagnetic field that occurs naturally from resonating with one's own movement.¹

In a healthy person who is *not* using the sympathetic (danger) mode, the heart's energetic field resonates with the vibrations of one's own movement. The heart waves amplify, grow larger still, from their entrainment with the brain wave patterns that harmoniously "fit" with the heart resonance.

If the brain waves are in sync with the heart waves, the heart feeling amplifies as it resonates with the brain as the heart and brain together enjoy the sensory input from the pleasant movements of conscious movement: movement is *supposed* to be a rewarding sensory experience, not a command-based method for getting from one place to another. Animals do not ponder how to move; except during times of imminent danger, they move in the manner that is most rewarding – from a sensory standpoint. Animals do not get Parkinson's disease.

Leaving all the electromagnetics aside for a moment, one might say that the heart expands with increased joy-of-being when one is aware of his own existence and the movements generated by his conscious imaginings.

As the resonance amplifies the heart feeling, the expansion and relaxation in the chest allows more life force to flow through the heart. This further increases one's feelings of well-being and joy. This is the happiness or joy that is supposed to occur during hatha yoga, Tai Chi Chuan practice, or the sheer joy of self-expression through movement.

Understanding the relationship between *feeling* and heart field expansion, *joie de vivre*, and joy-based, not fear-based, will to live, can be crucial in understanding the mental/emotional blockage of Parkinson's. Combining that understanding with the information about the two types of movement, adrenaline-based and dopamine-based, can help PDer's when the time comes to re-open the heart.

Some PDer's have been strongly resistant to the idea that the connection between the heart and the brain might be involved in the movement inhibition of Parkinson's disease. To help support this idea, I will share some ancient Vedic theories.²

An Eastern understanding of the relationship between heart and mind

The Vedas (Hindu scriptures) explain how Universal Life Force energy is transmuted into the individualized energy that drives the human body. Life Force (*prana*) courses into the body at the back of the neck, in the vicinity of the medulla oblongata (in the brain stem, which is also near the area from which the vagus nerve emerges). Next, when energy flows from the midbrain

¹ Looking ahead, when we get these PDer's to stop pretending that their heart is small, dark, or whatever dismal construct they have invented, and pretend instead that their heart is healthy, they may suddenly experience a wave of positive feeling: if so, they often say that this is the first wave of truly positive feeling that they have had in their whole life – despite decades of yoga, Qi Gong, Tai Ji, or other "joy-inducing" modalities.

² I am often asked how we came up with our extremely simple treatments for turning off the sympathetic system. The treatments are based on a Vedic understanding of the Self-delusory nature of sympathetic system thinking. While it may seem that a section on Vedic philosophy is completely unnecessary in this chapter, the ancient theory led us to the very simple treatments that we eventually developed.

into the body, the heart is the first substation. The heart serves as a regulator for how much energy can flow into the rest of the body.¹

Practically speaking, the heart might be thought of as an amperage regulator. After the vibratory energy that pours into the body at the medulla is stored in the brain and converted into energy that is usable in the body, it flows into the heart before it goes anywhere else. From the heart, the energy is distributed to the five senses and the sixth sense. From these energies, the heart, the sensory organs and the brain are constructed and maintained. These become the instruments of feeling and self-awareness.

More philosophically, the condensation of universal energy into individual bits of energy is done by creating the principle of Individual Ego. From Individual Ego is derived *chitta* (individualized feeling, self-awareness).

The relationship between feeling and self-awareness

In order for a being to imagine that he exists as an individual entity, separate from the cosmic energy, he must have self-awareness.²

The basis of self-awareness is feeling. Via the information experienced through the five senses and the sixth sense of pure heart feeling, one knows that one is. Without the ability to feel, one cannot experience. One cannot know that one exists.

For adepts of Vedic philosophy, I'll mention that, even on the astral level of existence, in which tangible feelings do not exist in the same manner, the soul still has self-awareness, because he can still *feel*, perceive, the vibrations of sensations such as color and sound. Even on the causal plane, in which one exists as pure thought, with no body or even the astral vibratory energetics of a body, an individual still has self-awareness because he still *feels* a response to different thoughts (expansive feelings if the thoughts are attuned with Right, painful constrictive

¹ Not everyone can tolerate the same amounts of energy flowing into the heart and body. A saint has conditioned himself, either consciously, through meditation and spiritual practices, and/or somewhat unwittingly, through pure devotion, to handle vast amounts of energy flowing into the heart. Oppositely, one who is extremely self-centered draws in exactly enough energy to maintain his life, and no more. Even in English, this latter situation can be referred to as "the heart being closed off to others." The heart, in such a situation, is not actually closed, but it is restricted to the energetic needs of the individual.

The Hindu teachings warn that the spiritual novice cannot tolerate the high levels of energy that flow directly from the Source. Paramahansa Yogananda writes for the modern audience that unrestricted flow of divine energy into one's unprepared body and consciousness would be like running one hundred thousand watts through a forty-watt light bulb: the filament would burn up.

The energy flowing into the body is coming from an infinite Source that is rooted in Love. The capacity of the heart to admit that energy into the body is based on the degree to which an individual can surrender to the influx of that energy, surrender to that Love. The opposite of surrender is resistance. If too much energy flows through an area of high resistance, heat is generated. If too much love flows through the heart of a person who is resisting, he will literally burn up. The heart serves to regulate the amount of energy that flows into the system, based on how widely the door of the heart is held open. Our will and habits, combined, tell us how wide to open the door to the heart.

² Self-awareness, awareness that one is separate or distinct from the Infinite, in this philosophical understanding, is *not* a construct of consciousness. For example, when a person wakes up from sleeping, from the *subconscious* state, he can still tell you whether or not he slept well or poorly. His self-awareness, his idea that he exists separately from the Infinite, never ceases, whether he is using the subconscious (during sleep), conscious, or superconscious state.

feelings if thoughts are attuned with Wrong). These *feelings* give believability to the delusory experience of self-awareness.¹

To the saint of any culture, who may vocalize in his native language but whose speech is always directed by the language of his heart, the inherent connection between feeling and self-awareness is obvious. However, the English speaker who has been taught that feeling and self-awareness are two different words representing two different states of consciousness may be puzzled by the statement that feeling and self-awareness are the same thing.

They are. This point is very important. It is confusion about this principle that can lead to the development of Parkinson's disease. Confusion about this principle can contribute to a consciously-induced and maintained dissociation response, and to a condition of partial recovery from Parkinson's.

¹ The Sanskrit language is exceptionally refined. For example, it has over one thousand words, each with a slightly different meaning, for the different aspects of God. Therefore, I found it fascinating that, in a book I read long ago, the word *chitta* is translated into English using two words joined by a slash: feeling/self-awareness. This rather suggests that, in the Vedic tradition, feeling and self-awareness are one and the same. More terribly, it also suggests that, at least to the English-speaking population, self-awareness and feeling are two different things. Is it possible that some English speakers honestly think that they can have self-awareness even if they have no feeling? What a horrible thought.

Who would want to have awareness of self and, at the same time, have no ability to register heart-feeling? If all world scriptures speak truth, and all creation was built out of the vibrations of love, why would a person want to have self-awareness and simultaneously have no ability to feel those vibrations, feel the love out of which he was made: the love that permeates the universe, the love that knows itself by experiencing, feeling, itself? One pundit has said that God, infinite and eternal, made creation because only so could He *experience*, could He feel through sensory experience, his own ideas. When the Divine energy in a person's eyes beholds God's creation of Divine energy condensed into clouds and trees, God's energy is beholding, feeling, and resonating with God's energy. God is enjoying, through the delusion of his fleeting, ever-changing creation, his own ideas. To do this, he must use feeling and self-awareness.

(My editor asked me to reference the above definition of *chitta*. While digging for the book in which I'd long ago read the above definition of *chitta*, I found an even more detailed translation that amounts to the same thing: "Chitta is a comprehensive term for the aggregate of mind-stuff that produces intelligent consciousness, the power of feeling." This was in *God Speaks To Arjuna: The Bhagavad Gita: Royal Science of God Realization*, Paramahansa Yogananda, Self-Realization Fellowship, 1995, p. 472. This fuller description still makes the same point: intelligent consciousness, or self-awareness, is one and the same with feeling. If a person imagines otherwise, a delusory pathology – such as the one that causes Parkinson's disease – is in place.

Next, I picked up another book, *The Holy Science*, by Swami Sri Yukteswar (1855-1936), one of the most respected and analytical yogis of his day. In this book, defining "Chit" (chitta is the individual's feeling, Chit is the universal form of feeling), the author declared synonymity between Universal self-awareness and feeling, The Universal Heart, and Universal Love. In other words, Feeling, Self-awareness, Love, and Heart are the same.

Getting back to the point, the only times that a person loses his intuitive connection with God (Heart, Love) are when, because of fear, he disconnects his brain waves from his heart waves. In this condition, a person may still have self-awareness, but he will have this awareness through the warped interpretation that his fear-based mind (the *manas*) puts on all incoming sensory awareness. In this condition, a person may be aware that he exists, but he cannot be aware of his true Self, or soul. To have soul awareness, Self-awareness, a person must have feelings that are being interpreted correctly, via a mind that is attuned with the heart (the *Buddhi* Mind).

Going off on another tangent, it may be of etymological interest that, at least when I started this project, India had the lowest rate of Parkinson's per population in the world. The British Isles – which use two mutually exclusive ideas, "feeling" and "self-awareness," in an attempt to describe what is, in essence, one inseparable unity – has a relatively high rate of Parkinson's.

Getting back to recovery from Parkinson's disease

Partial recovery from Parkinson's occurs when a PDer's unhealed injury begins to heal but the PDer remains locked into dissociative mode for certain compartmentalized behaviors and thoughts. In some cases, if he decided that his body had "betrayed" him by developing Parkinson's disease, he may have dissociated from his *entire body* at the time of diagnosis.

When using the dissociative mode, a person has decreased sensory awareness of his physical body, decreased feelings.

And here's the kicker: due to reciprocity, refusal to recognize and embrace the feelings of one's body and emotions, refusal to allow a proprioceptive and internal-sensation relationship with one's own body, can keep one from feeling joy. A joyless life is not worth living: if life is not desired, the heart prepares for death. It does this by decreasing the core levels of dopamine in the heart. This creates the same type of decrease in core dopamine that is observed in a dissociation response.

This means that, by consciously refusing to once again embrace one's own feelings even though the injury event is over and done with, a person can remain locked into the dissociative mode.

In humans, a fleeting dissociation response might last for a few minutes. A severe dissociation response may last for several hours. A consciously induced and retained dissociation response, one that has been cultivated as emotional protection or a sign of "superior virtue or intelligence" may last for a lifetime.

The dissociation response is designed to make an animal feel and behave as if dead. The dissociation response *decreases* the core level of dopamine in the heart. The dissociation response numbs the animal in preparation for death.

Whether or not a PDer proclaims "I *want* to be alive! I *want* to recover!" does not matter. If he is selectively dissociating from his heart, his *body* will behave as if it is preparing to die. Even if the PDer's foot injury is physically healed and the PDer is physiologically capable once again of releasing brain dopamine to activate movement, he may not be able to activate the level of *heart* dopamine needed to activate the brain's dopamine centers.

In order to fully recover, the PDer must consciously turn off the compartmentalized response that allowed him to minimize his heart's recognition of physical and emotional pain when engaged in certain thoughts or activities. Otherwise, whenever he is engaged in those particular thoughts or activities that he has determined are not to be trusted, his physical condition will orient towards an increasingly death-like state of immobility.¹

¹ The question arises, "Why do dopamine-enhancing medications for Parkinson's have to get into the brain, and not just get to the heart?" In idiopathic Parkinson's, the most common drug, L-dopa, does not actually convert into dopamine until it gets into the brain. Remember, the PDer's heart is busily operating a sympathetic nervous system response. An increase in the underlying amount of core heart dopamine will only increase the amount of the sympathetic system response. Because of the underlying dissociation response, a "play dead" response, a PDer can only be stimulated to movement if the L-dopa converts into dopamine in the brain, *overriding* the heart's instructions to play dead by flooding the brain with movement-inducing dopamine. As and aside, it may be of interest to note that heart researchers, in order to measure the activity levels of dopamine receptors in the heart, use a G-dopa dopamine analog, and not an L-dopa dopamine analog.

Also, this entire essay is overly simplistic. The body has many types of dopamine receptors, and they all activate different functions. These receptors are activated or turned off by various thoughts and by heart instructions. Any discussion of heart versus brain responses must include the electromagnetic effects that heart and brain waves

Joy is the king; the heart is the throne of the king

In Chinese medicine, the heart is considered the most important organ. The heart is referred to as the King or as the home of Joy. In some cultures, it is also the access point for Love or the Divine Mother. The brain is merely one of the subordinate organs.

We can see this principle even in western medicine: we know that a person can be brain-dead but remain alive. But when the body can no longer support life, when the will can no longer support life, or in some cases, when the heart is “broken” from grief, the heart no longer sends sufficient signals to the brain. Death ensues. Even in modern English our language still supports this understanding of the heart. When we simply cannot bring ourselves to do something, we say, “I don’t have the heart for it.” We don’t say, “I don’t have the brains for it.”

Cultural influence

Many well-meaning people have mistakenly learned, somewhere along the way, that the route to spirituality involves overcoming one’s “feelings” by pretending they don’t exist. First off, very often, these teachings are referring to emotionalism, not sensory perception. Secondly, in order to not be jerked around by one’s feelings, one should not suppress feelings or emotionalism. He must *transmute* feelings (*or* emotionalism stemming from feelings) into the underlying energies from which they are derived. This conversion can be done using either Wisdom or Love. The mere denial of feeling is nothing but a lie to oneself. These lies, also known as suppressed feelings or feelings from which one has dissociated, have a nasty habit of popping up somewhere else, sometimes in a form unrecognizable from the original, and sometimes distorted by the ego into something far uglier than the original feeling.¹

It is true that, in order to ultimately overcome Ego, to become one with God, one must conquer (not be dominated by) the *illusion* of feelings. To do this, one must recognize feelings

have on each other and the role of consciousness in regulating physiology. The western method of discussing one chemical response as if that response can be separated from the cascade of subsequent and integrated responses in a living system is increasingly recognized by advanced biochemical researchers as pure foolishness. However, this is the model that western doctors still cling to.

And what of the placebo effect? Does it affect the brain or the heart? The brain can be trained. While the heart responds to the resonances of every moment, the brain operates, to a large extent, via habit. Remember the example of music, in which a brain linkage to an old song can activate a remembered-feeling response when the song is heard again? That feeling is partly new response, and partly a learned response. In the case of the dopamine-enhancing drugs, a PDer can form positive, movement-allowing mental links with the experience of taking his medication. He will then be able to activate a learned dopamine-for-movement brain response when his brain makes the link between taking a pill and subsequent ease of movement.

When using a placebo, the positive thoughts and expectations generated by this brain link may resonate with the heart and allow the heart to shift its balance toward dopamine for awhile – but only for as long as the positive thoughts or expectations are flowing. It is important to note that those PDers who have *not* already learned, via the drugs, how to step lightly and easily, may *not* have a good positive placebo response. Instead, those PDers who have never learned to move easily (by taking drugs) tend to be more susceptible to the *negative* placebo response, in which they move more poorly when they think that they will move poorly.

The placebo response can only work by activating what a person is capable of imagining. It doesn’t matter if the PDer tells himself superficially positive thoughts: a PDer who cannot imagine what a good response to treatment *feels* like will not be able to respond to a positive placebo with a good response.

¹ I quoted this line earlier, but it’s worth repeating: “Truths suppressed lead disconcertingly to a host of errors.” From Paramahansa Yoganada’s *Autobiography of a Yogi*.

for what they are, not suppress or deny them. What are feelings, truly? They can be described as the sensory events generated by the Divine Intelligence experiencing Itself as creation. When a person looks at a tree, it is the Divine capacity for imagining vision in the person enjoying the experience, that is to say the *feeling*, of looking at Divinity, looking at itself, in the shape of a tree. Feelings are not bad; they are merely an illusion whereby Love can perceive Its own imaginings by pretending that It is an ego (a self-aware individual) experiencing creation.¹

IN SUMMARY

This brief introduction into heart energetics barely scratches the surface of the role – in healthy people – of heart-nerves, the heart’s electromagnetic waves and the phenomenon of

¹ I will share an example of dealing with feelings of foot injury that demonstrates the correct way to deal with physical pain.

Paramahansa Yoganada was working with yoga students on a construction project, when a large cement cylinder was accidentally rolled over his foot. His foot was utterly smashed. The swelling and discoloration were almost instantaneous. The great yogi said, “My foot is injured. Please help me to my room.” Notice what he did: he observed the injury. He did not deny the fact of the injury. He asked for assistance. (This humble request was certainly made for teaching purposes: he was setting an example.) He willingly accepted the help of others, and was helped up the stairs to his room. His students asked him to miraculously heal himself. “Ask God to heal your foot!” they begged him. Yogananda declined, explaining, “My Divine Mother already knows what has happened to my foot.”

The next day, the guru was barely able to hobble about with a cane. The guru’s disciples were deeply concerned because he was scheduled to speak before an audience of several thousand people in just three days. They were afraid that he would not make a good impression if he could barely walk. They had seen the guru work healing miracles on others; they begged him to work a miracle on his foot. Again, he replied that Divine Mother already knew about his foot injury.

The guru made a point of telling his disciples that he was not being negatively affected by pain. He understood the pain signals from his foot were merely communications to his brain that that foot was injured. His equanimity was untouched by fear. Thus, the pain signals merely conveyed information; they triggered no emotionalism. Additionally, using his ability to cognize matter and electricity as forms of light and sound, he did not even need to perceive the electrical signals as pain signals, *per se*.

On the day of the lecture, Yogananda’s foot was still horribly swollen. His foot did not fit into a shoe. The disciples were mortified by the idea that their teacher was planning to shuffle onto the stage of the great hall with one foot in a loose slipper.

They begged again that the guru work a miracle. He declined. Moments before his lecture, he waited in the wings. When he was announced, he kicked off his sandal from one foot and the slipper from the other. He strode barefoot across the stage. His astonished students saw from the wings of the stage that his foot was completely healed.

The most important points in this lesson are that he acknowledged his injury. He allowed those around him to help him. He observed the phenomenon of pain. He intuitively understood the underlying process by which light is transformed into matter, assuming the roles of physical body parts. He could perceive the physical experience of his smashed foot as a convincing transformation of light into broken bones, muscles, nerves and the brain’s response. He was not emotionally influenced by his smashed foot. At no time did he imagine that his injury was out of sync with the Wisdom and Love of the universe. When the time was right for his foot to be healed, it was healed. He may have also contributed to the healing by applying the principle of Mind over matter.

This is the exact opposite of being frightened by an injury and dealing with it by permanently dissociating from it, pretending that it never happened. This latter method, the denial method, is the “technique” used by people with Parkinson’s to prevent themselves from noticing their foot injury – a severe injury that “never hurt much” or that, in some cases, “never happened.”

I do not have a reference for the above foot injury anecdote. I recall coming across Yogananda’s own telling of it in a collection of transcriptions of his lectures. I have also heard the story from one of the monastics who serves at one of the ashrams founded by Yogananda.

heart-brain wave entrainment and the corresponding relationship between adrenaline-based motor function and dopamine-based motor function.

This chapter also introduces a tiny droplet of information about heart from Vedic teachings: the heart is the regulator of how much energy can be allowed into the body for the work of movement, thinking, and feeling, among other actions.¹

In closing, remember: the heart-mind integrating problem does not exist to the same degree in all people with Parkinson's disease. Each person with Parkinson's disease is unique.

As mentioned often, not *all* PDer's are maintaining a severe dissociation response that prevents access to their feelings. *Some* PDer's are only mentally blocked with regard to the existence of the foot injury. When these PDer's attention is gently brought to the foot, the blockage melts away and the foot, and the Parkinson's symptoms heal quickly and smoothly.

But in our experience, *most* of our PD patients have extended foot dissociation to other arenas of the mind – to the point that the mental/emotional blockages need to be consciously fixed.

The dissociation response must be turned off the PDer hopes to re-experience the integration of heart and brain waves and resume healthy levels of release for either type of movement-inducing neurotransmitter, either adrenaline or dopamine.

In order to turn off the dissociation, a person must feel safe. In order to feel safe, a PDer who does not remember what heart feeling is must learn how to experience heart feeling. After he can once again perceive his own heart feelings, he can practice generating the safe feeling.

¹ For those readers familiar with Chinese medical theory, the closed heart, or heart-brain dis-entrainment, is a condition that was referred to as Heart and Kidney (mind) Not Communicating. Over the years, the name Heart and Kidney Not Communicating was assigned to conditions in which various physical body functions in these two organs are not working in sync. However, the original syndrome by this name was a Taoist spiritual condition, not a medical one.



“...So near, and yet so far.”

- Alfred, Lord Tennyson

CHAPTER TWENTY-EIGHT

DISCOVERING PARTIAL RECOVERY

Hurrah! ...sort of

I started the Little Project in 1998. After a year of thrilling changes and even recoveries in many patients with Parkinson's, and by puzzling over possible Qi contortion scenarios, I had been able to put together a particular set of channel confusions that might conceivably develop from an unhealed foot injury at the center of the foot. These particular channel confusions *should* cause a specific collection of physical symptoms. These symptoms matched the symptoms of Parkinson's disease, right down to the dopamine dormancy in the brain.

I wrote about these channel irregularities in chapter five xxx, and supported this idea further in chapter seven xxx, with the map of symptom locations. Once I knew to look for these backwards flow patterns, my colleagues and I could easily, with our hands, detect them in my PD patients. I figured I was onto something.

I now had a satisfactory answer as to *why* some of my PD patients recovered when I treated their foot injuries.

I felt much better after I'd figured this out. I have never liked purely anecdotal medicine. To my mind, there needs to be a reason, a cause and effect relationship going on, to explain an illness and its cure. Otherwise, any so-called cure is a hit or miss event. An anecdotal cure might work some times, it might not work other times. But when I had objective observations of distorted Qi in the feet of PDers, obvious indications of an old unhealed injury, a logical explanation of how PD pathologies could get set in motion from an unhealed injury, *and* the rectification of these PD pathologies when the injury healed, that felt right.

The local neurologists were not impressed

I was certain that the treatment was reversing the course of Parkinson's. Of course, I didn't get any support from local neurologists. Right from the beginning, I ran head-on into the MDs' rule that “anyone who recovers from Parkinson's must have been misdiagnosed.”

Some ex-PDer's were bitter about this. Others assumed that the doctors were correct, and that they had, in fact, been misdiagnosed.

But I suspected that the MDs might be wrong. My strongest proof came from the medicated patients; I had worked on people who'd had PD for years, who, prior to their treatments, had responses to their medications that were typical of PDer's responses to high-ish doses of antiparkinson's medication. When, following treatment, these people's Qi started running correctly and their PD symptoms started to fade, they suddenly started having reactions to their medications that corresponded to what happens when *non*-PDer's take the meds at high-ish levels. These responses were pretty terrible, and led to our no longer working with anyone who has ever used the meds, but the reactions were undeniable, nevertheless. This suggested that a genuine, *chemical* change was occurring in people whose Qi was once again flowing correctly. These people had *not* been misdiagnosed.

When MDs starting writing into the charts of recovered PDers phrases like, “I don’t know why this patient pretended to have Parkinson’s disease for the last six years, [because she now has no symptoms of Parkinson’s]” my hunch that I’d found a cure for Parkinson’s was confirmed. I was almost ready to notify the press and call for a celebration.

Then came the unexpected changes in some of the PDers who’d been recovering. The happy ending of my Little Project was put on hold.

More than just a foot problem - sometimes

When this project first got going, some people recovered completely in response to the techniques we used to heal their foot injuries. As expected, some people recovered more quickly than others.

I also saw that some people, after recovering from their adrenaline-producing foot injuries, began exhibiting new, extreme levels of anxiety or depression. Sometimes, the new levels of anxiety were overwhelming. Other times, the anxiety showed up once in a while, in a format resembling a panic attack. These attacks were accompanied, in some cases, with a temporary return to Parkinson’s-like movement problems. However, the movement problems were usually far, far worse than they had ever been in the past.

Local interest in the Little Project began to stir, and a few other local acupuncturists had started treating PDers. I was glad of this, especially when some PDers starting having unexpected outbursts of anxiety or panic. My colleagues were able to confirm from their own experience some of the unexpected outcomes I was starting to see.

PARTIAL RECOVERY

We had treated more than fifty people at this point, and six of them had fallen into some sort of weird condition in which structural symptoms appeared to be improving, but anxiety, movement initiation, or tremor had become worse. In some of these cases, the worsening symptoms were intermittent and clearly related to spates of anxiety. In other cases, they came on hard and fast and accelerated beyond anything associated with the expected pace of steadily worsening Parkinson’s disease.

Because they had all shown significant, lasting physical improvement before their anxiety or severe slowness set in, we referred to the condition of these six people as “partially recovered.” None of these people had ever used medication. We set out to figure out what was going on with this group.¹

¹ Since then, after working with hundreds of PDers, we know that the percentage of people that collapse into a condition of “partial recovery” is much higher than our original six out of fifty. At that time, we were still treating mostly medicated PDers. The medicated PDers were more likely to go the other way during recovery: euphoria, rapid addiction to the medication and weekly, sometimes daily, increases in dose. Then, at the new high levels of the drugs, horrible problems set in: hideous, excruciating dyskinesia, insanity and in a few cases, death. However, as long as they were high on their drugs, they weren’t susceptible to anxiety and motor inhibition. Therefore, we didn’t notice partial recovery symptoms in medicated patients.

After we stopped working with medicated PDers, we could generate more accurate numbers. We then realized that a *majority* of our unmedicated PDers developed symptoms of partial recovery. Then again, the people who came to me for help with their “movement problems,” who had clearly had symptoms of Parkinson’s, but who had *not* received an official diagnosis of Parkinson’s, *all* recovered easily; none of the people who lacked an “official diagnosis” became stuck in partial recovery.

I even recall one of these patients laughing merrily while discussing the curious fact that I mostly worked with people with Parkinson’s. She chortled, “Wouldn’t it be funny if it turned out that that’s what *I* have?” She

Although each of the partially recovered had his own set of mental rules for events that should, to his mind, cause the temporary return of Parkinson's disease, a commonality ran through these cases. Most of their structural symptoms were gone: some had return of facial expression, return of arm swing, return of the atrophied muscle between the thumb and index finger, return of handwriting ability, and/or return to good posture: improvement all along the Stomach and Large Intestine channels. However, they were increasingly the victims of lack of interest in life, or anxiety, or even panic attacks. And when they were the most blasé or anxious, they had severe movement initiation problems and/or tremor. If they were anxious almost all the time, they might only move well during the brief periods when they were accustomed to be anxiety-free: during safe activities such as walking in the woods, doing the laundry, or sewing.

These easy-to-move moments did *not* necessarily occur when doing tasks that are generally easy for people with Parkinson's. For example, sewing, with its small motor requirement, is usually not possible for people with advancing Parkinson's. But in one partially recovered PDer who enjoyed sewing, she might be tremor-free while sitting at the sewing machine. On the other hand, this same PDer (who could once again ride her bicycle and go up and down stairs easily and turn doorknobs and smile radiantly) would tremor violently if she did anything having to do with alphabetizing or adding up numbers. (It seemed significant to me that these two latter skills were probably learned when she was around age seven, which corresponded to her age at the time of foot injury.)

Another PDer was convinced that she was getting worse. Even though her handwriting was becoming large and easy, and her arms and legs were relaxed (no longer stiff), and her facial expression had returned, she was increasingly subject to self-pity. When I asked her if she thought she was recovering, she told me that she didn't deserve to recover.

She got so that, by the end of the day, she was exhausted from dragging her limp (no longer rigid) body around. When it came time to get into bed, she was simply too tired to even figure out how to climb into bed. In order to get into bed, she had to pretend that she didn't have Parkinson's. Then, by pretending, she could easily get into bed, adjust her pillows and get the blankets just how she liked. Then she could go back to suffering from the heavy weight of her weak and limp body: suffering from her Parkinson's disease. When I asked why she didn't pretend to not have Parkinson's all day long, she looked at me with surprise. "That wouldn't be honest," she replied.¹

actually had a somewhat advanced case, had almost no use of one arm, had no facial expression, a moderately bad tremor and was using a walker to assist with her tiny, shuffling steps. I assured her she did not have Parkinson's disease. She recovered very quickly and easily. I asked her to see a neurologist after she recovered, and to tell the neurologist all her symptoms. I was curious as to what the neurologist might say. The neurologist listened to her list of symptoms and told her, "If you actually had the symptoms you've described, and your acupuncturist got rid of them, then you were neurotic." This same neurologist told another PDer who was recovering that "There is no way a foot injury can affect your brain. I'm going to see about having your acupuncturist arrested for practicing medicine without a license."

¹ This same person decided to take my advice and pretend that she didn't have Parkinson's. After several days, she was feeling so good that she went to play tennis with a friend. She hadn't played tennis in years. She told me in an email that she loved the feeling of once again whacking the tennis ball with all her might and that she really enjoyed playing. However, while using some fancy footwork to return a tricky shot she tripped over her own foot and fell down. She stopped playing right then. Her email to me concluded by saying that falling down was proof that it wasn't safe to pretend that she didn't have Parkinson's. She needed to have Parkinson's in order to be safe.

In the past, these people had at least tried to use their formidable wills to conquer any anxious thoughts. But now some of them were plunged into powerful anxieties or depression, sometimes around the clock. Of course, increasing anxiety can be a symptom of Parkinson's disease. But some of these people, despite obvious indications of physical improvement, were flinging themselves headfirst into the anxiety pit and they just *knew* they could never climb out. What had happened to these previously confident people, with their early, mild cases of Parkinson's disease? When a few of these people tried taking low doses of Parkinson's medication to ease their difficulties, the medications produced dyskinesias within a matter of a few weeks (an adverse effect of the drugs that should only start after several *years*). Evidently, these people could no longer tolerate dopamine-enhancing drugs. They didn't actually have Parkinson's any more. What did they have?

Had we created a monster?

And more

Then, as time passed and our caseload grew, we noticed that a few more people were starting to behave as if they too were heading in the direction of partial recovery. Many cases were only mild: a person would recover from half a dozen symptoms of PD and then announce that he knew he couldn't actually get better, and from then on, he would be stagnant, getting neither better nor worse. What was going on?

Here is a typical email from a partially recovered PDer. The email arrived after we started telling our patients that we suspected a negative attitude component in cases of partial recovery:

"My foot injury is long since gone and I am nearly back to health. I am doing Tai Chi exercises every day. However, lately I am in an up-and-down state, not sure how else to describe it. Often it seems I am losing ground, motion-wise, but then I do something in less time than usual, like a sink full of dishes or preparing dinner.

"A few days ago I couldn't get my feet to cooperate driving the car (a standard shift), then yesterday, I thought, "I can do it!" and drove to get groceries with little trouble. Sometimes if I've been sitting awhile I get caught on the couch, but the next minute I'm fine or am walking about on the spongy cushions, closing the blind with flawless balance. I seem to be able to do what I decide I can do (I hear you chuckling), but the deciding has become the tricky part!

"I've been watching my thought patterns and was surprised, well, not that surprised [after talking with you], to note how often the thoughts spun off into disaster scenarios.

"Another thing I might add regarding wariness; one of my nicknames around here is "The Disaster Forecaster" which ought to be self-explanatory."

This patient was extremely intelligent. She was also a therapist. However, she could not see her own illogical and self-serving behaviors in the same clear light with which she viewed the quirks of others.

The main reason she felt that she deserved to have Parkinson's was that, when she was first diagnosed, she had immediately thought to herself, "Good. Now everyone else will have to do all the work for a change." She had immediately been consumed with unforgivable guilt over having such a thought. After many months in our program, as she began to experience symptoms of recovery, she felt guilty about it and realized that she "deserved" to have Parkinson's for life. This and other types of negative thinking are normal when a person is locked into sympathetic mode. However, at this time, we did not yet understand the mental component underlying Parkinson's; we could not understand how a person might be once again capable of movement but not able to emotionally avail himself of it.

Not yet understanding that the underlying problem was one of dissociation from the heart, we assumed that these people merely had ordinary anxiety-type problems. We hoped that maybe, if we helped them overcome their anxiety, their situational or intermittent problems would go away. After all, we could help them deal with their anxieties using standard anti-anxiety treatments based on Asian medicine. If that didn't work, there are dozens of techniques available these days for helping people overcome their buried, subconscious anxiety triggers. We would find something. No problem!

Our research continued but the solution evaded us.

We knew we needed to do more than figure out the problem; we needed to find a solution. Only by reversing the *all* the symptoms of Parkinson's, curing the whole problem in all or in nearly all PDers, could we convincingly state that we had found a consistently effective cure for Parkinson's disease. Some of our patients were distrustful of our hesitancy to make press announcements and "go public" with our findings. But we needed to figure out the hang-up in partial recovery before we announced a cure for Parkinson's.¹

¹ The editor of the *American Journal of Acupuncture*, before publishing my first article, told me that I needed to go slowly and not blazon our discoveries too early. I paraphrase her words, "You only get one chance. Your hypotheses will step on a lot of toes. If you go out with a theory that is too specific or too definite, and a person is able to find one small error in your wording or your phrasing, the whole body of your work will be dismissed and even ridiculed on the basis of that small misstep. You will never again be taken seriously. So go slowly."

She had been publishing the journal for twenty-six years. As I spoke with her on the phone prior to sending her my first article, she said that her heart was telling her this: the reason that she had founded the highly respected journal twenty-six years was to someday publish my research. She said that my findings had implications that went far beyond Parkinson's disease. Her kind words gave me hope for the Little Project and added to my sense that this project was a part of something more important than my curiosity. Her words also gave me a clear picture of just how careful I needed to be to not go off half-cocked.

When, four years into the project, we realized that medicated patients might be far worse off by recovering than they would be if they just stayed their course with their medications, I repeatedly said prayers of thanks to that editor's word of advice and caution. By going slowly, by not prematurely creating publicity for our ideas, we had saved countless medicated patients from potential harm.

Later, when we fully understood the significance of the mental/emotional component and the unique problems of people stuck in partial recovery (they usually cannot tolerate antiparkinson's medications if they are partially recovered, and so are left without treatment options if they fail to recover completely) we were able to better screen patients in terms of their interest in and potential ability regarding overhauling their personalities. Attempting recovery from a mental/emotional disorder is not for everyone: it can't be done merely because a spouse desires it or because a person doesn't want anyone to know that he is fallible: many people have wanted us to "cure" them extra quickly and even secretly so that no one would ever know that they had a "problem." These frightened people are not good candidates for recovery.

When we understood the significance of the dissociation response, we were again grateful that we had held off on prematurely making a big public splash about our ideas.

I would like to take this moment to thank that brilliant editor, B.G. Grace. In its day, the *American Journal of Acupuncture* was the most highly regarded of all the English language peer-reviewed acupuncture research journals. In the early 2000s, When the National Institute of Health first decided to include research on alternative and complementary medicine in its web-based open-to-the-public search engine, the *American Journal of Acupuncture* was the only acupuncture journal with academic standards rigorous enough to be included in their database.

B.G. Grace was thorough. I was amazed when, just before publication, she called me to question the page number that I had used in a footnote reference; she had found a copy of the edition I quoted and my page number was incorrect. She had personally verified my every footnote, right down to the page numbers in the specific edition! She had also, she confessed, spoken to a physics professor to have my statements about parallel electrical circuits confirmed.

Coming up next

The next chapters track the developments of our struggles with partially recovered PDers from the beginning of the Little Project up until early 2008. We collected thousands of bits of data that confirm our hypotheses, but as I fly through the following chapters I will touch down at only a few crucial points, giving specific examples from only a few case studies.

I have chosen a chronological format for the next few chapters, and not just to keep the reader hanging in suspense. The chronological format helps me keep everything straight and, hopefully, a time line format will emphasize the ongoing nature of our work: this project is very young and is very much still a work in progress. Also, following a time line allows me to answer a few frequently asked questions about the formation of the Parkinson's Recovery Project and the Parkinson's Treatment Team.

Finally, my husband, who teaches writing at the local University and who has helped the project enormously by proof-reading my writing and by teaching me better writing skills, likes the time-line format. He says it adds a touch of "you are there."



She correctly anticipated the fuss that would ensue even from my small article in her journal. She told me that she would not run the article until I got an unlisted phone number and a post office box, thus creating a barrier between my personal life and my research. (These measures turned out to be inadequate, but were still helpful in keeping all but the most aggressive at bay.) She was very concerned about what flack might start to fly if and when my research ever hit the big news media. Antiparkinson's drugs are a multi-billion dollar industry. Parkinson's research on the already disproved dopamine-cell death model brings in megabucks in research grants to researchers. She wondered how the drug companies and western researchers might respond to my hypotheses.

She said to me, maybe jokingly, "They're going to have to kill you."

“Through Thy grace the sudden shafts of wisdom will dispel error accumulations of countless centuries.”

- Paramahansa Yogananda's Whispers From Eternity

CHAPTER TWENTY-NINE

PURSUING ANSWERS: 1998 TO 2003 – THE YEARS WITH MEDICATED PATIENTS

1996-1997

After treating the old foot injuries of three people who happened to have Parkinson's-like symptoms, the PD symptoms went away. None of the three had ever taken antiparkinson's medications.

1998

As noted in chapter one, I did a pilot study, published an article, and started a website.

The website query asked if any one else could feel the same reversed Qi flow patterns that we'd felt in the Stomach channels of PDers. It also asked if anyone else had noticed a Qi problem at ST-42 that might suggest an unhealed injury at that point. The 40-page query was, at that time, addressed to acupuncturists. I was hoping for some sudden shafts of wisdom to show up in my email inbox.

Although FSR treatment can be performed by anyone and the treatment program does *not* usually require the use of acupuncture, acupuncturists are more experienced than most at feeling energy currents, and they understand the channel jargon I was using. So my queries were addressed using lingo of traditional Asian medicine. Almost immediately, responses started coming in, along with questions. The questions, their answers, and results from continuing research were added to the webpage.¹

As early as 1998, a few more PDers had recovered. My excitement over their recovery was tempered by my frustrations that these PDers were then told by their doctors that they must have been misdiagnosed.

1999

We noticed a mental/emotional shift that followed the foot healing.

Some PDers who recovered from Parkinson's disease experienced an unexpected flood of emotion and a different type of *mental* competency. Some noted changes in sensory perception.

¹ I was hesitant, at first to mention one of the strangest findings: many PDers had been cancer survivors, and that their cancer removal scars were on the Stomach or Large Intestine channels on the side of the body that had first showed symptoms of Parkinson's.

I shared this strangeness with my advisor, Dr. Fred Jones. I told him that it seemed very possible that backwards flowing or otherwise aberrant electrical currents might someday prove to be responsible for many types of cancers. He agreed that this might be a logical conclusion of our findings, and wouldn't that throw a wrench into the current paradigm for treating cancers of "no known origin." Then he said, laughing heartily, "They're going to have to kill you."

In some cases of sensory perception shift, colors were suddenly brighter, sounds were more pleasant.

In terms of mental competency, some found themselves, without even trying, imagining faces and figures in passing clouds or in the leaves of trees. Some found that, after nearly a lifetime of not being able to imagine or visualize positive or playful images, they could now create these mental images effortlessly.

As for emotions, many experienced “losing control:” dissolving into tears when confronted with situations that had previously been perceived as banal, such as the sight of a young child walking a small dog, or while watching the evening news’ “human interest story.” The strange thing was, they didn’t mind losing control even though they had been, in the past, somewhat reluctant to give free rein to their feelings.

In many cases, these emotions and abilities to visualize had not merely been missing during the few years since having been diagnosed with Parkinson’s disease; very often, the emotions and imaginings felt utterly new, as if they had not ever been felt during the PDer’s adult life. It was not uncommon for a recovering PDer, smiling through his sobs, to confess that he had never before been able to cry.

On the other hand, some PDers whose feet were restored to normal function and whose physiology showed reversal of Parkinson’s symptoms (return of smile and other facial muscles, correction of posture, return of blood flow and warmth to previously cold and inflexible feet, ability to sleep deeply, return of senses of taste, smell, etc.) did *not* experience any change in their perceptions or mental imagining abilities. Some of these partially recovered people became drastically *more* anxious or emotionally wary. And despite their physiological improvements, they sometimes had rapid worsening of movement inhibition and tremor.

As in the case of the PDer with the dentist phobia that I described in chapter three, I saw that *some* of the people in this latter group had no Parkinson’s symptoms – as long as no emotional issues loomed on the horizon. However, after experiencing intermittent periods of days or even weeks with no PD symptoms, these people could suddenly collapse into complete paralysis or teeth rattling, body-wide tremor if an unexpected call was made on their fears and/or emotions. Others dove into a complete free-fall of anxiety, panic, and immobility with no periods of relief.

The people who could move normally when they were in a good mood clearly did *not* have a neurotransmitter deficiency. But their ability to initiate the types of mental processes, the types of thoughts, which *release* neurotransmitters for correct motor function had become extraordinarily mood dependent.

We had no idea what was at the root of this weird behavior, but there were distinct differences between those who recovered easily and those who slipped into partial recovery. Those who recovered easily experienced a major shift towards positive imagining and capacity for feeling. Those who became stuck in partial recovery became suddenly *much less* capable of positive imagining and feeling.

So a mental/emotional component was hypothesized and added to the foot injury/electrical disarray hypothesis. The emotional component was evidently not present to the

same degree *or direction* (inclined towards getting better or inclined towards getting worse) in all PDers.

We spent at least an hour a week talking with each PDer in our program. We probed their personalities and life histories and, in many cases, interviewed their spouses. As PDers recovered or got stuck in partial recovery, we looked for correlations between their emotional posture and their rate of recovery.

In addition to the already mentioned differences between people who recovered easily and those that got stuck in partial recovery, we noticed something else: the professional musicians recovered very easily and quickly. We had five professional musicians with Parkinson's, including a composer and a conductor. They all recovered very quickly, with no emotional glitches.

Stymied

The people who were partially recovered were frustrated, and so were we. It was clear that the partially recovered PDers had some emotional rigidity, but we couldn't be sure how emotion could contribute to Parkinson's. Also, if emotions did contribute to Parkinson's, did that mean that the foot theory was incorrect? (At this point, the term "psychogenic parkinsonism" had not yet been coined.) Did this mean that the reversed Qi problem that we'd seen in all the PDers in our program wasn't actually the cause of Parkinson's disease?

True, at this point in the program, we had seen *some* people recover from Parkinson's disease simply by having the foot injury addressed. But we clearly had not yet figured out the way to help people whose physical symptoms were melting away in response to foot-injury therapy but whose minds were now and then being pulled, as if by an intermittent or constant tractor beam, into paralyzing negative emotions. Since we did not have a way to reverse this mental condition, we could not *prove* anything about its relationship to the accompanying movement initiation problems – problems that looked, in some cases, like *accelerated* symptoms of Parkinson's disease. Even though many of these partially recovered PDers had many improvements in the physical condition of their bodies, that didn't count for much if they couldn't consistently move, or if their tremor became an intermittent monster.

We certainly couldn't say that we'd found a one-size-fits-all treatment for Parkinson's.

GROWTH OF THE INQUIRY

In one year, the little Internet inquiry grew from forty pages to nearly ninety. I kept adding pages of writing, describing the treatment therapy that I was using and noting recovery symptoms that seemed to support the original hypothesis. The number of emails coming my way from PDers and acupuncturists continued to grow.

A majority of the emails from PDers were about problems with their medications. At that point in time, a local neurologist had laughingly assured my patients that, if they ever recovered, they could just stop taking the medications at that time. No problem.

I naively assumed that the doc was correct, and was still working with medicated PDers.

Creation of a non-profit organization

I was still scratching my head over the channel theory portion of the problem when some forward-looking friends got after me to form a non-profit organization to pay for the website. A PDer from San Francisco whom I'd met only once spearheaded the formation of the non-profit.

He moved back to Chicago and I lost touch, so I can't give him due credit by name. I'm sorry. He was a real live wire. He came up with the name "Parkinson's Recovery Project." He arranged for his lawyer son-in-law to do the incorporation paperwork for free.

By the end of 1999, the Parkinson's Recovery Project had, and still has, tax-exempt status with the United States IRS and can accept tax-deductible donations.

The Parkinson's Recovery Project's mission is educational and charitable. The organization provides the vehicle by which we can keep our publications and updates available, for free download, on the Internet.¹

2000

The "Other" side, the injured side of the body

Numbness and poor proprioception

Many PDer's had poor proprioceptive awareness of the injured foot. (Proprioception is the ability to know where a body part is even if the eyes are closed.) Many a PDer, when sliding a foot into his trousers, can't really be sure where his foot is from the time the foot is seen entering the cloth until the foot is seen coming out the bottom of the pant leg. Sometimes, this decrease in proprioception extends to the hand and arm on the injured side of the body. As one PDer explained it, "I don't have eyes on my hands anymore; I can't see where they are unless I'm looking at them." In fact, it was only his *right* hand that couldn't "see" when he was trying to get his arm through his sleeve. He was always able to know where his left hand was. His PD had first shown up on the right side. (Eventually, as the Parkinson's becomes bilateral, the loss of proprioception extends, somewhat, to the healthier side.)

Also, even though many PDer's had a high level of numbness in certain areas on the injured foot, it took us several years to realize that PDer's rarely knew that they were numb. I first discovered the alarming level of foot numbness when I tried to get some Qi moving through the foot by needling the famously sensitive SP-3, near the ball of the foot. After noticing that all the

¹ Unsolicited donations help to cover the expenses of keeping the website running, hiring a web professional once in a while to make the website updates, and paying for the mailbox fees, the odds-and-ends office supplies, a small computer and printer and a desktop photocopier. It's a shoestring operation, but it's a beautiful, optimistic shoestring: it does the job. Hopefully, someday when funds allow, we can hire someone to do videotaping of our patients and our treatment techniques. These videos could be made into an instructional CD that could accompany future editions of this book.

Our greatest dream is to get adequate funding to pay for before-and-after PET scan analysis of at least twenty of our PD patients. These scans may provide a hint of objective confirmation of what appears to be a permanent reversal of all Parkinson's symptoms in our fully recovered patients. Such a project would require hundreds of thousands of dollars.

At present (2006), our only funding is unsolicited donations from individuals and the donations of the PD Team, which gives a percentage of its fees to the non-profit. We usually rack up a few thousand dollars a year: just enough to cover costs. A few unpaid volunteer acupuncturists, including myself, do all of the office, correspondence, and collaborative research work. I write the books in my "spare time" and publish them for free download on the Internet.

All of us involved in the Project feel honored to be doing this work. It sounds corny, but the Little Project is a labor of love.

PDers who still had foot injuries were nearly numb at this point, I tested many PDers for numbness.¹

The numbness was nearly always worse on the side that first showed symptoms of Parkinson's. Needling the injured foot usually got a poor response, but needling the healthy foot at this point very often got a lively response. By this means, we were able to determine that most PDers have some degree of numbness on the injured-foot side, even though they almost always insist, at first, that their feet have perfectly normal sensitivity.

The numbness and the poor proprioception on the injured foot side were not *too* surprising, given the foot injury and the blocked flow of Qi. What was surprising was what we discovered next: the mental blocking of the *awareness* of the *existence* of whichever side of the body first showed symptoms of Parkinson's.

Diminished mental awareness of the existence of the injured side, the “other” side

Most of our PD patients seemed unable to fully cognize the *existence* of whichever side of their body had first manifested symptoms of Parkinson's. They had less mental awareness of the existence of that side of the body.

For example, if a PDer was asked to imagine light in both sides of the body, the side that had first manifested PD symptoms was considerably dimmer – if there was any light at all. The injured foot, often, was mentally inaccessible and/or could not light up at all. More strangely, if a PDer had injured himself on some other part of his body, long ago, on the same side of the body that had the injured foot, he could not recall which side of the body had been injured, even if the scar made the location obvious. However, he might have normal recall of the locations of any injuries that had occurred on the healthy (non foot-injury side) of his body.

For that matter, many PDers had trouble relating the word “left” or “right” to the injured left or right side of the body. Their healthy side of the body could be identified as being the left side or the right side. The injured side was usually thought of as “the other side.” This is a bit tricky to explain. I will give an example.

A Pledge of Allegiance example

One PDer whose PD started on the right side of her body when she was in her late forties told us that, since she was six years old and had to say the classroom's daily Pledge of Allegiance to the flag while holding her right hand over her heart, she always had to perform the

¹ The following example may further illustrate the PDer's inability to feel parts of his own body. I had one PDer with a grossly displaced set of ankle bones on her right foot. She had never noticed any pain in the ankle. In fact, she had been skiing on this impossible ankle for years.

Week after week, I held her ankle. After each FSR session on her ankle, I would ask her, “How does your ankle feel now?”

Her reply was always the same: “How should I know?”

I kept trying to explain to her that she, and no one else, should and could know how her own ankle felt, but she never understood what I was talking about. And then, after more than a year of once-a-week sessions, her ankle relaxed deeply and the bones slid back into place. The next morning, this PDer called me at five a.m. on my home phone.

“I knew you'd want me to call any hour of the day or night with such big news,” she said, inaccurately. “I woke up this morning and my ankle is swollen to three times its normal size. It's black and blue; it looks just horrible. I can't even walk on it. But the amazing thing is I can really feel it; it hurts like hell. I knew you'd want to know, I just knew you'd be thrilled. I finally understand what you've been asking about when you ask me how my ankle feels. I'm feeling my ankle!” Even after the ankle healed, she continued to be able to feel the existence of her ankle.

following mental steps: She would look at or mentally acknowledge her left hand. She was certain that her left hand was not the hand that should be used for the Pledge, so she would place her “other hand” over her heart. She was not stupid. She was an honor student throughout school, and she was right-handed. She had excellent motor skills and had enjoyed sports. However, she realized during our work together that she was always momentarily uncertain about which hand to use if someone asked her specifically to use her right hand, or which way to turn if she was told to “turn right.”

She also told me that, despite her experiences with driving in both the US and in England, she had never been able to understand what was meant by the phrase, “In the US, they drive on the right side of the road.” “After all,” she explained to me, “a road is a road. A road doesn’t have a right side, does it? Right relative to what?”

It wasn’t until she recovered from Parkinson’s that she understood what was meant by the “right side of the road.” After recovering, she also realized that she had never actually acknowledged the right side of her face when she looked in a mirror.

“Which side did the Parkinson’s symptoms show up on?”

Surprisingly, many people whose Parkinson’s has slowly, after a few years, become somewhat bilateral cannot even state with certainty which side of their body first manifested symptoms. They may know for certain that their PD symptoms did *not* appear first on the (relatively) healthier side, but they can’t be sure if the symptoms *did* appear first on the “other” side. These people are not stupid. They are extremely aware of their physical problems. They just can’t say on which side of the body the symptoms started.

“Which ankle?”

For example, if a PDer said that, in the past, he frequently twisted his ankles, I asked him, “Which ankle, the left one or the right one, did you usually twist?” It was not uncommon for the person to answer: “I don’t know. Maybe both. Probably both.”

If I have already observed that his PD symptoms are primarily on, say, the *right* side of his body, I will follow his “I don’t know” with a pointed question: “Do you ever sprain your *left* ankle?” Very often, this specific question will make the person pause a moment, stare at the healthy ankle, and then reply, “No.” In fact, he may continue, he’s pretty sure he’s *never* sprained that left ankle. He may even state, “No, that’s my good ankle, I don’t sprain that one.” From there, I can ask him if he usually sprains his right ankle. In response to this side-specific question, he will usually answer yet again that *he doesn’t know* which side it is that gets sprained, but he does know that he sprains his ankles a lot. And it’s not the left ankle; possibly, he might conclude, it’s the “other” one.

This non-awareness happens even with memorable, terrible injuries or broken bones. Many times a PDer has told me he recalls a *severe* ankle injury, one that kept him on crutches for weeks. If I ask, “Was it on the (name of whichever side the PD first manifested) ankle?” He may answer that he just can’t remember which ankle it was. Then I change my question and ask if it was on his (name of *uninjured* side) ankle. “Oh, no, my (name of *uninjured* side) ankle has always been fine, that’s the strong one.” He may have a *strong* memory of a severe injury, but he may be vague as to location; it’s as if the location of the injury was in a place that, possibly, because of diminished awareness of that side of his body, does not fully exist.

This very common and powerful demonstration of mental disassociation from the injured side of the body was often repeated in further inquiries with the same patient. After enough questioning, the PDer may start to understand that he has two kinds of awarenesses for the two sides of his body: he has the healthy side, which he can identify with the word “left” or “right,” and he has the side on which PD symptoms first appeared, a side that he might call the “other” side. The PDer doesn’t know much about the “other” side; when the “other” side gets injured, he might only know that he’s been injured – but as soon as the overt symptoms of the injury clear up and the visual or pain cues are gone, he may not be able to conjure up a clear memory of the location, especially with regard to *which side* of the body had the problem.

Yet another example of the “other” side situation

When working with a new PD patient, I need to determine for certain whether or not the person was correctly diagnosed, whether or not he actually has Parkinson’s disease. I like to ascertain on which side of his body the PD symptoms first appeared so that I know where to start feeling for the Qi disorder and where to start with the Tui Na therapy. I often employ a simple test.

I start this test by asking the PDer a few distracting questions about whether or not he has asthma or digestive problems, and then, if I suspect the Parkinson’s disease started, for purpose of example, on his *right* side, I will, without warning, calmly ask the patient to raise his *right* arm. If his Parkinson’s did start on the right, he will usually perform the following response: he will pause, then he will look at his *left* arm or move his head slightly to the *left*, as if he is thinking about his left arm. He will mentally acknowledge that the left arm is the left arm, and therefore not the right-side arm, and then, by process of elimination, conclude that the arm on the other side of the body must be the right arm. After a telling head turn (to the left) and a pause, he will then raise the arm that is *not on the left* side of his body.

The PDer’s mental process of first ascertaining that the left arm is an arm, but somehow not the arm I am asking about, was often quite visible via his body language.

Every once in a great while, the patient would even *raise* the left arm in immediate response to the instruction: “Please raise your right arm.” He might then quickly add something such as, “Oops, you said ‘right, didn’t you,” after which, he would lower his incorrect arm and put the correct arm in the upright position.

After this, even if the PDer doesn’t know which side his symptoms started on, at least *I* have a pretty good idea.

By the way, to check the validity of this testing method, I sometimes asked a PDer to raise the arm on his healthy side, instead of the PD side. When I asked about the healthy side, PDers didn’t pause, didn’t look at the opposite side. They simply raised the correct arm. Of course, if I then wanted to also check his PD-side response to this test, I had wait at least ten minutes to catch him unawares.

Clumsiness of the “other” side

PDers have often injured themselves repeatedly on the side of the body where symptoms first appeared, on their “other” side. Because they do not have good body awareness and self-perception on that other side of the body, that other side might often hit the doorframe when going through a door. That other side might have the elbow that always bangs into the edge of the table. That other side may have the hip that bumps the furniture while crossing the room.

Because there is limited mental awareness of the existence, let alone the size and location, of that other side, that other side of the body is often the “clumsy” side. However, if you ask the PDer which side of his body is the clumsy side, he will say that he doesn’t know, or that he doesn’t think that one side is clumsier than the other. Only when you point out that his assortment of scars and stitches and the various injuries that he *does* know about are certainly not on his “good” side might he realize that he doesn’t have whole-body clumsiness, but has, in fact, one-sided clumsiness.

We noticed that people who recovered easily also regained “other side” awareness easily. The ones who were getting stuck in partial recovery usually had “other sides” that remained elusive, even non-existent to their closed eyes, despite healing of the foot injury and the resumption of correct Qi flow!

When we first discovered the “other” side unawareness in PDers, it was all very interesting, if not downright bizarre. In one person, we might have dismissed it as a personality quirk. When nearly all PDers manifested some level of unawareness of the “other” side, the PD side, it seemed significant. Of course, we were completely clueless about the best way to approach it. We tried dozens of techniques and exercises that we hoped might restore awareness to the “other” side.

None of them worked.

The expanding website

The number of pages of information on the website continued to grow. However, it was mostly a hodgepodge of short case studies and ideas, posted in four major spurts of additions and revisions. Each flurry of writings incorporated the previous bits but added new material, mostly in the form of Frequently Asked Questions and Answers. I referred to these updates as “editions.” In late 2000, I assembled the material together into more of a book format. This 350-page version of the growing body of material was titled *Recovery From Parkinson’s: A Practitioner’s Handbook*. This book was posted on our website and was available for free downloading.

This 5th edition of our research piled together all of our writings on the subject up to 2000. This book had a chapter format and some donated, professional artwork. It had no index and we still had many unanswered questions. This edition was the first one to be addressed to the layman as a How-To-Treat handbook rather than as a query to professional health practitioners.

2001

Inability to visualize self or scenarios with positive outcomes

We were beginning to realize that partially recovered PDers had more than just anxiety problems and an inability to acknowledge the injured side of their bodies. Their capacities for imagination seemed to be stuck in “Negativity or Nothing” mode. We discovered this when we tried to get partially recovered PDers to help their own healing process via visualization.

In part because we realized that these people often couldn’t cognize the injured side of the body, and partly because visualization is supposed to be a good healing tool, we had started asking PDers to visualize light in their injured feet. As we kept plugging away at this task, we came upon another PDer commonality: an aversion to and/or difficulty with visualization of light in the injured area.

Darkness inside

In the early days of our research, we did not ask patients to be actively involved in the treatments that we were doing on their foot injuries. This was because, as researchers, we wanted to know the extent to which the treatment could be effective whether or not the patient's consciousness was involved.

In the earliest stages of our project several patients had recovered – so far as we knew – from *passive* acceptance of Tui Na therapy. These earliest recoverers had not been actively involved in any sort of mentally therapeutic processes during the treatments– at least none that we knew of. When we started trying to establish the simplest common denominator for effective treatment, we did not want to introduce the variable of patient involvement.

However, after we realized with a certainty that most patients' foot injury could eventually respond to Yin Tui Na whether they were awake or sleeping during the treatment session, we started experimenting with having patients becoming involved in the process. We hoped that patient involvement might accelerate the foot-healing process.

It is not uncommon in physical therapy work for a therapist to ask the patient to mentally focus on the area being treated. This mental focus can be provided by many means, including imagining the breath flowing into the area, imagining the area being filled with light, or even mentally talking to the area with reassuring tones.

After a few years of working with PDers, during which *some* PDers had completely recovered with no specific, pointed mental involvement, but others were stuck in partial recovery, we decided to add a new component: asking PDers to participate in their own recovery process. While performing FSR therapy on our PD patients, we asked them to try to visualize light, energy, breath, or some form of vitality in the areas that we were working on.

The responses that we got from most of the PDers – people of strong will power and determination – were decidedly unexpected. In a majority of cases, we were told things like “I've never been able to do visualizations and I don't want to,” “I can't make light go into my foot and that's that,” “The foot area is murkier than the rest of the leg and I can't change it,” or “Visualization is not a part of my personality and I'm not interested in changing,” and even “Visualization is a sin.”

Despite verbal encouragement and repeated attempts at lighting up the foot or body part in question, most PDers who were willing to at least give it a try usually found that they were simply unable to imagine light in an injured area. Benton's case study makes a good example of this.

Benton

I was holding the ankle of Benton, a PDer. I had started by holding his leg at the knee and had slowly worked my way down to the ankle. I stopped at the ankle. Not only was there no Qi flowing in the ankle, it felt like the ankle of a corpse. That ankle was stony, cold, and absolutely unresponsive. It felt to my hands as if there was a big, black hole in the middle of his ankle. The hole was about two inches high and an inch and a half across.

I asked Benton to mentally picture some light in his left ankle. He tried for about fifteen seconds and then said he couldn't picture his left ankle. He couldn't even picture his own left leg.

Benton was a professor of anatomy. He had written a book on anatomy. The book was full of pictures. I asked him to mentally imagine a picture of a left ankle from his anatomy book. He tried, but he still couldn't even picture a left leg, let alone an ankle.

I asked him to just imagine a left lower leg and ankle floating in space, not related to him in any way. He couldn't do it.

Meanwhile, I was still holding his ankle. It felt like there was a big black hole in the middle of his ankle.

Benton's wife was sitting on my office couch, watching us. I asked Benton to mentally picture his wife's left leg. He couldn't do it. He could imagine her from the face down to the knees, but his mental picture of her stopped at the knees.

I asked him to try to think about his daughter. Could he picture her playing, or running, anything that had legs involved? He tried, but his mental images of her all ended at her knee. Meanwhile, I was marveling at what felt like a big, black hole in the middle of his ankle.

I asked him if there was anyone in the world whom he might be able to think of as having a leg and an ankle.

Benton was getting a frustrated. He was an anatomy professor, for goodness' sake! He didn't want to talk about it any more. I stopped talking to him and silently continued holding his ankle. It still felt as if there was a big, black hole in the middle of his ankle, but I hadn't mentioned that to Benton. I didn't want him to know about the big black hole, but I did hope that he could figure out how to see it in his mind's eye so that he could start healing it: **one can't heal what one doesn't know exists.**

Finally, after about five minutes, he opened his eyes and said, "I've just thought of someone whose feet I just know I'll be able to picture. I have been sitting at my guru's feet once a week for twenty five years." (Benton belonged to a spiritual community in New York.)

Benton closed his eyes again. His voice softened, and he continued, "I can picture my guru's feet. I have spent so many hours sitting at my guru's feet; I can picture them perfectly. I can see them when I close my eyes." He started describing to me what they looked like as he conjured up the mental picture. "They are soft, they are supple, they are so perfect. His feet are golden brown, I can see them perfectly...and (Benton's voice filled with horror), *there's a big black hole in the middle of his ankle!*"

Not just the feet

Some PDers were unable to mentally observe, fill with light, or otherwise acknowledge other injured body parts, not just the injured foot. I recall one PDer whose mental image of his own body showed the lungs being filled with blackness. When we talked about this, he recalled that when he was five years old, he had been sick in hospital with pneumonia and given up for lost. The priest had been called to the hospital to administer last rites.

Now, forty years later, and with no conscious worries about his lungs, he could not even imagine a single ray of light penetrating the thick murkiness that appeared when he tried to visualize his lungs. Also, as he tried this exercise, he was aware of fear in his heart that was associated with both this imagined blackness of the lungs and his PD tremor.

Very often, a PDer can imagine light flooding down a limb until it comes to the part of the limb that was injured long ago *or* it arrives at a body part that is no longer functional due to encroaching Parkinson's. Therefore, many people with no history of hand injury, but with a hand that has recently started tremoring, may find that they can only imagine light halfway down the arm. As their mental searchlight nears the area of the tremor, an area of relatively new fear (fear

due to tremor and loss of physical control), they find that they cannot penetrate the Stygian darkness.¹

All excited over a dead-end

When we realized the extent of PDer's inability to visualize, we wondered if we'd stumbled on the answer to the partial recovery problems. Were these people anxious *because* they were mentally missing a body part? It seemed plausible enough that a person might have anxiety about using a body part that didn't exist. Maybe, on some deep down level, the missing body part was itself the source of the anxiety.

If this was the case, we could gently, slowly, help these people to acknowledge the missing body part, and the anxiety would go away! Hooray!

But fixing the visualization turned out to be problematic. Even if we struggled for an hour or two each week, helping the person with word and deed to create a mental image of light in his "off limits" area, the following week the area would be sealed off even more tightly. If we asked the PDer to practice during the week the visualization exercises we did in session, they usually reported, during the next week's session, that they had been unable to do it on their own.

Something else was going on that was causing these areas to be mentally inaccessible. Merely opening them up wasn't enough: they usually shut right up again *even harder* within a few hours, or certainly within a day or two.

It was curious that, during the moments of visualization, recovering PDers could often move much better than usual. However, the improvement in movement subsided as soon as the day came to a close, or sometimes as soon as the person left the treatment office.

Techniques that didn't work

After we realized that many PD patients were unable to acknowledge certain body parts, we set to work on finding ways to clear away their mental blockades. For over two years, we worked on asking PDers with "blacked out" areas to enter into their forbidden zones via an assortment of loving, friendly, self-accepting methods. We had them visualizing light, breathing into the area, asking their heart to heal their feet, imagining the area expanding or radiating with vigor – you name it. We tried all sorts of techniques.²

¹ We had to wonder whether or not the inability to imagine light in PD-damaged areas (areas of atrophy or rigidity from aberrant Qi flow) came about because of the cellular damage from wrong flowing Qi or if, conversely, that body part became more susceptible to aberrant Qi flow because the PDer was slowly, subconsciously adding body parts to his collection of off-limits, unknowable, unlightable body areas. Our findings now suggest that it's some of both.

It appears that events such as tremor are able to begin when a body part, due to disarray of Qi, finally severs its mental connection. In the case of tremor, when the atrophy in a limb becomes great enough and the disassociation becomes severe enough, the limb begins to move in time with the long-standing internal tremor. Experiments in which some PDers have been able to successfully integrate a "detached" body part back into the fold show that the tremor in that body part can cease so long as the mind maintains conscious awareness of that body part. However, if an internal tremor is still lodged in the brain, the tremor will return as soon as the consciousness reverts back to "normal."

² Because I am often asked specifically about NET and EFT (Emotional Freedom Technique), I will note that we did have a number of PDers try these techniques repeatedly using various practitioners. The results ranged from fleeting to zero. These techniques help a person get past certain feelings that were creating emotional blockages or pain. But as we were to learn later, PDers who had dissociated from their hearts didn't feel that they

Most PDers spent hours struggling to do these techniques. Most of them hated the processes or were even repulsed by them and refused to do them. Others really tried their best at it but their hearts weren't in it, so to speak. Even those who were able to finally fill an area with light, talk to their injured feet or insert images of healing icons into their off-limit places found that these exercises were pretty much a waste of time; within a matter of hours, the dark places would be inaccessible once again or the darkness would be discovered lurking somewhere else nearby, having migrated. Some people struggled mightily to get light into an area and were somewhat successful. However, we soon realized that, in most cases, these people had merely created an imaginary layer of light and sweetness over the dark areas.¹

We were no better off than before. This discovery of inability to visualize seemed, at the time, like a dead end.

The Parkinson's Treatment Team

Continuing on with the chronology, about this time a group of Santa Cruz (California) acupuncturists, students, and I formed what later became the PD Treatment and Research Team, also known as the PD Team.

One purpose of the team is to provide, for a moderate fee, a week or two of treatments for PDers who want to travel to Santa Cruz to briefly experience first-hand the protocols we use. A

had problematic feelings or blockages; they didn't feel much of anything. And as for the simple *heart*-calming techniques of the Heartmath Institute, they were completely incomprehensible to the PDers who on whom we tried them. PDers couldn't even figure out how to follow the directions. Too much visualization was involved.

¹ Because the reader, especially if he has Parkinson's, may be saying to himself right about now that these mind-body disassociations must only be problems for "really messed up people" and therefore will not apply to him, I want to share one quick experience with a PDer's inability to perceive light in her mildly tremoring arm.

One sunny afternoon, I was describing the inability to mentally visualize injured body parts with a PDer whom I had just met at a reception. She had been working with an FSR practitioner for a few months and felt she was making some progress. I wanted to let her know about some of our latest findings, especially the significance of what we were calling at that time "a mind-body disassociation."

She told me, in no uncertain terms, that if a person can visualize the healthy side of his body but not the PD side, then that person must be crazy, probably even psychotic. "That's completely ridiculous," she sneered. She then told me, with a bit of a swagger, that if anyone could recover from Parkinson's disease, it would be her, "...because I'm a Buddhist. I meditate." She was blond haired, blue eyed, and her native-California accent was bold with certainty. She had in full measure that self-confidence part of the Parkinson's personality that assured her that she could master any difficulty if she really put her oh-so-forceful mind to it.

So I asked her if she could imagine light glowing inside her nose. She paused, crossed her eyes, closed her eyes, and mentally focused on her nose. "Yes, of course I can imagine my nose full of light. I meditate. I'm a Buddhist, like I said."

"That's fine, that's great," I replied. I had noticed that her right hand tremored but not her left.

"Can you picture light in your left arm?"

There was a pause while she closed her eyes and focused on imagining light in her left arm. Then she declared, with faint exasperation, "Of course I can. I meditate every day."

So then I asked her if she could do the same with the right arm. She assured me she could, and then closed her eyes. A few seconds passed, and then a few more. After a longish pause, with her eyes still closed, she announced, "Well, I can tell I have an arm." I asked if she could imagine light in her arm. There was a longer silence.

"Well, it's harder than the left, but I can do it." She remained very still, with her eyes still closed. Her breathing rate slowed, as if she was concentrating deeply. Then she said, very slowly, and with a voice that had lost much of its brashness, "I can't see anything. There's nothing there. There's nothing but darkness."

more important function of the Team is that we include, with the treatments, free training for the patients' accompanying health practitioners.¹

Another very important function of the Team is our weekly Team meetings. We discuss the visiting cases and our own difficult cases from our private practices. We plan treatment strategies and work on honing, simplifying, and translating into common English our explanations of the treatment principles we're using. Visiting practitioners can attend these meetings and learn about the individualized way we approach each case.

The goal of these meetings and the treatments we provide is not to cure as many people in the world as possible: we are too few, and people with Parkinson's are too many. Our goal is this: by experimenting with variations on treatment techniques, by experimenting with ways of training the visiting practitioner, by finding the best ways of communicating what we know about the cause and treatment of Parkinson's, by understanding to the fullest the core underlying cause of Parkinson's *and* the sources of its individual, seemingly infinite, variations, we hope to learn as much as possible and share our findings with as many people as possible – via our free web-publications.

Our hope is that this book will allow the greatest number of concerned friends or health practitioners to become self-trained in the simple art of Yin Tui Na and the techniques we use for helping PDers overcome their mental/emotional blockages. In this way, the greatest number of people can be treated.

Our goal is *not* immediately to challenge and overthrow the way that western doctors understand and treat Parkinson's. We understand that changing any medical treatment paradigm takes at least twenty years: long enough for the medical students of today who stumble across radically new research to become the med-school teachers of tomorrow. But for people who have Parkinson's disease today, twenty years is too long to wait. So, although challenging the current medical paradigm is not our goal, we do feel that those who are looking for alternatives should be able to avail themselves of our findings. Therefore, our approach is to make freely available, via the Internet, research that PDers themselves can use today.²

¹ For more information about the PD Team, please visit the website at www.pdtreatment.com

² Dr. Fred Jones, in his continuing role as advisor to the project, assured me that, because our work is so counter to prevailing thinking, and because there are no definitive tests for Parkinson's disease, all of our recovered patients will be considered, by western doctors, to have been misdiagnosed. Therefore, we should not promote any one patient or any ten patients as being "proof" of our findings. As he pointed out, "All it would take is one doctor, a doctor who's never even met you or the PD patient, broadcasting to the media that your patient was obviously misdiagnosed and the world will take his word against yours – every time.

As mentioned earlier, Dr. Jones advised that, although the single-case study is a valid method for reporting one person's response, we must, to change a paradigm, rely on large numbers: when we have a thousand people who have recovered, we can make a strong case for our findings. Until then, every person who recovers will necessarily be considered, from a western standpoint, as an anomaly: a case of misdiagnosis.

The "large numbers" method is a not uncommon approach to changing a paradigm. When Pasteur was being widely ridiculed for his germ and immunization theories, his first victories against infection were considered circumstantial. It was not until he inoculated an entire flock of sheep that his theories caught on. His inoculated sheep grazed in the same infested pasture as a non-inoculated flock. *All* the non-inoculated died. *All* the inoculated remained healthy or suffered only mild, passing symptoms of the disease. Even this did not convince everyone – especially not the doctors – but farmers began lining up for the inoculations. After that, the doctors fell in line whether they believed or not.

Large numbers and patient demand are what change paradigms, not excellent logic or proof on a limited scale. The other thing that changes treatment paradigms is, of course, money: advertising a product whether it works or not can quickly change a paradigm. But there is no product of profit to be made for anyone in our findings: the

The clinic: 1999 through 2002

The PD Team was an outgrowth of the free Parkinson's clinic that I started up in Santa Cruz.

From spring of 1999 to December, 2002, I ran a free Parkinson's treatment clinic at the acupuncture college where I teach. At this clinic, student interns from the college provided free treatments for local people with Parkinson's. The clinic also hosted one guest per week from outside of our local area. Guests came from around the world to present their Parkinson's symptoms to the class and be treated by student interns.

The clinic patients, our private patients, and other patients from afar with whom we worked closely, if intermittently, were the basis of a four-year observation project in which, without planning to, we came to understand the workings of the Parkinson's medications in a much more intimate and accurate way than *any* prior scientific study.¹

We kept close observations (at least an hour per week of interview in our offices – and many patients also kept logs and charts) on over a hundred patients who were being treated, of whom over 65% were medicated. We discovered alarming, in a few cases, fatal, differences in the recovery patterns of medicated patients when compared to unmedicated patients.

This project culminated in our decision to *not* work with PDer's who had ever used any dopamine-enhancing medications for a period longer than three weeks. The results of this project led to a book, published in 2003, which describes in extreme detail our discoveries about the workings of the various anti-parkinson's medications.²

In late 2002, we established a new policy: we would not perform, or provide informational support for, recovery therapy on medicated patients. I thought at the time that the new policy was very clear. I thought that it explained the unreasonable risks involved in recovery

treatment is easy and can be done by anyone. The fact that there is no big money to be made from our findings will probably slow, rather than speed, any paradigm shift in the treatment of Parkinson's disease. On the other hand, PDer's tend to be self-starters, so possibly they will simply leave the MDs behind on this one.

In order to promote our findings, we assume that we must work at the grassroots level until a thousand or more PDer's have recovered or until western doctors agree on a definitive diagnosis for Parkinson's. If and when they have such a thing, we should be able to prove that we are reversing PD by showing a reversal of the test parameters in those PDer's who recover. (In case you are wondering, PET scans, though helpful, are not yet accepted as definitive by the medical community. The results of PET scans often conflict with doctors' diagnoses of Parkinson's disease.) And so, as yet unaligned with the western medical community, we continue to work towards our goal: doing research and providing information so that the most people can be healed in the least time.

¹ Based on a new understanding of how these drugs work in the various brain areas, we became able to predict exactly how and when the various side-effects and On-Off patterns would be triggered in any PDer taking the drugs. Prior to our new hypothesis that the drugs were uptaken and released in three different brain areas at three different rates, the onset and wear-off timings of the drugs and their side effects had always been considered unpredictable, especially when changing dosages. Using our new hypotheses, we were able to make accurate predictions several weeks in advance about the upcoming changes in On-Off timings and side effects of the drugs in response to changes in dosage – predictions which were then borne out by the un-notified PDer's.

² Walton-Hadlock, JL, *Medications of Parkinson's Disease or Once Upon A Pill: patient experiences with dopamine-enhancing drugs and supplements*. Parkinson's Recovery Project, 2003. 638 pages. Available for free download at www.pdrecovery.org.

of PDerS who had ever used dopamine-enhancing medications for more than a few weeks: PDerS in whom drug-induced brain changes had most likely already occurred.¹

However, despite my strong warnings, many PDerS who had been taking medications for a long time chose to believe that what I actually meant in my book was that we *would* help them if they *stopped* taking their medications.

To counter this wrong thinking, we placed a draconian statement on our website in 2005 stating in no uncertain terms that we would not work with any person who had *ever* taken dopamine-enhancing medications for more than three weeks.²

This policy change marked a major step forward in our research. Up until late 2002, we'd spent most of our time dealing with the horrors that developed as medicated PDerS found out the hard way what we meant by "hideously addictive," as in "After the foot injury heals, the medications may, within 72 hours, become hideously addictive. Even if a person has already stopped taking the medications, the unaccustomed physical and emotional pains of recovery may cause the PDer to look back with longing at the medications. Thinking that, because he got off the drugs easily the first time, he can start or stop them again without a problem, he may find

¹ Putting it very simply, the risk is this: after a person has brain damage from the drugs, he may always have symptoms of drug-induced parkinsonism – and there is no cure at this time for this syndrome. This syndrome is a degenerative one and may not be obvious in the early stages. Eventually, a person with this drug-induced parkinsonism may need to take dopamine-enhancing drugs to ameliorate his condition. However, the drugs are much more dangerous, more addictive, and sometimes even deadly in a person who does *not* have *idiopathic* Parkinson's disease.

The drugs can be somewhat benign *if* dosed correctly *if* a person actually has idiopathic Parkinson's disease. The drugs are quite dangerous in a person who does *not* have *idiopathic* PD, **even if he does have drug-induced parkinsonism**. A person who *does* have idiopathic PD and who has been using the drugs for more than a few weeks may have already sustained some drug-induced brain damage. Therefore, he may need some amount of drug help down the road to help with his degenerative, steadily worsening *drug-induced* parkinsonism. These drugs will be less dangerous if he still has idiopathic PD. A drug-using PDer may therefore be better off in the long run – at lower risk for drug-induced mental illness or drug-induced agonies – if he maintains the brain-protective qualities (protection from the drugs) provided by idiopathic PD.

This is why we say that a PDer who has ever taken dopamine-enhancing drugs for more than a few weeks may be better off, in the long run, if he does not try to recover. We also say that every person is an individual and must follow his own heart with regard to drug use. While we would not dream of telling a person what to do or not do with his drugs, we will not treat nor give any advice whatsoever about treatment to a PDer who has ever used dopamine-enhancing drugs for longer than three weeks.

PET scans in the famous EllDopa study of 2002 compared the levels of brain change in recently diagnosed PDerS who took low, medium, or high levels of a dopamine-enhancing drug for a period of 40 weeks. The amount of brain changes at the end of 40 weeks corresponded directly with the dosage level of drug. The control subjects who were taking placebos had only the very low amount of brain change that is expected, over time, in people with Parkinson's disease. Those at the highest levels of drug use showed the most brain change (damage).

On the other hand, a person who takes the drugs who does *not* have *idiopathic* PD can experience *very* rapid, *very* dangerous levels of brain change, and may, in some cases, be visibly suffering from the side effects of the drugs within a few months. Although the drugs do cause brain damage in people with idiopathic Parkinson's, they cause much more damage, much more quickly, in the brain of a person who does not have, or who no longer has, Parkinson's. Therefore, because people with (currently) incurable brain damage from drug use may someday need to take antiparkinson's medications, we will not work with people who have ever taken the medication for more than a few weeks.

² Even so, we still get inquiries from people who have taken the drugs for many months, or even more than a decade, asking if we will make an exception and work with them if they get off their drugs.

that, in his changed – and in some cases, seemingly worse – condition, the drugs will have become hideously addictive.”

Although we continued to work with a few recovering PDers who were already in our program and who had gotten off the medication, we never again knowingly took on a new PD patient who had used antiparkinson’s medication for more than three weeks.

This meant that we no longer had a mishmash of euphoric (recovering and drugged) and panicked (partially recovered) people in various stages of recovery. With the euphoric ones gone, now we could see clearly that the unexpected mental changes in our never medicated, partially recovered PDers were not related to the overmedication psychoses that we had seen.

We were seeing in partially recovered PDers the unexpected problems of poor proprioception, partial numbness, lack of injured-side-of-body awareness and the mental inhibition of positive imagination and/or positive visualization.

We were seeing these problems steadily, and we were seeing them whole – we thought. Attempts at directly addressing these problems seemed, in many cases, to make them worse instead of better. Clearly, these problems were not root causes, but were stemming from some deeper form of illness.

We had no idea what to do about it. But at least we were no longer dealing with the distracting complications from antiparkinson’s medications.

Placebo research and an increased emphasis on positive attitude

At around the same time, I started reviewing placebo research related to Parkinson’s disease. This research suggested that dopamine release in PDers was highly susceptible to positive or negative mental suggestion. Positive expectations cause dopamine release; negative expectations inhibit dopamine release.

People with advanced Parkinson’s who had been in sugar-pill placebo studies were often able to move normally if they thought – mistakenly – that they had been given their usual antiparkinson’s medication.

Based on the findings of placebo researchers, we were increasingly concerned that PDer’s reluctance or inability to imagine their own body or, for that matter, any positive outcome involving the body, might be perpetuating the inhibition of dopamine release. Even if the foot injury was healed, Qi was running correctly, and dopamine release was potentially possible, dopamine could not be released if a PDer had a negative attitude about his body or his ability to expect joy.

We started asking patients with increasing fervor to try to visualize their own bodies and to work on their attitudes.

They often responded vehemently against to our suggestions for cultivating positive attitude and expectation. The best way to summarize the dominant attitude was “I don’t want to change. I want you to fix me so that I can go back to being exactly who I was.”

Patient responses to the placebo findings

I clearly recall what happened when I made these suggestions to a patient whose major complaint was that the new pain in her hip prevented her from taking a normal step: she had to

drag her leg because any movement in the hip joint was excruciating. I'd been working with her for nearly two years and she had made much progress. Energy was once again coursing down from her neck, over the torso, past the hips, and through her foot. The nerves in her body were coming back to life. The more feeling she got in her foot, the more feeling she got in her hip, as well.

Her hip had evidently been injured at some point in the past. Before we started treating her Parkinson's, her hip had been numb. As the nerves in her hip began to resume function, the nerve signals coming from her hip were extremely painful.

In the past, I'd treated her while she passively rested. Because of our new concerns with patients' negative attitude and patient inability to imagine or feel a connection with body parts, I asked her to please try and focus her attention on her left hip while I supported the hip with Yin Tui Na.

She said no.

I said that her hip wasn't going to be able to heal very easily if she refused to acknowledge that she had a hip.

She lashed out at me, "No way! What don't you understand about pain? I am *not* going to think about anything that hurts. The whole point of life is to avoid pain. The whole point of life is to not have pain. You must be crazy if you think I am going to make myself think about the very thing that I'm trying to avoid."

I suggested that maybe, if she sent her attention to hip, she might find that the pain was slightly less. Pain is a call for attention. Very often, if a person calmly focuses on his pain, the pain signals decline. Oppositely, when a person frantically seeks distractions from his pain, the pain becomes more insistent. I suggested again that she might need to pay a little attention to it.

She exploded with rage. "I just told you, the *problem* is the hip! I am *not* going to focus on a problem. My whole game plan is about avoiding even knowing that I have a hip. Your job is to fix it, my job is to not know it's there."

I replied that the body can't heal something that it doesn't know exists, that it is consciously denying. She countered that her job was to find the doctor, the doctor's job was to heal the problem.

She never came back.¹

¹ As a curious aside, this patient felt, as many PDers do, that she was deeply spiritual, a deep thinker. I asked her if her spiritual seeking ever led her to join any particular religious group. She replied that she had gone to a church once, but as she looked around the people in the pews, she could tell at a glance that none of them were perfect, and why should she spend time with people who weren't perfect? So she never again went to a church. I asked her if she was perfect, and she replied that she didn't know, but that *she* was at least trying to be.

While this little vignette may seem like a silly response from one individual, her responses were actually very revealing, especially because they were so similar to the responses of many other PDers. The idea that other people are potentially "bad" and that the PDer is nobly trying to be good despite obstacles, is not uncommon amongst PDers. I have to suspect that the sympathetic nervous system, which requires one to maintain vigilance, which elevates the ego to a position of importance over the heart, and which gives out a steady stream of "be good and be careful" commands, is the culprit.

These people are not purposefully trying to project spiritual arrogance: their out-of-control ego, being commanded by the perpetually "operating at full bore" sympathetic nervous system, has no other way in which to operate. The parasympathetic system, which allows a person to be relaxed, amused, and observe himself as a tiny part of an enormous, and perfectly fair and balanced universe, is barely operational in PDers. When PDers recover, they experience a glorious personality shift. Some even say that they have become human again, for the first time in decades.

Other PDers also dropped out of the program when we shared the placebo information and introduced the idea that patient attitude played a role in triggering dopamine release. We suggested that, maybe, the patient should take some responsibility for his attitude, or that he must be willing to do a little work, if necessary, to mentally acknowledge that his injured body part did actually exist.

It seemed reasonable to us. But many patients dropped out rather than participate in exercises designed to help with positive expectation.

Meanwhile, a PDer in our program had created an Internet chat group called PD Recoverers. Some wonderful friendships had developed through the chat group and, though I had never visited the site myself, it seemed like a great resource for PDers.

My understanding was that it was started as a site for people who were in our program so that they could compare notes. When people started dropping out of the program when we suggested that they might need to make mental or emotional adjustments, quite a few of them turned to the chat group.

A few of my continuing patients told me that, thanks in large part to Recovery Project dropouts, the chat group had taken a very negative turn.

Over time, even as the chat group remained a wonderful source of mutual information and support for some PDers, it also became a regular forum for hostility towards our program.

This type of discussion is all a part of the lively give and take that accompanies all scientific inquiry. But PDers who were just discovering our website often visited the chat group as well. Then, when they communicated with us for the first time, they often opened their queries to us with, “Before I learn any more about your program I want you to explain all the bad things I’ve heard in your chat group.”

We had to explain that we have nothing to do with the chat group, and that we have not even visited the site.

But this just added to our ongoing frustration that some people had recovered and others had not. And it even seemed as if those who were the most adamant that they shouldn’t have to cultivate a positive attitude or expectation were the ones who had the worst experiences in partial recovery, in mood related lapses into movement inhibition and tremor.

“I need to talk with someone who’s recovered”

Another emotional stumbling point for many PDer was their conviction that they *needed* to cultivate negative attitude to prevent the development of false hope. Many of these people told us that the only way to counter this negativity was meeting up with people who had already recovered.

The pioneer patients had recovered without any examples of people who had recovered. But as the project began to grow, many PDers told me that they could not have a positive attitude unless they met someone who had recovered. In the early years of this project, this seemed logical to me. I made arrangements so that most of the PD patients who came to visit in the early years met at least one person who had recovered from Parkinson’s.

However, despite their statements that they needed to meet a recovered PDer in order to have a positive attitude, the negative-mindset PDers uniformly had *no* shift in attitude from meeting a recovered PDer. Instead, they became *more* doubtful. Their remarks were usually

something like “Just because that person recovered doesn’t mean anything about me. I’m different,” and “That person seems perfectly healthy: obviously he never actually had Parkinson’s disease. He must have had a very mild case, or else he was probably misdiagnosed.”

I quit arranging meetings with people who had recovered.

Where are the recovered people?

Some PDers were highly suspicious of the program because recovered PDers weren’t making speeches or appearing on Oprah. I explained the problem: people who recover may be told by their doctors that they had been misdiagnosed; the ex-PDer might be told that he’d only had a pinched nerve or a bad case of Bell’s Palsy. They might be told that their Parkinson’s symptoms had been manifestations of neuroses. Certainly, they will not be told that they recovered from Parkinson’s disease.

It is rather daunting for a person to stand up and say, “I know in my heart that I recovered from Parkinson’s, but my doctor says that actually, I’m just a nut case.” Instead, people who recover have two choices: they can be bitter towards the doctor who “misdiagnosed” him and who made him worry and seek alternative treatment unnecessarily, or he can rejoice privately because he knows that he actually was successfully treated for a condition that is *not* and never has been incurable.

Also, the dominant emotions of people who recover from Parkinson’s are not necessarily feelings of pride or victory. Sheepishness, humility and gratitude are often the strongest feelings for many people who recover. They may go into the program intending to be victorious. But during recovery, they may realize the extent to which they have lived a life dominated by a drive for victory, intertwined with intentional numbness and negativity (which never seemed negative at the time, but seemed like heightened logic and efficiency). In the end, they are humbly grateful for recovery – but they are more sheepish than proud.

The end of 2002

We were relieved that we weren’t going to have medication horror stories any more. We were grateful that some people had recovered from Parkinson’s. We felt that we had somehow failed the many partially recovered PDers who had dropped out. We were pleased that some members of the Parkinson’s Recoverers chat group had formed supportive friendships. We shrugged off the news that some members of the chat group were posting bitter remarks about our program. We were concerned that so many people were hostile to the very solid western research showing that dopamine release in PDers is almost completely mood and expectation dependant. We were determined to continue.

We did not yet realize that PDers were locked into sympathetic (danger) or dissociative neural modes. We did not know that, in this mode, a person *cannot* visualize happy endings, cannot easily access the creative, imagining, pretending parts of the brain, cannot let his guard down. We did not know that the most important symptom of the dissociation response was the numbing of the flesh and the resulting inability to truly feel, to the fullest, the existence of the body. (In a selective dissociation response, the numbness can be, in the beginning, limited to just an injured area. Over time, more body parts may become incorporated into the “off limits” area of the brain.)

We did not know that a person who is locked into a dissociation response with regard to one arena of his brain will often learn to subconsciously use dissociation in response to other, less threatening negative events.

We did not know that a person receiving a diagnosis of Parkinson's disease often feels betrayed by his body. Then, since his body has betrayed him, the PDer mentally applies the same dissociation technique to his body that he applies to anything he doesn't trust: he dissociates from it. We didn't know that the rapid decline that some people experience following diagnosis with Parkinson's disease was a part of the same process of dissociation and denial that they use for anything they don't like. There was much we didn't know.

Also, the placebo research was strange and powerfully suggestive of a mental/emotional angle. And though most of our partially recovered patients deeply resented or struggled mightily with the impossible idea that they needed to cultivate a more positive attitude, we wondered, nevertheless, if something in the mind or emotions might hold a clue to the mystery of the partially recovered PDers.



*How long, how long, in Infinite Pursuit
Of This and That endeavour and dispute? “*

The Rubaiyat of Omar Khayyam

CHAPTER THIRTY

STILL PLUGGING ALONG: 2003 TO 2006

2003

No sudden shafts of wisdom had shown up to explain what we were now referring to as the mental/emotional blockage of Parkinson's disease.

The severe anxiety, inability to visualize light, and inability to really relate to the “other” (the injured) side of the body didn't seem to respond to any treatments. Not only that, most of the PDerS responded to the various mental/emotional treatment ideas as if they were anathema. Clearly, we had not gotten to the root of the underlying problem.

Some members of the growing ranks of partially recovered PDerS clearly felt that we hadn't done a good enough job. Nearly all of them felt that, despite hundreds of small improvements, they still officially had Parkinson's disease. So long as *any* PD-like symptoms showed up, anytime, anywhere, it was usually held as proof that the PD was absolutely unabated. Improvements did not count. Any remaining symptom, no matter how small, was regarded as proof of *worsening* Parkinson's.

And it was no matter that their remaining symptoms were now clearly initiated by *their* own emotional and mental ups and downs; they wanted *us* to finish fixing them.

In one lovely example, I was working a PDer who had recovered in many ways, but who still refused to spend any time noticing that he did, in fact, have a left arm. When he arrived for his weekly session, I asked if he had spent any time at all in the last week turning his thoughts to the existence of his left arm, trying to connect with it somehow – using any of the more than five techniques I'd offered him the week before.

He laughed and said that he'd forgotten. Then, in all sincerity, he said, “Can't you do that part for me?”

When I asked him how I was supposed to change his thoughts and make him acknowledge the existence of his own arm, he said, simply, that he didn't know what I might do, but *he* certainly wasn't going to do it.

Another ex-PDer, *completely*, not partially, recovered, came in for his monthly visit. He was once again working on his juggling, exercising several hours a day, and very much enjoying his retirement, although recently, as a favor to a friend, he had taken on a part-time job as an editor. When we'd first met him, he was nearly paralyzed, unable to drive, hunched over, with no facial expression.

He proudly told a PD Team member, Chris Ells, that he'd sent away for a new book, a book about exercise for people with Parkinson's. The book features exercises for people who aren't moving well, to help them stave off encroaching immobility.

When Chris asked him why, since this book was clearly inappropriate for him, he still wanted to identify with having Parkinson's disease, the ex-PDer was stunned. He had assumed that Chris would be pleased that he was continuing to fight the Parkinson's. Instead, Chris wondered sadly out loud just how long the ex-PDer wanted to identify with an illness that he no longer had. The ex-PDer replied that he didn't know. I asked him why he felt he still had Parkinson's. He said that he now took naps in the afternoon. He'd never taken naps prior to his diagnosis of Parkinson's disease.

He then completely stunned me by saying, since he still took naps in the afternoon, should he look into assisted living? Should he stop living on his own, admit that he was never going to get better, quit his job, and give up trying to recover?

I was speechless.

Frustration and bafflement

Between these two attitudes, recovered PDers who didn't want to let go of their diagnoses, and partially recovered PDers who didn't want to do the admittedly hard work of changing their feelings towards their body, we were feeling frustrated. What had all of our work actually accomplished? The people who recovered had gone on their merry way. Their doctors had told them that they had been misdiagnosed. How could we argue with that? And those who had turned into emotional basket cases certainly still had something wrong, even if it was no longer a neurotransmitter deficiency.

We were certain it couldn't be a neurotransmitter *deficiency* problem once a recovering PDer was able to move absolutely normally *when* he was feeling good: moving with no signs of cogwheeling, foot dragging, and slumped posture. Even if, when frightened, he reverted to a spurt of shuffling, no facial expression, and tremoring, we weren't willing to call that a "temporary display" of idiopathic Parkinson's disease. Heck. I had seen people shuffle, stare dumbly, and tremor violently during the cold and achy stage of flu. People can behave the same way when in shock.

These PD-like symptoms, movement inhibition and tremor, when occurring because of flu or shock, are caused by the temporary inability to release dopamine, not because of an underlying dopamine insufficiency. And we know perfectly well, based on placebo studies, that dopamine *release* is expectation dependent. A person's brain releases, at any given moment, exactly as much dopamine as he *thinks* he needs to express his feeling of joy, and no more.

Feeling safe causes an initial surge of dopamine in the midbrain. Then, joy, a feeling of joie de vivre, the sheer joy of being alive, reinforced the safe feeling and leads to a continued release of dopamine. Dopamine does *not* make people joyful. Being joyful allows the release of more dopamine. That release of dopamine may further trigger other thoughts and behaviors that lead to an increase in happiness, but the initial jolt of dopamine, if it has stopped flowing in response to fear, is released in response to feeling safe. After the fear is gone, the expectation of joy can initiate dopamine release.¹

¹ The reason that alcohol, methamphetamine, cocaine, opiates, nicotine, and antiparkinson's medications work is that they jump-start the dopamine release process. Instead of relying on one's own feeling of safety or feeling of joy to release dopamine, which can then trigger the thoughts and behaviors that release still more dopamine, one can use these artificial dopamine-enhancers to get the ball rolling. However, because they are addictive, they set in motion brain changes that raise the bar for the amount of dopamine needed to set off the spiraling good feelings and moves. After the drugs wear off, even more dopamine than before is needed to trigger the first manifestations, the motor and thought expressions, of safety or joy.

When a person has a bad case of the flu or is in shock, he is not able to feel his usual amount of joy. Therefore, he shuffles and mumbles and tremors and loses his balance. These are common symptoms of any condition that causes a temporary inhibition of joy.

Increasingly, it was looking as if the partially recovered PDers had some problem that prevented them from having the basic joy of living that allows for steady dopamine release. Not only that, it seemed as if they also weren't able to release sufficient adrenaline to deal with their little daily fears. What these people now had was an inability to release adrenaline *or* dopamine in adequate quantities, except during those times when they so far forgot themselves as to accidentally enjoy themselves. Then, they could move normally. But when they remembered that they were supposed to be worried, critical, or wary, or still have Parkinson's, they also lost the ability to release sufficient neurotransmitter for effective thinking or movement. Baffling. And how did this relate to so many PDers' reluctance – even refusal, in some cases – to visualize, imagine, or have a sensory relationship with the injured sides of their bodies?

Still, we consoled ourselves, our work might not have been been pointless. If nothing else, the discovery of Rebellious Qi in PDers might someday be significant in helping to diagnose Parkinson's.

We were still pleased about the hypothesis of Rebellious Qi and its relationship to Parkinson's. We even felt that, if nothing else, it further proved the existence of channels and it might serve as a diagnostic tool for idiopathic Parkinson's – a notoriously difficult illness to diagnose correctly. This next bit explains a bit about how we came to trust Rebellious Qi as a good diagnostic tool.

Using the foot injury/electrical flow reversal as a diagnostic tool

We are frequently asked if *all* the people we see who've been diagnosed with Parkinson's have the foot injury and the electrical flow problem.

The answer is a qualified yes. Early on in the project, we saw, as many neurologists can attest, that there is much misdiagnosis in the realm of Parkinson's disease. A significant percentage of the people who came to us for treatment clearly did not have Parkinson's. Sometimes this error was due to self-misdiagnosis by unqualified patients or their friends, but it was more often due to sloppy work by the diagnosing neurologist. Many times we sent questionable PDers to get a second opinion, and the second opinion often came back negative: *not* PD.

We developed a new diagnosis procedure at the free clinic. A six-to-eight person panel observed a patient during his intensive, one- to two-hour intake that put the presumed PDer through his paces. We looked for all the classic, western-medicine recognized symptoms of idiopathic Parkinson's disease and asked a long list of questions, even before we started examining the foot or leg. Following the rigorous intake, the panel (made up of the core health practitioners in the free Parkinson's clinic, all of whom had studied Parkinson's disease as a specialty in school) voted on whether or not a person appeared to have Parkinson's disease.

This all took place *prior* to checking the foot and leg. The voting had three options: Yes, Parkinson's; No, not Parkinson's; and Uncertain. The opinions of the panel were usually, though not always, unanimous. If the vote was not unanimous, we did another round of examining and asked more questions.

After the voting, we felt the feet and assessed the Qi running in the Stomach channels of the patients.

Slightly more than twenty-five percent of the people who came to see us for their “Parkinson’s” appeared to have been misdiagnosed. This number, twenty-five percent, was close to the results of autopsy-based determinations of Parkinson’s. That study showed that twenty percent of the people who had been diagnosed with Parkinson’s were found, during autopsy, to actually have had a different syndrome (i.e., twenty percent had been misdiagnosed).¹

Using our voting-on-the-symptoms method of diagnosis, we arrived at about the same percentage misdiagnosed as the various autopsy studies: we also found the same percentage of misdiagnosed when we subsequently used disrupted Qi in the Stomach channel to form a diagnosis. Most importantly, the patients who, based on symptoms, did *not* seem to merit a diagnosis of Parkinson’s disease were the same ones who did *not* have indications of unhealed foot injury nor did they have electrical currents running backwards in their legs.

On the other hand, all of the people who clearly had classic symptoms of idiopathic Parkinson’s disease, as defined by the standards of the American Academy of Neurology (AAN), *did* also have indications of unhealed foot injury and *did* have electrical currents running backwards in their legs.

So, in terms of numbers for people who clearly had classic Parkinson’s, our hypothesis about the foot injury and leg currents was holding up. By 2003, after examining hundreds of PDers, we started to feel tentatively confident that foot and Stomach Qi assessment might be considered a reasonable method to help confirm or deny a diagnosis of Parkinson’s disease.

In the midst of this jumble of people with questionable diagnoses that came to us looking for help, it seemed as if, in the case of those few diagnoses that were unclear or questionable, the foot injury/backwards-running Qi test seemed to be a sort of acid test. If a preponderance of evidence suggested non-PD, the foot and leg test supported us. Oppositely, even in cases where people seemed as if maybe, possibly, they were heading towards Parkinson’s but their symptoms were as yet very mild or intermittent, the foot and leg irregularities were usually solidly established. (These foot and leg Qi-reversal symptoms may have been evident for decades prior to the visible onset of Parkinson’s, as in the case study of Tim in the first chapter.)

¹ “New AAN Guidelines Released For Parkinson’s Disease,” *Neurology Today*, Vol 6, no.7, April 4, 2006, p. 8. When I first started on the Little Project, the percent of misdiagnosed PDers, based on autopsy studies, was nearly 30%. The newer studies suggest a lower number: twenty percent. The percent of misdiagnosed is changing, but this is probably *not* due to an improvement in diagnostic skills. This change may be because of PDers taking medications. Nowadays, most PDers are encouraged to take medications as soon as they are diagnosed (despite decades of research proving that PDers who delay taking medication can also delay the onset of the dreadful adverse effects). What the autopsy people fail to take into account is the fact, proven in the EllDopa study of 2002, that the medications themselves cause brain changes. If a person takes antiparkinson’s medication for several years, his brain is likely to show signs of dopamine decrease *whether or not* he actually ever had idiopathic Parkinson’s disease. A misdiagnosed person who takes antiparkinson’s drugs may develop changes in his brain that resemble Parkinson’s disease. Therefore, the autopsy study will see the drug-induced brain damage, and declare, incorrectly, that he had been correctly diagnosed with Parkinson’s.

Also, antianxiety and antidepressant drugs may cause changes in the brain resembling those changes that occur in drug- and toxin-induced parkinsonism. Because the autopsy people are only looking for signs of decline or dormancy in certain brain cells, and are not able to determine what caused that damage, the changes set in motion from years of drug use, including antiparkinson’s drug use, may well be altering the accuracy of the autopsy tests. In other words, it may well be that the number of misdiagnoses is still just as high as ever, but the number is being hidden because of the use of antiparkinson’s and other mind-altering drugs.

We had found evidence of unhealed foot injury and backwards running electrical currents in *all* of those PDers who had unmistakable symptoms of classic Parkinson's disease. Looking at the numbers from another angle, we only found the foot problem and the electrical disarray in about seventy-five percent of the people who came to see us.

So this was very encouraging. The percent of misdiagnosed in the autopsy study was, as noted, approximately the same percent that we ended up with using our "foot/electrical disarray" exam. As the footnote below will explain in greater detail, this is only a sort of "negative proof," but still, it was encouraging.¹

The Parkinson's Personality

The earliest medical reference to the Parkinson's Personality that I have read of was published in the 1930s, when it was still safe to discuss personality patterns. Although it is somewhat risky, ever since the 1960s, to associate a personality pattern with an illness, the Parkinson's Personality is so recognizable that research in this field continues, despite the professional risks.²

¹ Our observation that people who evidently had been *misdiagnosed* also were found to *not* have the same foot and electrical symptoms as the correctly diagnosed PDers is called, in science, a "negative proof." A negative proof cannot be used to clinch a theory. However, this type of negative proof, though circumstantial, lent additional weight to the positive proofs. "All the PDers we examined *did* have electrical disarray" is an example of a positive statement, and possibly a positive proof.

² The reason that such research is not widely broadcast has to do with current social policies: doctors are not supposed to "blame the victim." Even if particular attitudinal stances have been shown to cause or contribute to a particular illness, it is not considered wise to mention this to the patient. Suggesting that a patient is in any way responsible for his own illness is almost the same as saying that the "victim" of the illness brought it upon himself. While, in many cases, this cause-effect relationship may be absolutely true, it is not politically correct or legally safe to say so.

For example, it is not medically "correct" to point out to a lung cancer patient that his decades of smoking may have contributed to his illness. It is more correct, at least in public, to blame the tobacco companies for putting temptation in the smoker's path.

As another example, doctors currently are not *allowed* to use the word "obese" with regard to their obese pediatric (under age 18) patients. To do so is currently considered "negative" and possibly harmful to the child's self-esteem. The child might feel bad if he is termed obese because, in our culture, we tend to assume that obese people have brought their problems upon themselves. The child, therefore, may feel that he is being accused of responsibility for his obesity. This would be "blaming the victim," and it is politically, socially, and legally unacceptable. (And while I'm on the subject, an MD was *successfully* sued by an obese adult after the doctor "injured the patient's feelings" by telling the adult that he was, technically, obese.)

Of course, an MD can use the word "cancer" with a pediatric or adult patient because our culture does not consider that cancer has anything to do with the cancer patient's behaviors. Therefore, the cancer patient is an "innocent victim" and not subject to blame.

The point here is that, even though the Parkinson's Personality is discussed and even researched by MDs, these same doctors might be putting themselves at risk of a lawsuit if they ever suggested to a specific PDer that he has, in any way – for example, by having a specific personality – brought this illness upon himself.

The recent article, "Personality traits and brain dopaminergic function in Parkinson's disease," published in the highly regarded, top-of-its field journal, *Proceedings of the National Academy of Sciences, USA 2001*: 98:13272-7, authored by Valtteri Kaasinen, MD, PhD, is proof that the personality of Parkinson's is still a valid subject for research, despite the reluctance of clinical MDs to dip into this can of legal worms.

One of the problems with the ongoing research is that, although alert doctors who work with PDers often have a strong sense that PDers have a unique way of interfacing with the world, it has always been hard to put one's finger on the exact nature of the Parkinson's Personality. Each PDer is unique, of course. And yet there *is* a difficult-to-define something that binds most of them together.

Our work has given us a rare perspective. As we worked closely, weekly, with people recovering from Parkinson's, we witnessed the personality changes that typically occurred during recovery. As PDers recovered and shed the guardedness, excess intellectuality and/or suppressed anxiety that is fairly typical of Parkinson's, new personalities emerged. These new personalities could, because of their experiences on both sides of the coin, describe somewhat objectively their old, pre-recovery personalities.

Very often, the recovering PDer realized, usually *for the first time*, that his highly competent, materially successful, or highly intelligent persona had not brought him a particularly peaceful, satisfied, or joyful life. As PDers recovered and became able to compare and contrast their pre- and post-Parkinson's prioritizing and values, we were able to more deeply to understand what constituted the so-called Parkinson's Personality.

What we couldn't figure out, and what the fully *recovered* PDers couldn't put a finger on, was how to point the way to the portal to joy or contentment for those partially recovered PDers who were still hiding behind life's sofa.

Historically, after the discovery of dopamine insufficiency in PDers in the late 1950s, the Parkinson's personality, like all other symptoms of Parkinson's, *was* automatically attributed to a dopamine shortage.

The experiments performed at the end of the 20th century by the research team led by Valtteri Kaasinen, MD, PhD (previous footnote) were done to determine whether or not dopamine loss actually is the key to the personality. The researchers expected that it was.

However, researchers found that the core personality/behaviors are unchanged under the influence of L-dopa; their guess had been wrong. The researchers were forced to conclude that the Parkinson's Personality is not caused by dopamine-insufficiency. Also, research suggests that the Parkinson's Personality can be evident decades before Parkinson's symptoms appear.

Our research suggested that a combination of selective dissociation and certain mental habits that are characteristic of the sympathetic mode were causing the "personality." The use of brain altering drugs such as L-dopa does not necessarily turn off adrenaline-based mental habits, nor does it restore heart feeling if the dissociation is still in place.

Because partially-recovered PDers could sometimes move normally, we could concur with Dr. Kaasinen's research: we could assume that the personality was not related to insufficient dopamine levels.

Clues to the Parkinson's Personality mystery

As noted already, some PDers have, at some early age, usually in childhood, experienced a life-threatening (to the child's mind) fear that could not be laid to rest. Others have decided consciously to pretend that they could not feel physical or emotional pain. Sometimes this charade was instituted to mask a childhood injury that might have aroused parental wrath.

Sometimes this mental stance was a life- or mind-saving childhood or war-time necessity. Sometimes, the attitude was instituted because of chronic mental or emotional stress, including

cultural or family-based attitudes that were hostile to or did not support sympathy for physical and/or emotional pain.

This attitude of denial of physical or emotional pain helped provide an explanation for the non-healing of the foot injury seen in all PDer's: the injury remained unhealed because, according to the PDer's mind, the injury didn't happen, or, at any rate, was not painful and did not need healing.

In some PDer's, it appears (based on recovery symptoms) that the feel-no-pain attitude was *initiated* to deal with the foot injury. Other PDer's come to realize, during recovery, that this attitude was already in place due to other stressors, and that they automatically applied the ongoing mental/emotional protection at the time of foot injury.

During the PDer's post-injury lifetime, the attitude may have remained associated only with the foot injury, or the mental trick may have been expanded to protect the body and feelings from pain during other life events as well. In some PDer's, the attitude may have snowballed in the brain's compartmentalization process until the PDer might have emotional guardedness over nearly every aspect of his life.¹ This guardedness, once set in motion, may have allowed the PDer to have lived his life using adrenaline as his primary motor neurotransmitter. The PDer may not have used dopamine for motor function for most of his life.²

This astonishing mental achievement requires an enormous amount of emotional and mental self-control. PDer's are notoriously highly intelligent. They are also notoriously unable to be flighty, irresponsible, or flamboyant with their deeper emotions.

¹ I wrote "nearly every aspect" of his life instead of "all aspects" because stories abound of people with advanced PD, people who can barely move, speak, or eat, who can move perfectly normally in highly specific, "safe" situations. I've already written about this phenomenon, but let me provide a citation. The case of the unmedicated PDer who is absolutely paralyzed except when an easel and paints are set up in front of him was written up by Oliver Sachs, MD, and published in the *World Parkinson Congress Journal: Creativity and Parkinson's*, 2006, p. 1. In this case, after several minutes of sitting in the presence of his art supplies, this man can stand up and, moving freely, proceed to paint. He can move almost normally until he stops painting, and then the paralysis resumes. The same sort of story is told of violinists who have to be helped onto the stage, but who, when the conductor raises his baton, can play as beautifully as ever. I knew one PDer who could always move perfectly normally on his birthday. I know another who moves perfectly normally and his persistent tremor completely stops when he is doing jigsaw puzzles. When he is doing these puzzles, he moves perfectly normally, no sign of movement inhibition or tremor.

Even in PDer's whose ever-increasing anxiety levels have caused the brain to incorporate more and more "compartments" under the aegis of emotional guardedness and dopamine-inhibiting fear, there *may* still be some brain arenas, such as painting, singing or, in the above example, birthdays, which have, in some PDer's, remained free from the growing emotional guardedness. Therefore, in these rare PDer's who have one or more "safe activities," or one or more brain compartments or mental arenas in which dopamine flow is still emotionally acceptable, their dopamine can still be freely released – but only during these highly specific activities.

These times of easy movement are very different phenomena from the well-known ability of unmedicated PDer's to move normally during times of emergency. The latter is due to a surge of adrenaline. The former examples are due to surges of dopamine.

² As you will read later, the symptoms of Parkinson's disease appear when the energy that comes from pure self-awareness or feeling, also known as the thrill of being, the dopamine-using heart-energy that releases either adrenaline *or* more dopamine, whichever is appropriate, begins to flag. Dopamine levels may have been at a very low, dormancy level for decades before PD symptoms appear. Only when the *emotional energy* required to maintain the PDer's powerful, and eventually, exhausting, mental wariness, one that requires using adrenaline as the neurotransmitter of choice for most thinking and movement processes, becomes, due to an increasingly shut down heart, insufficient, do the symptoms of low everything – low dopamine *and* low adrenaline – become visible.

We often wondered how this severe level of responsibility and self-control was related to the mind-body disassociations that we'd discovered while trying to get PDer's imagine, visualize, or feel their own bodies. What was the connection?

We didn't know. But starting in 2004, we no longer waited to see if the person would recover easily and quickly or slide into a condition of partial recovery. Instead, as soon as we started doing Yin Tui Na with a PDer, we also started right in on trying to get the PDer to have some sort of relationship with his feet. Simultaneous with the Yin Tui Na therapy, we worked on helping PDer's imagine inanimate light or energy in an injured area, in the "other" side of the body, or in areas that were succumbing to tremor or other PD symptoms. Some of these fairly traditional exercises and mind games were mildly successful in temporarily allowing the person to imagine a tenuous or flickering light in previously dark areas.¹

But then, after spending more than a year pondering PDer's difficulty in imagining light in injured areas and their disassociation with the injured side of the body and thinking that we were up against something pretty hot, we discovered a much more severe manifestation of the mind-body disassociation than we could have possibly imagined. It had been there right along, but we had never thought to ask PDer's about it.

An imaginary separation of physical body and energy

Not only was it hard for most of our PDer's to imagine an inanimate light in a specific body part; most of them, when asked to imagine their whole body filled with an animated image of their own body made out of light, did something completely unexpected. They imagined a body-of-light that was physically separated from their own body. Most attempts to integrate the imaginary body of light and the physical body were "repulsive," "disgusting," or "impossible."

For many PDer's, an entire body made of light, an imaginary light-body capable of moving as a body and being the driving force behind one's actual physical body, was *much* more of a threat than the mere inanimate light that we'd been asking them to imagine.

Many of our PDer's were extremely averse to imagining their physical body filled with animate light, even when we suggested that getting over this mind set might enable them to conquer their remaining PD-like symptoms. Much as they claimed to want to recover, they weren't willing and/or able to create an animate, light- or love-filled self-image.

After stumbling across this particular mindset, we spent most of the next two years trying to get to the root cause of this fear and aversion. We did not yet know that this phenomenon was called "depersonalization," and was a symptom of both selective and automatic dissociation.

2004 AND 2005

We discovered the aversion to a "whole body made of light" when working with a PDer who had been trying hard to imagine light or energy in his arm.

After this PDer assured me that he was finally able, after much struggle, to imagine light or energy in his arm, I decided to explore yet another angle of the situation; I asked him if he could imagine that this energetic arm made out of light was able to move at the same time that

¹ Rebecca Weinfeld, LAc, a PD Team member since 1999, has decades of experience as a psychiatric in-patient nurse. Since becoming an acupuncturist in 1999, she has continued her ongoing study of the new treatments for mental/emotional disorders. She's been a steady source of new ideas for the rest of the team, teaching the rest of us everything from muscle testing to "tapping" techniques.

his arm moved. I also asked him if he could imagine the arm-made-out-of-light floating up out of his body, having a life of its own, if you will.

Sadly, he was completely unable to imagine any sort of movement in the light image that he had so laboriously constructed. The light image that he had created, with sweat and tears, was only an inanimate beam of light sitting like a lump, albeit a bright lump, in his arm. He could not imagine this light moving or exhibiting any signs or symptoms of motion, or for that matter, being actually *connected* to his body.

This led us to explore whether or not our other Parkinson's patients who seemed to be stuck at some level of recovery were also unable to imagine *movement* in the body parts that they had, with great effort, manage to temporarily "light up."

It turned out, once we started asking about it, that other PDers who *had* learned to imagine light inside had also constructed only an inanimate sort of light that sat like a lump in the body, incapable of movement or any relationship with the body part. I already mentioned this phenomenon briefly in chapter twelve, in the section on depersonalization. In this chapter I will go into a few more details. Please forgive a few redundancies.

The inquiry that revealed an imaginary separation of physical body and energy went like this: "Imagine your whole body filled with light. Don't worry about any specific areas that are dark, just imagine as much of your body being filled with light as you can. If possible, this light in your body should have arms and legs and fingers and toes just like yours. Make it a really wonderful light, a light that is beautiful, radiant, full of joy. Make it a light that you can love, one that feels safe and wonderful. Can you do that?" I then gave them a few moments to let them enjoy that beautiful image. Then I asked them *where* that body of light was located.

Where is your body?

I have tested this exact same question on healthy people, people who've never had Parkinson's disease. A healthy person will typically answer my question as to the location of his body-filled-with-light in this way: "Huh? What do you mean? The body of light is inside me. Isn't that what you asked me to imagine?"

Note that my instructions had been very carefully worded: "Imagine your whole body filled with light," and then the question, "Where is this body of light located?"

A body floating in space

The vast majority of my PDers answered my question "Where is it?" with the following type of reply: "It's in the mountains," or "It's floating a few inches above my prone physical body," or "It's at the beach," or "It's standing behind me," or "It's ten years old, and it's skipping over the rocks at my boyhood vacation home," or "Part of it is in my body, but one leg (or arm or some combination of limbs) is sticking out to the side, away from my physical self."

Again, I am careful to phrase my question so that the patient should assume that I am asking him to imagine his physical body being filled up with light *in situ*. I do not ask where the person imagines that a lighted-up version of himself might be *easiest* to imagine. I will share some more examples.

It's safe: it's locked up

Again, here is the question sequence: "Imagine your body being full of radiant, joy-filled light. (Long pause while the person does this.) Where is that body full of light located?"

I think the most alarming response I ever got in response to my queries was this sequence that started out with a reply of: “Oh, it’s OK.”

I replied, “What do you mean, ‘it’s OK’? Where is it? Is it inside your body?”

She answered, “Oh no. It’s locked up. Like in a cage. It’s safe.”

I was curious. “Your body made of light is locked up. Can it get out of the cage if it wants to?”

“Oh, no. There’s a guard.”

“Is the guard a friend or an enemy?”

“He’s a friend. He has a long grey beard, and he’s protecting me.”

The patient was lying on the treatment table with closed eyes. I exchanged a glance with her husband. He goggled at me with concern. I goggled back and shrugged my shoulders. Neither of us had had any inkling that this construct was in her mind, or why. But it certainly explained why she couldn’t connect her physical body and her mental image of herself made out of light.

As an aside, despite the strong insistence from a few PDerers that this inability to visualize the body is a sign of rare sensitivity of soul and advanced spiritual detachment, we have learned via the healing process that this detachment is not spiritually based: it is based on fear.

Getting back to the main problem, we started to refer to this new situation as an inability to integrate the physical body and a mental image of the energetic body. This was even more bizarre than the first mind-body disassociation that we’d first discovered, which was the mere inability to imagine inanimate light in a specific body part. Also, there was a much higher level of emotional resistance to integrating the body-of-light with the real body than there had been to merely imagining a spot of inert light in some body part. The following case studies demonstrate.

Some examples

Mort

For the past few months, Mort had been working on visualizing light in his foot. I was, at this point, more concerned about Mort’s sometimes shuffling walk than about his useless left arm. His arm had been unusable for a long time.

Mort was the second person on whom I sprung the procedure of filling the body with light and then locating that body-of-light. When I asked him to imagine his body full of light, he told me that he could, and that the body full of light was floating nearby. I asked Mort to try to juxtapose the floating image with his physical body.

When he started connecting his mental image of his body-made-of-light and his physical body, Mort was able to imagine that his head-of-light was integrated with his physical head. So far, so good. He was able to get his mental image of his light-neck and his physical neck aligned. He then started moving his light image into position in his physical arms. As the mental image of arms-made-of-light started to flow into his left arm, he jerked violently and exclaimed, “That’s disgusting!”

I asked him what was disgusting. He was surprised. He had not realized that he had said anything out loud. When I asked him again what was disgusting, he replied, “Having light in my

left arm.” He had started trembling violently from the experience. I asked him what was disgusting about having light in his left arm. He answered, “It just is. It’s disgusting. There is no other word for it. Don’t you know what disgusting means? I won’t do it again.”

What is interesting is that Mort had slowly gotten to the point where he was able to imagine an *inanimate* sort of light in his left arm. However, when he tried to put a vitalized light-filled version of himself into the left arm, the experience was so foul that he refused to try it again.¹

Haime

Next, I tried the procedure with Haime. Haime’s mental image of his body-of-light was hovering nearby, about a foot away from Haime. When I asked Haime to imagine his light-body reinserting itself into his physical body, he was able to get the heads to match up. But when he tried to join the neck-of-light and his physical neck, he said, “I can’t do this. I’m afraid I’ll become too big.” At this point, he started crying.

When I asked why being too big might be a problem, he answered through his tears, “I will be proud. I don’t want to be proud and arrogant. I’m afraid to connect the necks.” Haime stopped coming in shortly after this.

¹ Over the next year, Mort became nearly incapable of movement, even though he had been diagnosed only two and half years earlier. He knew about the therapeutic mental exercises that we hoped might be helpful, and would do them with me when he is in my office. At these times, he could move somewhat normally. However, when he returned home, he would find that he didn’t have the time or interest to actually continue these mind-retraining exercises. Considering we were trying to teach him a completely different way to see himself and the world, an hour of therapy once every few months was obviously not enough. He needed to learn to live, permanently, with his left arm. However, he was not interested in trying to actually have any energy in his arms or in his body, though he desperately wanted to not have Parkinson’s.

His defense for his inability to want to change was that his parents were not very emotionally sensitive. This type of defense, in which the PDer blames some difficulty in his past for his reluctance to attempt change, is not uncommon in those PDers who recover part way and then suddenly become much worse in certain arenas. But even without the “poor me” defense, many PDers simply did not fancy the idea of changing themselves.

While this attitude may make no sense to the idealistic reader, I can assure you that every doctor knows what I am talking about. It is an old truism that most people would rather die, literally die, than change their mental, physical, and emotional bad habits. How many people continue to smoke cigarettes even while hoping they will not get lung cancer? How many people continue to overeat even though they know that they are injuring their digestive tract, their heart, and their vitality? How many people continue to give their emotions free rein even though research has shown that people who are quick to anger have a higher incidence of heart disease?

The same human tendency for stubbornness exists in many people with Parkinson’s disease. Some PDers, while asserting that they want to recover from Parkinson’s, did not want to even try to change the fear- and adrenaline-based behaviors and attitudes that sustain the illness. As one adamant PDer put it, “I don’t want to have Parkinson’s but I want to still be ME. I don’t want to turn into a sappy sort of person!” Like another PDer who tearfully asked, “But if I get rid of my fears, what will be left of me?” some PDers, ultimately, would rather have Parkinson’s than change.

What we needed to find, then, was a way of helping PDers change that would not seem threatening or seem like work. We needed to find a method of self-change that was so mentally or emotionally rewarding, so much fun, so joy-producing, that PDers would *want* to do it, despite the fact that such a change might alter their idea of who they were, their personality. This was a big order – especially in light of the fact that so many PDers consider fun to be self-indulgent, and therefore bad, and many PDers are almost incapable of remembering what joy had ever felt like.

Melica

Melica's light-body was only partly out of her body. In fact, only her left leg was outside of the physical frame. Her mental image of her light-legs had her left leg crossed over her right leg, at the level of the right knee. In point of physical fact, both her legs were stretched straight out on my treatment table. When I asked her to try to put her mental vision of her left leg back into her actual leg, she balked. "I know this will sound crazy," she said, "but if I put the light-image of my leg into my leg, I'm going to get angry. I can't stand the idea of losing my temper. I'm not going to put that leg back inside."

Looking for bodies made of light

After this, I started asking every one of my Parkinson's patients to imagine his body filled with light. When they told me that they could do this, I asked them where the images were. As soon as the PDer told me that he could picture the image of himself made of beautiful light, I asked him where it was and then asked him to reinsert the image into his body. Consistently, the mental self-images were partly in and partly out of the physical bodies, or floating around in far off lands or even a different age from the patient's actual age. As for reinserting the errant body or body parts into the physical body, most PDers had enormous resistance to the idea.

Some of them cried, some just shrugged it off and said they didn't want to do it. Others were afraid that if the light-body parts got into their corresponding physical-body parts, they would discover cancer or some horrible thing in that body part. Some of them kept changing the subject repeatedly, suddenly saying things like: "Before I forget, I want to ask you if you enjoyed your weekend." One person was able to do it easily only so long as I was holding his feet.

These people were not psychotic. They were able to acknowledge that their imaginings made little or no sense, but they were unable to anything about it. They were reporting to me as accurately as possible their perceptions about their bodies and their body-based imaginations.

Unaware of being unaware

Some people, when asked to imagine their bodies filled with light, assured me that they were imagining light filling their physical forms. I soon learned to be very suspicious of these assurances. I soon began adding another query. When a PDer told me that he was imagining light throughout his body, and his whole body, in his imagination, was lit up with perfect, uniform light, I would ask him to specifically look at his problem limb. Whatever limb that person seemed to have the most trouble with, I would ask him to check carefully and see if that limb was part of the "whole body full of light."

Very often, a person with, say, extreme left arm rigidity, might tell me that he was imagining his whole body filled with light, in situ. I would then ask him to specifically look at his left arm. There would usually be a pause, and then the person would realize that, in fact, his left arm-made-of-light was sticking up straight in the air, even though, of course, his physical arm was nestled, rigid, alongside of his body.

Many of my patients were surprised to find that a recalcitrant body part was not where they thought it should be. Most often, when they assured me that everything was accounted for, that the whole body was lit up from within, I had to draw their attention to the body part that usually dragged, tremored, or was rigid. Lo and behold, *that* physical part of the body, when they really looked carefully, was *not* conjoined with the lighted-up body image; that part of the body, in the imagined body-of-light version, was missing or sticking off in a different direction from that specific part of the physical body.

This was similar to the situation with inanimate light in which a PDer might assure me that he could imagine light all through his foot. When I placed my hand directly over the portion of the foot that felt the most damaged, and asked him if he could imagine light directly beneath my hand, he would reply, more hesitantly, something along the lines of: “Well, there’s no light exactly right *there*. But I *can* see light in all the parts of my foot where I can see light...”

Good news

At this point, the reader might be shaking his head and saying: “Are all PDers nut cases? Is there any hope for me if I have PD? Am I that deluded?”

To assuage these fears, I want to share my experience with Lucinda.

Lucinda

Lucinda had completely recovered from her foot dragging, her lack of facial expression, her hunched posture, and even her adrenaline-charged attitude. Her only remaining problem was her increasingly problematic tremor. When I had first started working with her, her left hand only tremored once a month or so. Now, three years later, I only saw her once in a great while, for issues other than Parkinson’s. She had stopped coming in regularly when her other symptoms went away. She hadn’t been overly concerned about her mild, intermittent tremor, and had assumed, as had I (in the early stages of this project), that the tremor would go away by itself once the foot problem was resolved.

She came in to see me because of a chest cold. While I was working with her, I asked about her old symptoms. None of them had reappeared. But when I asked her how her tremor situation was doing, she said that it was becoming more frequent, and was even starting to be a bit of a problem: whenever she was under stress, or when she was conducting a choral group, her left hand tremored noticeably. By good chance, I had just stumbled upon the body-of-light reluctance problem that many PDers had.

I asked her to imagine her whole body filled with light. She closed her eyes, and after a few seconds told me that she was all lighted up. I asked her if her light-body was inside her physical body or floating around somewhere. She replied that it was connected to her physical body. So then I asked her to look carefully at the light-version of her left arm, and tell me if it was in sync with her physical left arm. Her actual arms were flat on the treatment table alongside her body.

She started laughing out loud. “Well, whaddya know!” she said. “My image of my left arm is folded across my chest! (More laughter.) No wonder I can’t get any control over it. That arm’s not going to do me any good *there*. I’ve got to get that naughty arm image back where it belongs!”

I was so grateful to Lucinda. So many of my PDers got sullen or resentful when they realize that their mental self-image and their physical self were not connected. This wonderful woman was amused! And, for what it’s worth, she was also a musician, and, I happened to know, successfully working at letting go of resentments and blame. She told me that she was learning to trust that the universe was always taking care of her, in spite of apparent setbacks. She told me, “Every time something hasn’t gone the way I’ve expected or hoped, I think of it as a gateway moment.”

I taught Lucinda a simple visualization therapy that a few PDers had used, successfully, in their attempts to override their mind-body disassociations. Lucinda mastered it within minutes. By doing the exercise, she immediately and permanently regained complete control of her arm.

The tremor ceased. It never came back. While she was doing the exercise, she recalled that she had broken that arm when she was four years old.

A few hours later, she met some friends downtown. When one of the women gently disagreed with Lucinda's idea about something, and sided with the idea of another woman in the group. Lucinda astonished the group by declaring, "If you agree with her, you can't be my friend!" The stunned friends were silent for a moment, and then quickly took their leave of Lucinda. Lucinda told me later that, even at the time, she was wondering to herself, "Where did *that* come from?" Lucinda was immediately consumed with the thought that what she really needed was a pink tutu. Since she was downtown, she walked over to the ballet shoppe and looked at the tulle skirts. She knew, on some level, that she didn't really want to buy herself a pink tutu. She was not a dancer. She had a "conference" with herself in which she negotiated a deal: the tutu desire could be traded for the desire for a banana split. She went to the ice cream parlor, ate a banana split, and never tremored again. And from then on, when she imagined her arm being full of light, the imaginary arm was right where it should be.

I will be describing the visualization therapies in the chapters on treatment techniques. I only shared this particular case study at this point in the book, though this chapter is getting a bit long, because I can imagine the dismay that some readers might be feeling at this point. I wanted to show that these mental blocks were, in some cases, easily dispelled. On the other hand, the reader should know that many PDers struggled against these imagination-based therapeutic exercises as if their very lives were on the line. And yet, strangely enough, these PDers could usually imagine with ease a negative outcome or disaster.

A common pattern

Going back to the chronology, meanwhile, the other members of the PD team were also doing the same body-made-of-light exercise with their PD patients, in order to see if their patients also had a "detached" light-body. After one week of asking all our PD patients about their body-of-light, we all knew we were up against something that we had not anticipated.

The PDers who were not progressing rapidly in their recovery or who had moved into the ranks of the partially recovered had evidently managed to disassociate their physical form from their own body-image. No wonder they had increasing rigidity, slowness of movement, poor balance, and trembling from anxiety. They had lost the connection between the idea of energy and the idea of the physical reality of their body.

No wonder they were failing to recover normal movement despite the return of normal energy flow in the legs! It was as if they had a body that was returning to correct physiological function, but a mind that was determined that the real part of the body, the energy-filled part, the beautiful part, the good part, was not connected to the physical form.

VARIOUS LEVELS OF MIND-BODY DISASSOCIATION

Not all PDers have the same degree of disassociation. I know I'm being highly repetitious here, but during recovery from the foot injury, *some* PDers automatically resume a relationship with the injured foot and the long-missing injured side of the body. Some even remember when they decided to pretend that they could not feel pain. However, in most of the partially recovered PDers we've worked with, some level of mind-body disassociation has remained in place even after the feet recover.

Mild disassociation

Sometimes, the disassociation has been fairly mild, merely preventing the PDer from being able to cry or experience joy. Even a mild level of disassociation can make it difficult for the PDer to perform any type of mental work that involves visualizing, imagining, or pretending – *if* the visualization or imagery is directed towards a “good” mental image or a positive outcome. Many a PDer with only a mild mind-body disassociation is not able to mentally picture his own body being in a state of health, or mentally imagine, pretend, create, or visualize any sort of mental picture of himself moving in a healthy manner.

A more bizarre level of disassociation: imaginary functional bodies or alter-egos

Others have far more complex disassociations. We discovered that some PDers had formed complete mental disassociations from their physical bodies, to the point of having an active alter-ego outside their physical bodies. These alter-egos had usually been created during childhood or early adulthood. These alter egos were imagined as being physically separate from their physical bodies.

Some PDers were always aware of their imaginary mirror-image (left and right sides reversed) or correct image versions of themselves, standing silently by, just a few feet away. When some PDers were asked to “fill your body with light,” they tuned in to the ever-present alter-ego, standing nearby.

Before continuing, I will give two extremely quick examples of what I mean by “mirror image,” alter-ego personalities.

Honorina created her mirror friend on the day of her high school graduation. She was an American, attending school in Germany because her missionary parents were in China. Her mother showed up in Germany for the graduation. When Honorina asked where Father was, Mother calmly replied, “He died six months ago. We didn’t tell you because you would have wanted to come to China for the funeral, and that was out of the question. When Honorina felt her chin start to quiver, her mother said, “I do hope you are not going to make a scene about this.”

Honorina excused herself to go upstairs and get her hat. She remembers going upstairs to her room and staring at her face in the mirror. She commanded the person in the mirror, “You can do this! Don’t make a scene.” And for the rest of her life, the person in the mirror went through life with a smile on its face.

Honorina had been a vibrant, champion tennis player and lover of opera, a mother of three and beloved by all. Honorina died in her late 80s. At Honorina’s funeral, her daughter told me that she had never seen her mother express any emotion other than a proper level of contentment and or happiness. “I have never seen my mother upset about anything.”

Honorina had never told anyone about her mirror image persona until, just a year before her passing, we asked about her emotional history.

Sharing my amazement

When I first heard about Honorina’s relationship with her “person in the mirror,” I was so amazed that I mentioned the mirror idea to a few of my patients. One of them, Hope, said (I paraphrase very closely), “Oh. Yeah. I do that.” My jaw dropped. Hope continued, “My mirror image does all the hard work. When I feel like I might lose control, I just look in the mirror and say to her, “Be tough! Don’t be a wimp!” She [the person in the mirror] can deal with anything.

She never gets her feelings hurt. She's my mirror image, but I can only see her down to the waist. Maybe that's because I've never lived in a house with full-length mirrors.¹

The alter-egos

Those are just two examples of the “functional alter ego” phenomenon that we've found in PDer's. While this alter-ego phenomenon has not been present in a majority of our PD patients, we no longer consider it unusual when an imaginary friend pops up. One of our PD patients even had full-blown multiple personality disorder. Interestingly, all of his personalities had Parkinson's. As he started to recover from Parkinson's, a “master personality” emerged which could integrate the other personalities.

Learning about the existence of these alter-egos was helpful. It helped ready our minds for the discovery, still to come, that many people with PD have consciously dissociated from their hearts – and therefore, their bodies.

There are no hard and fast rules about the roles of the imaginary alter egos. Sometimes, an “other” self or an “aspect” of a PDer's personality is considered to be performing all social interactions and physical events so that the “real” person never needs to risk being exposed to emotions or pain.

Some PDer have reversed the roles just described: the real self – the one with feelings, the one that needs to be protected – is imagined as floating in the ether a few inches, a few feet, or a thousand miles away, while the numb physical body – a body that, via pretending, can't be hurt by physical or emotional wounds – goes through the motions of life.

Some PDer's allow their real selves to do good-hearted, sometimes philanthropic work, while their alter-egos or mirror images dealt with any physical event or social interaction, real or anticipated, during which physical or emotional pain or any sort of negative outcome might occur.

These alter-egos and mirror image personalities did *not* automatically go away when the foot injury healed. Getting rid of these not uncommon mental constructs turned out to be doable, but it required focused work on the part of the PDer and a willingness to drop the ruse: to be once again susceptible to pain and/or pleasure.

Even though we were discovering the unexpectedly wide range of PDer's' mind-body disassociations, we still had, as yet, no consistent method to help PDer's overcome these fear-based mental/emotional constructs. We experimented for several years with a wide sampling of traditional and modern self-analysis and self-love healing techniques, to no avail.

¹ I heard a powerful story of real friendship from Hope after she decided to get rid of the Mirror Woman. Hope had confessed to her good friend at work about her habit of using the mirror personality instead of her physical self when she needed to be tough, and how, working with me, she was learning to not rely on the Mirror Woman any more.

But one day at lunch break Hope had gone to the muffler shop for a car part. She had tripped on an electrical cord that stretched across the grimy floor of the muffler shop. When she stood up, her impeccable white shirt was smeared with dark grease, as were her hands and knees. Hope started trembling violently and “feeling like everyone must think I'm a moron.” She rushed back to work, and hurried past her friend to get to the washroom so that she could look in the mirror and get a grip on herself.

Her friend jumped up as the grease-covered Hope went scooting past, and grabbed Hope by the arm. “You don't do that any more, remember?” Stunned by this interruption in her lifelong routine, Hope was able to calm down and tell *herself* the spine-stiffening words that she would have said to the Mirror Woman. This meant, of course, that the accident had happened to Hope, and not to the Mirror Woman, but with her friend's help, Hope was able to accept this, although it was embarrassing, and therefore emotionally painful.

Rewriting the Recovery Handbook

In 2005, I started a revised edition of this book, mainly to get rid of the outdated references to Parkinson's medications that had been in the previous edition. I also wrote up a few chapters for the website about our discovery of what we were calling a "mind-body disassociation" and its apparent relationship to fear. However, the mind-body reconnecting techniques that I offered up in those chapters were techniques that most PDers struggled with, disliked, or even hated. And, except in a few cases, such as Lucinda's, the benefits of practicing the techniques seemed to be short term.

Still, we posted the information to let people in the PD community know that we were still plugging away at the problem, that we hadn't given up on those PDers who were partially recovered.

This 2005 edition was never finished. I was slowly plodding away at it, updating some of the information and posting the revised chapters on the website. But even as I was writing, I was wondering if we were ever going to find a way to get partially recovered PDers to join the ranks of the completely recovered. I was pretty sure that the Parkinson's personality, the inhibition of positive-outcome imagination, the inability to imagine one's body being full of light, and even the alter egos and multiple personalities were all related, somehow, to the problem of partial recovery. But despite all our research and treatment experiments, we had run out of ideas.

In January, 2006, I went on a silent retreat for nine days of prayer and meditation. My heart pleaded for answers to my questions. Or, if no answers were to be forthcoming, I prayed that my patients who were stuck in partial recovery might recover, even if I never understood the process behind their recovery. I started going through the list of patients for whom I regularly implored healing. I started with Hope.

I received an answer!

"She has to do it herself."

I was simultaneously thrilled and disappointed. I prayed more insistently that God please heal Hope. The answer came quickly this time, resonating in my heart. The voice repeated, "She has to do it herself."

So I demanded that He tell me what it was that she needed to "do." My heart was silent.

2006

Two weeks later, we had a breakthrough. It happened on Feb 10, 2006: exactly eight years to the day from the time I first got up to speak at the local Parkinson's Support Group to say "I've noticed some similarities in the feet of people with Parkinson's. I'll give several free treatments to any person with Parkinson's who lets me examine his feet..."



“Weep my child, for he who is without tears has a grief that never ends.”

- Mexican proverb

CHAPTER THIRTY-ONE

THE CASE OF THE MISSING HEART

We did not discover a *solution* in 2006. But in 2006, the direction of the visualization techniques changed in a way that led us, eventually, to the heart dissociation. I am including all the visualization techniques that eventually led us to the heart because these techniques are things that PDerers may want to try if they decide to get their hearts open. I do not include the techniques in this chapter in the section on Treatment Techniques. That would be too redundant, even for me. But they still might prove fun or helpful for a PDer. They might also demonstrate the inventive way a person can play around with visualizations while working on feeling safe and getting his heart back.

Gilbert’s neck

On February 10, 2006, I was working with a PDer who was doing very well; most of his symptoms were greatly reduced and he felt increasing vigor. He still had a faint tremor in his right hand but he wasn’t concerned about it. He was more concerned about stiffness in his neck: I was planning to treat his neck with FSR.

Before starting on his neck, I asked him if he could close his eyes and try to visualize light in his neck. He could, and he could also see a spot of darkness in the back of the neck, near the skull. I asked him what the spot looked like. He replied that it looked dark and squashed. I told him to imagine it growing darker and more squashed.

The “Exaggerate the Problem” technique

This technique, in which we ask PDerers to visualize any perceived problem as being exaggerated and far worse than it actually is, developed out of our discovery that PDerers, for the most part, have a very hard time imaging sweetness and goodness, but they can easily and happily imagine a bad situation growing worse. Sometimes, when positive visualization proved impossible, we asked PDerers to pretend to chop off offending body parts in order to allow the mind to venture near to those areas, prior to initiating healing in those areas.¹

¹ I know that the idea of perpetrating mental havoc on an injured or frightened body part flies in the face of all the peace-love-sweetness techniques of modern psychology. But we had found that nearly all PDerers can easily, cheerfully, gain access to forbidden body parts by pretending to mutilate them. So we often started PDerers down the road to imagination by letting them create horrors on themselves. Spouses and practitioners were appalled, but the PDerers got a great kick out of it. Often, after pretending to mutilate or create a worst-case scenario such as cancer or gangrene in a formerly “off-limits” body part, then chopping off the “bad” body part and replacing it with a new imaginary body part from my office cupboard of “imaginary new body parts,” the PDer could actually feel that “missing” body part for the first time in years. The results didn’t last, but many PDerers could walk more easily, for several hours, after pretending to chop off an offending foot.

Knowing about this trick of negative thinking, this “back door for mental access” to mentally forbidden areas, was very helpful, ultimately. Later on, we took advantage of this negative access route to help PDerers make the first step in turning their hearts back on.

We recognized the need for PDers to focus on their fears and apply some mental attention to ignored areas, thus allowing their minds to recognize problem areas and start healing them. It turned out that, in the preliminary skirmish with this type of mental-disassociation problem, it didn't really matter whether one imagined the problem getting worse or getting better. The great thing was, in the beginning, to be able to turn the previously reluctant, even dissociated, mental focus onto the problem area.

This technique does not cultivate a negative attitude, although it may seem like it at first glance. It would be more accurate to say that this technique forces a person to face his fears. By firmly facing his fears, even exaggerating them, a person is often able to soon recognize them for what they really are: small events that have been blown out of all proportion. The power of the fears is thus diminished.

Getting back to Gilbert, by using this technique he was able to look at the dark spot in the back of his neck and imagine it getting bigger, darker, and more bruised and hideous. After about thirty seconds, when he grew bored with this, Gil stopped imagining and allowed the image to go back to its original condition.¹ As is common with this technique, the dark spot became very small and much less dark, and then disappeared.

Please note: even with emotionally healthy people, sometimes several repetitions are required to completely erase or remove the imagined problem from the area. And, as we learned later, if a person is maintaining a dissociative stance towards some body area – a contingency that we didn't know about yet – the dark area will soon return or it will just show up somewhere else. Still, because this technique was helpful for some people, and was better than nothing, we were using it on nearly all the PDers at this time.

I was curious to see if Gilbert was imagining that the injury to his neck had twisted something in the brain stem. I asked Gilbert to imagine that he was looking at his brain. (I have since found that even those PDers who cannot visualize their body are able, in nearly *all* cases, to easily visualize their own brains: they like their brains.)

After he had taken a gander at his brain hemispheres, I asked him to imagine what a perfect brain should look like, so that he could discern in what way(s) he imagined that his own brain differed from "perfect."

Bear in mind that Gilbert is not a biologist and has never studied what a brain should look like. This means that what I was asking him was, based on his own concept of what a healthy brain might look like, how did his *idea* of his own brain differ from his own *idea* of a perfect brain.

Gilbert could see that the left side looked smaller than the other. I asked him to make the "too small" side even smaller and to keep it small for as long as the experience was interesting. For about half a minute he imagined it smaller, almost to the point of disappearance. Then, he relaxed his focus and, to his mind's eye, the brain sides were suddenly symmetrical.

Next, he said that the left side seemed to be setting at an angle instead of facing forward. I asked him to torque the angle even more so. He did this. When he was done, the left side seemed to have aligned itself to its correct position.

The reader needs to bear in mind that his brain side probably did *not* actually realign instantaneously – if, in fact, there was any misalignment. Instead, the significance of this process

¹ Losing interest in the exercise usually signifies that the problem has become, to the imagination, less problematic.

is that Gilbert was sending a signal to his brain saying that, if there was anything skewed – physically or *perceptually* (in his mind’s eye) – it was OK with Gilbert if the body started healing that *problem of self-perception*. Gilbert, by finally paying attention to an area that didn’t, to his own mind, seem quite right, was giving conscious permission for his mind to pay attention to that area – and make his own self-image of that area become healthy or correct.

Where the mental focus is, there the life force can flow – assuming that the heart is turned on and that person is willing to be capable of feeling. The body’s innate healing ability can only work if there is energy, feeling, and self-awareness in the damaged area and the mind has access to the injured zone.¹

This technique – mentally looking around inside the body at various body parts and comparing the findings with what “perfect” body parts might look like – allowed Gilbert to “see” places in his brain that he *imagined* weren’t quite right: places that he had, at some point, actively blocked off from his own awareness or imagined to be “wrong” somehow.

By focusing on these areas, Gilbert was unblocking them or “dis-imagining” the barrier to the problem. If these areas actually *were* injured in any way, they weren’t necessarily instantaneously healed by this game, but they might be once again accessible to the body’s normal healing process.

Up until now, the techniques that Gilbert was doing, under my direction, were pretty much normal for what we’d been having all the PDers do, lately.

Ever since discovering the extreme level of mind-body disconnect in most PDers, I had grown accustomed to doing this visualization technique (imagining the problem getting worse or chopping off the problem area) as a sometimes, but not always, effective method for initiating healing in blocked areas.

But at this point, I suddenly felt inspired to ask Gilbert to do something new, something I had never thought of before. I asked Gilbert to look around in his head and find the place where there was an excessive amount of electrical activity. (Western medical findings show that a part of the brain, a part possibly associated with the tremor of Parkinson’s, seems to have an excessive level of electrical activity.)

Gilbert quickly imagined that he was looking at the place where this was going on. I wasn’t really sure what he should do next, so I suggested that he make the electrical firings worse. He did this for half a minute or so, and then said he was finished. I asked him if the area had changed. He said that the area was still firing off too much, but that it was somewhat calmer.

I cannot explain why, but I then asked Gilbert to look around the perimeter of the excess-activity area. I wanted him to see if all the connections running into and out of the area were correct.

¹ As a fun footnote about the body being able to block off healing, maybe it is worth noting that Vioxx, an arthritis-pain masking drug, was found to prevent the healing of broken bones. I saw this in my own medical practice: a patient who took Vioxx for arthritis pain fell and broke his hip. His doctor could not understand why the hip replacement never “took.” New bone failed to form following the surgery; he was never able to walk on the new hip. A year later, in winter of 2002, I read in a science journal that Vioxx recently had been shown to prevent formation of new bone. I shared this information with my patient, who brushed it aside, saying that if Vioxx caused problems with bone growth, the doctor would have known about it.

This may relate to our subject: *when the body is unable to know that pain or injury is present, the processes that initiate healing may be unable to work properly.*

As the reader probably knows, Vioxx is now off the market. Turns out, it increases the risk of heart failure. And no, most doctors didn’t know about that fact until it made the national headlines.

Again, neither Gilbert nor I had any idea what “correct” should look like, but Gilbert was able to examine this area for “correctness.” He said that all the connections running into and out of the area were healthy and correct, but that there was one place where the connection was missing. Possibly because he was a computer technician, he said that one socket leading into the area was sitting empty. It looked as if one of the “plugs” that should run into the area was missing.

I asked him to look around inside his brain and see if he could find the missing plug. He found it and hooked it up.

Getting plugged in

I happened to be standing near his head while he was doing this. From where I stood, I was able to see that his faint hand tremor had suddenly come to a complete stop. Not only had it stopped, the hand had relaxed deeply. The hand looked different than it did when the tremor merely “stopped for a while.” The hand looked so different. Radiant. For that matter, Gilbert looked different.

I asked him to unplug the connector that he had just plugged in. His hand resumed its very faint tremor and I realized that Gilbert’s face, eyes closed, looked faintly more tense or concerned. Also, his shoulders seemed to draw infinitesimally closer together. His *chest* seemed tighter. I asked him to plug the thing back in. His tremor stopped and he visibly relaxed again.

I asked him how it had felt in his chest when he plugged the thing in. He said that he felt more peaceful and his mind was less anxious. I asked him if he had noticed the stopping of his tremor and he said that he had. He also said that with the plug in place, he had no *internal* tremor. Instead, he felt *consciously* healthy, although, prior to doing this experiment with the plug, he had *not* felt unhealthy.

I asked him to try to keep himself “plugged in” until our next session, and he left.

Another PDer connects with the heart

I was so astonished at the utter change that had temporarily come over Gilbert when he imagined himself reconnected to some random, imaginary plug, that I tried the same experiment on the next PDer I saw that day.

Aggie

Aggie was doing very well. She’d visited our clinic twice before, at six month intervals. Many of her symptoms were gone but she still felt stiff and “unnatural” in her right arm and leg.

She had recently discovered that she could move easily if she skipped. She said that skipping made her feel happier, and that it was just impossible to move stiffly when she was in skipping mode. She was experimenting with mental attitude adjustments to get her mind into this happier state more often, but she felt that there was still some underlying problem that was keeping some body parts stiff.

I repeated the same technique with Aggie that I had done with Gilbert. I started by asking her to pretend that she was looking for anything wrong in her neck, and then we moved on to the brain itself. She imagined that she saw a bruised area in one part of her brain and a flattened area in another part. She increased the bruising and increased the flatness, respectively, in those two areas. Then I asked her to look for the area in her brain with increased electrical activity. It did not take her long to find it. (These games of pretending have no claim to anatomical correctness. Aggie’s electrical-excess area was in a completely different location than Gilbert’s and it looked different, but it was unmistakably, to Aggie, “the area with too much electrical activity.”

We later found that it didn't matter what part of the brain we asked the PDer to look for. It might be the brain area in charge of shoes, or the brain area that "doesn't want to be looked at." Although, at first, I asked people to look for areas with excess electrical activity, we soon learned that this location was not significant. I could get the same results by making hook ups with the imaginary brain area that knows about driving a car or "all your memories from age three." The benefit of the exercise turns out to be hooking the brain up to the heart, not finding a specific problem area in the brain. Now I merely define a brain area to help with the process of imagining some area in the brain that might be disconnected from the heart. Considering that this is a pretty large field for most PDers, it doesn't really matter what area we use.

I asked Aggie to increase the level of electrical activity for as long as seemed interesting. As had happened with Gilbert, when she finished this process, the electrical activity had decreased, but in her imagination it was still more active than her imagination thought that it should be.

I asked her to examine all the connections flowing into and out of the electrically over-active area. She did this and found that a few connections weren't quite right. She mentally imagined them as being worse, and this quickly allowed them to be healthy.

I wasn't sure how to ask the next question. With Gilbert, he had been the one to mention that a socket was empty. Aggie hadn't said anything about a missing connector and I wasn't sure I wanted to put ideas into her head, so to speak. Nevertheless, when she didn't volunteer anything, I asked her if there might be any other connectors lying around loose that possibly were supposed to be connected but weren't. She looked around for a moment and then surprised me by saying that she'd found one.

She hooked it up.

I saw her body relax just a bit. I asked her to unhook that last one for a moment and then hook it back up again. I could see her, almost imperceptibly, tighten up when she unhooked it and loosen up when she reconnected it. When she reconnected it the second time, she also placed her right hand over her heart and sighed. I had her disconnect and reconnect several times. Each time she connected it back up, although I had not yet suspected the heart connection, she would press her hand down onto her left chest and her whole body seemed to relax into the pressure of her hand. She was obviously feeling something going on in her heart area.

I asked her how it felt to connect that plug. She said that her mind felt calm and her heart felt opened up. She felt very good with the connector in place. I told her that I thought it might be the heart that she had just reconnected to her brain, and she said that was exactly what it felt like.

This was the first time that the idea of a physiological heart component had occurred to me. Although many recovered PDers had said that they'd experienced a "change of heart," or an "opening up in the chest," this moment with Aggie was the first time I suspected that some physiological event in the *heart* might be involved in cases of partial recovery. At this time, I was unaware of the research showing a heart nerve dormancy in people with Parkinson's disease.

We did some experiments while she kept her hand over her heart and focused on having her "heart plugged in." The first experiment was with speech. She was starting to tell me something about how she used to focus her eyes differently, and I asked her to start over again, and to tell me as if she were speaking from the heart. Her voice became more resonant and

melodic, and there was a sense of dignity, somehow, as she spoke. After about two sentences, her vocal timbre became thinner and her speech increased in tempo and become less compelling. I asked her if her heart plug had become disconnected. She checked her mental image and saw that, in fact, the plug had fallen out.

Attributing this disconnect to habit, she plugged it back in and continued talking. When she had spoken her piece, she acknowledged that she had felt a shift in her vocal production when she'd become "unplugged" and that she understood, for the first time in her life, what it meant to "speak from the heart."

She also said that she understood suddenly what it meant to "open your heart." She said that she had heard that phrase many times in church, and it always sounded like a noble sentiment, but she had never realized that it was an instruction to be taken literally. Her heart felt open, and it felt wonderful.

Also, her extremely faint tremor stopped while her heart was connected.

I decided to try an experiment. Prior to working on her head, she had said that her main remaining problem was her right arm. In fact, she said that if her right arm was able to move in a relaxed manner, she would consider herself to be essentially recovered. I had, at that time, asked her what it needed to happen in order to get her right arm working better. She had said that she didn't know.

A sudden shift in body awareness

As she imagined that her heart was hooked up to her brain, I asked her again what her arm needed. This time, Aggie immediately replied that the problem in her right arm was coming from her right leg. She focused on her right leg and could clearly see a blocked off area in her hip. I asked her to make it more blocked. She imagined this for less than a minute and then relaxed her focus; the blockage was gone. Without doing any techniques to bring light into her leg, she easily saw, in her imagination, that light streamed past her hip and through her thigh until it came to another blockage in her knee. She repeated the formula and that blockage was gone. Continuing down the leg easily, she saw a small glitch at her ankle. I asked her to make it "glitchier."

(I never told a person exactly what to do with the problem. I always used the same words that the PDer used and just ask that the problem be exaggerated. This way I don't need to understand what a person means when he says the problem is too "bleak" or "too linty," words that often convey nothing to me but which obviously mean something specific to that PDer.)

As soon as the ankle glitch was gone, Aggie could see clearly that the smallest toe was too "dark and bent." I asked to make it darker and more bent, which she did. And then she told me that she was fine; she said that she knew in her heart that her leg, finally, was fine.

Imperfect arm

But when Aggie moved her arm, it still did not feel perfect; it still felt "unnatural." I asked her to make sure that her heart was hooked up. She confirmed that the heart was hooked up and that there were no blockages. At this point, I realized that she might not actually know how to move her arm. She moved it as if arm movement began at the shoulder, as if the arm had no relationship to the rest of her body.

I reminded her that the power for large movement function comes from the base of the spine. Now, she was easily and comfortably able to imagine energy flowing in her body, a new ability that seemed to have accompanied the imagining of a brain-to-heart hook-up. I asked her

to imagine energy flowing up the spine, out through the shoulder, and down to her fingertips. When she moved, she must use her whole body, just as any athlete or musician knows. When one moves the hands while playing a violin or swinging a baseball bat, the movement does not start with the hands; the movement starts in the base of the spine and flows up and over to the arms and out the fingers. The base of the spine is like the handle of a whip; the fingers move like the whip's cracking tip.

Aggie practiced several times imagining that energy was starting at the base of the spine and flowing up and out her fingertips. As she imagined it, she threw her arm outwards and up towards her head, imitating the graceful arm sweep of a ballerina. (Aggie had loved ballerina coloring books when she was a girl.)

She was starting to get frustrated with the arm movements because they still felt stiff and unnatural. I kept reminding her to make sure her heart was hooked up. She kept flinging her arm up over her head (she was lying down on the treatment table). Suddenly, after about the fifth try, something changed: as she imagined the energy coming up her back, her spine moved, as if in time with the impulse. When the energy got to her neck, her neck and head gracefully swayed slightly to the left, balancing the movement of her right arm as it floated out and up.

Aggie opened her eyes. They started to fill with tears. "That felt so good. That felt SO GOOD. That felt *SO GOOD!*"

She continued practicing for a few more minutes. I reminded her several times that it might take some time to overcome her past habit of being disconnected from her heart, that it might be a slow process making these changes permanent. I kept reminding her to check that the heart was still connected. And then, the session was over; another PDer was waiting in the wings.

Lydia

I wasn't sure that I would do the heart-mind connection visualization with Lydia, the next PD patient of the day. I had never met Lydia before. She was forty-three years old. She'd been diagnosed five years earlier. Her close friend from massage school had been her FSR practitioner for the last four months. I welcomed them both into the clinic and began the intake. Lydia shook my hand at the door and hugged me, then thanked me with warm words for the work we were doing. Her stony, emotionless face and body language contrasted with her verbal expressions of warmth.

My first impression was that Lydia's case was quite advanced. She was in a lot of pain from the excruciating rigidities in her legs and hips. Her face was nearly expressionless: her right-side face could not move; she spoke out of the left side of her mouth. Her arms were bent at the elbow, and all her small motor movements were painfully slow and rigid. She labored to remove her shoes. Her right foot was grey, as if it belonged to a corpse that had been a few days in the water. Her voice was not resonant. Her tremor was not particularly large, but there was clearly a faint tremor, especially throughout the right side of the body.

She told me that she had never used PD meds, but that she had, three weeks earlier, started using muscle relaxants once or twice a week. She used the pills because the rigidity in her legs was so painful that she had been unable to sleep for three nights in a row. That's when she had gone to the doctor for the pills.

Then, before I had a chance to reply, she announced defiantly: "I have *not* done the visualization exercises in your book. I don't do visualization; I'm not a visual person. I can't do it. Besides, you didn't do a good job of explaining what you meant. Was I supposed to be imagining that I am looking at my legs from the outside or the inside? I have no idea what you

were even talking about.” She continued in this vein for quite awhile, emphasizing that she was not a person who did visuals.

I asked if I might determine the direction of Qi flow in her legs and arms. (I do this by holding my hands a few inches above her skin, using my hands as Qi detectors.) She agreed, and lay down on the table.

Backwards Qi times three

Lydia had Qi flowing backwards in all three of the leg channels that flow from the head to the toes. The Qi in the other three leg channels, the ones that flow from the toes to the torso, was extremely diminished. The Qi in her arms was flowing backwards in two of six channels on both arms: each arm had *different* channels that were running in reverse.

I felt her feet. They were like the stone feet of a gargoyle. They looked dead. Aggie’s practitioner piped up for the first time: “I think Aggie’s feet look a lot better than when we started.”

This was an advanced case.

Should I try the heart-brain connector idea? She had already told me that she couldn’t visualize. I decided that Lydia had nothing to lose, so I told her, “I want to try something with you that I haven’t done before with someone in such an early stage of treatment.

“I’ve been working on a new technique, but it requires visualization skills. The other two people I’ve done it on have been working with me for a while, and their leg Qi had already been corrected. Their Qi was flowing the right way through their feet prior to my doing this technique with them. But if you’re game, I’d like to try it with you.”

Lydia and her practitioner had written in their brief patient-history notes that recently, on three occasions immediately following an FSR session, Lydia had felt somewhat less rigid. The looseness did not last more than a day, but nevertheless, this temporary experience with loosening up gave me reason to suspect that, even though her feet looked like death, a narrow pathway might have been cleared in the feet. This pathway possibly allowed Qi to flow correctly for a short while until the overwhelming trend of the blocked up legs caused the Qi to revert to “backwards.” This was the only encouraging news that they had, but still, it seemed promising.

Lydia was willing to try the new technique.

I started out the same as I had with Gil and Aggie. I asked her look at her brain and see if the two sides were symmetrical and aligned correctly. She could do it easily. As noted earlier, most PDers, even if they “can’t visualize,” can easily pretend that they are looking around inside their heads. Then again, for a few PDers, even thinking about getting ready to try to visualize any body part is frightening; visualization itself is nearly impossible.

In Lydia’s case, one side of her brain was too big and the other side was too small. I asked her to work on them one at a time. I asked her to make the small one smaller until the exercise was no longer interesting and then let the image drift back to whatever it was going to become. When she was done, it was a little bigger, but did not match the other side. So I had her work on the other, “too big” side, making it bigger.¹

¹ For some reason, over 95% of all the PDers we’ve tried this on have imagined that the left side of the brain is much too small or the right side is much too big. This does not conform to any brain scan findings. This does not fit with the idea that the logical side of the brain, the left side, is the side most used by PDers. This also does not have anything to do with the side of the body that first developed symptoms of Parkinson’s. Based on

When I explained to her, briefly, that PDers usually don't like to imagine good stuff happening to their bodies, but they are great at imagining bad stuff, she laughed out loud – her first physical manifestation of positive emotion since she'd arrived.

We continued imagining the brain, finding a few imaginary bumps and bruises and enlarging and exaggerating them until they diminished or disappeared.

Next, I suggested, “Now find the place in your head where there is too much electrical activity.”

She found it quickly. I told her, “Increase the amount of activity as much as you can.”

She did that. She said she increased it so much that “smoke was coming out of her ears.” When she stopped doing the exaggeration and the smoke cleared, she still imagining that there was a problem in the “electrical activity” area.

I said, “Look all around and see if the connections going in and out are all in good working order.”

She told me that there were a specific, modest number of connectors, and that they were all frayed and rotting.¹ (At this point in the book, I will not tell the PD reader how many connectors she imagined, lest the reader think he too must see the same number of connectors. Some PDers see five, some see dozens, some see a great mass of connectors. It doesn't matter how many or how few a person imagines.) I asked her if she wanted to fix them straight out or first make them worse, following our “make it exaggerated” gambit, so that they could then rebound into health. She told me that she was going to rip them all out and let new ones form.

I was holding her feet as she did this. I sat there for about a minute, giving her time to tear out the old connectors. Then I asked her if they were now in better shape. She said that they were now doing fine. I waited to see if she had, on her own, noticed that anything was missing. She hadn't.

I asked her if possibly there was one connector missing, or if there was room for another one, or if there was a connector floating around, not plugged in to its correct spot in this area. She said, “Maybe.” But she didn't see one readily.

I then took the initiative. I told her that very possibly there was a connector that was coming from the heart, and it was supposed to be connected to this area of excess electrical activity. She said she would try and find such a connection.

subsequent findings, I have to wonder if the left side of the brain is usually imagined to be smaller, darker and more foul because the left side of the body is thought of as housing the heart – an organ that was usually imagined to be too small or too dark or too diseased, and certainly not capable of being beautiful.

Also, as we discovered later, the Qi flow in the portion of the left-side Stomach channel that courses over the heart is often running backwards or at a standstill in partially recovered PDers, even if the rest of the channel is flowing the right way. This electrical pattern over the heart occurs to varying degrees in any person who is in sympathetic mode. We do not know, but conjecture that this electrical pattern may be the reason that most PDers have more difficulty imagining the left side of their brain.

¹ I hardly want to mention how many connectors she saw, for fear that PDers will think that they need to imagine the same number. However, I find it curious that the most frequently imagined “correct number of connectors” for brain areas were five, six, eleven or twelve. It seems to me that this number possibly relates to the five senses and the heart types of connections: six in all. Maybe people who mentally imagined eleven or twelve were simply having paired connectors for each sensory function and the heart function. Again, the correct role of the mind is that of a secondary processor for sensory feeling – not the “seat of ego and cleverness.” If a person needs information, the heart, attuned to Wisdom, can always supply it. However, if the mind is chronically dominant, Wisdom is rarely available. Instead, bits of accumulated information have to take the place of Wisdom. PDers are often keen gatherers of information and trivia.

Lydia gets hooked up

About a minute later, she told me that she'd found the wire that was supposed to go to this area, but that it wasn't even in her head: it was down by her heart. She had had to bring it up from her heart. It couldn't pass through her body: she had poked the wire out through the chest wall, run it alongside the neck, and then poked it into her head behind the ear. Then she had hooked it up to the place where it was "supposed to go." (Strangely enough, this imaginary pathway is very similar to the route of the vagus nerve, the parasympathetic nerve that communicates between the heart and the brain when one is feeling contented.)

I then asked her to temporarily disconnect it, notice how she felt, and then connect it again.

She said that, with it connected, she felt that her mind was calmer and her heart was bigger.

That was good enough for me. I asked her to please try and keep it connected while we did some mind-body visualization work. She agreed, and we started.

I asked her, with no preparatory explanation, to imagine light streaming through the inside of her left arm (her one functional limb). There was a pause, and then she said that she had done it; there was light down to her fingertips. I compared this ease of light flow with her flat-out statements, not twenty minutes earlier, that she could not do visualization, that she didn't know what I meant by visualization or by the phrase "imagine light in your body."

Next, going for broke, I asked her to send light down into her right leg (her worst limb).

After a short pause, she said that she couldn't get the light any further than the inguinal groove (the groove where the leg meets the torso). I asked her why not, what prevented her from visualizing her leg.

She reverted somewhat to her previous attitude about visualization. She said, "What do you mean? What am I supposed to be looking for? Am I supposed to be looking for my guts? My bones? My muscles? What?!"

I said, "Make sure the heart is still connected."

She replied, "Oh. Yeah. The connection fell out. I'll put it back."

I then prodded her to continue by asking her once again to try to get light into her leg.

This time, she said, "I can't. It can't get through."

I asked her why not. She told me again that it couldn't. We went back and forth a few times: me, using various vocabularies, me asking her why not and her replying simply that she couldn't.

Finally, I asked what was there that was preventing her from getting the light through. She replied (in a tone of voice that suggested "well, duh! It should be obvious to *anyone...*"), that there was a wall in the way.

"Oh. A wall," I said. "Of course. Go ahead and make it more wallish. Exaggerate it."

She was quiet for about a minute, and then she chuckled. I asked her what was going on. She said that the wall had turned into a wad of string. I suggested she make it more string-ish and more wad-ish. A few moments passed and she giggled. Then she explained, still with her eyes closed, "The string turned into a ball of lint, so I blew it away. It's all gone now."

The rest of the journey into her leg was difficult but doable. Filling the leg with light was slow going. The leg was thick and murky. She ended up using a roto-rooter type of drill to cut through the muck. Please bear in mind that twenty minutes earlier this person insisted that she did not do visualization and did not even understand what I meant by the word.

Based on my previous experiences with PDers I felt certain that, if she had not first connected her brain to her heart, she would not have been able to visualize the injuries and obstructions inside her leg. This type of visualization, in which injuries become recognizable, is extremely hard for most PDers, even those that have tried for months and who can create spurts of imaginary light in various areas. I was baffled as to what role the heart-brain connection was playing. Why could Lydia easily detect the “wall” in her inguinal groove after hooking her heart up to her brain? Aggie, too, had easily been able to tell where her problem spots were – after she pretended to connect her mind to her heart. Was the heart somehow the missing link for people who were stuck in partial recovery? And of course, the heart wasn’t actually missing. Were recovered PDers somehow creating a mental construct in which they were pretending that their brain and hearts were disconnected?

At this time, I was not aware of the physiological shifts in the neural circuitry that essentially disconnect the heart and brain wave patterns during times of crisis. Nor was I yet aware that, by virtue of neural reciprocity, a person could create this same shift in circuitry by pretending that he is cut off from his feelings.

An unexpected Qi flow shift

Even before she got down to her feet, I realized that Lydia’s legs looked different somehow. They looked not just more relaxed, but energetically changed, almost as if they were brighter, more alive.

I reached out and felt the Qi flow in her legs. It was running correctly in all channels. Evidently, the “wall” at the inguinal groove had been obstructing Qi flow enough that, even with the work her practitioner had done, there was not enough Qi momentum to get the Qi running consistently through the channels in the correct direction. Now, with the “wall” gone, the Qi was able to flow easily and correctly. I was stunned. I had never seen such a rapid correction of Qi flow.

The Qi began streaming through the Stomach channel points on her face as well, no doubt because the healthy pattern in the Stomach channel was now allowing the arm Large Intestine channel to flow correctly over her face. Her face lit up in a smile.

I could hardly believe my eyes.

This was the fastest I had ever seen the face part of the channel make the transition from Off to On. Admittedly, reconnecting the heart and mind was not the only work she’d done; she’d been getting regular FSR treatment for four months. A few times, recently, following an FSR treatment, her face had momentarily exhibited expression. But this time, the entire face was lit up.

Evidently, the previous FSR on her foot had been invisibly working, even enabling her to experience brief moments of corrected Qi flow, but it had not addressed the “wall” in her inguinal groove. Possibly, she was still dissociated from her heart with regard to some inguinal groove injury. Maybe the “wall” was a part of that dissociation. The “wall” might have been the mental construct that caused the Qi flow to be minimal and caused it to keep reverting back to its old, wrong path.

I asked her about the wall. She said that she recalled no injury, but that ever since she was a young child, every sneeze or cough had caused a painful pulling sensation in that area. Also, every time she ran, even as a small child, she would get a “stitch” in that spot. Probably, through the years, her mind had walled off that area to prevent the pain. She may have used dissociation

from her heart to perform the walling off. At any rate, with the wall gone, Qi was once again flowing through her feet.

As for her feet, right there before our gawking eyes, they were changing from grey to pink. Her practitioner, thinking that this was the sort of marvel we saw all the time, said how impressed she was with these techniques. As for myself, I needed to sit down.

Her session was over. I reminded her that she should make sure, over and over, that her heart was plugged in, gave her a hug, and sent her on her way.

Later that day

Laura Walter, a member of the PD team, called me that evening. Laura had been the very first team member to see Lydia. Laura's phone call interrupted my talk with my husband, as I was excitedly, no, frantically, told him of the day's events.

Laura opened the phone call with "We've got a problem with the new person." She went on to say that she'd never seen such distorted Qi flow, such a severe frozen-face on such a young person, and that the feet reminded her of one of our earliest PD patients – the one with "the worst feet ever."

Laura went on, "I think we need to be honest with her: I don't think we can help her. Her Parkinson's is advancing so fast, I think we should tell her that she's too far gone."

I turned off the excited, soprano voice I'd been using on my husband and assumed my most casual tones. With a voice almost too bland, I assured Laura that all those problems were going away: the face was now practically normal; the feet had turned pink; and the Qi was running correctly. I agreed that her body was a mass of injuries and that it might be a long, painful journey as she discovered and healed all her injuries and relearned normal movement. But I assured Laura that the Parkinson's patterns had been turned around and that the worst of the job was behind us.

After Laura laughed at me, I told her what I'd been doing all day. I gave her all the details. She hesitantly thanked me, expressed polite surprise, and rang off.

The next day

I saw Aggie first. In case you are wondering why I was seeing these PDers two days in a row when we usually recommend once-a-week treatment, Aggie and Lydia were from out of town. They were visiting Santa Cruz and seeing various members of the PD Team every day for a week. Both Aggie and Lydia were receiving treatments twice a day, from two different PD Team practitioners each day. That was especially valuable to me today, because it meant that I would have a team member to corroborate what I was doing.

I'm not sure what I expected from Aggie, but I was surprised by her. She walked in and announced, "I had a meltdown yesterday."

She went on to say that she was angry with her beloved husband because he could shower so quickly. She was frustrated with herself and angry that it was taking so long to recover. She was resentful, sad, jealous, and boiling over with emotions. The worst emotion was the rage she felt at herself because of her failure to keep the heart plugged in. She kept checking to see if it was plugged in, and it usually was not. Also, she was bitterly disappointed that, upon awakening this morning, she was still moving the slow way that she usually moves in the morning. She was blaming her failure to keep the heart connected with her seeming inability to move in the normal fashion that she had done yesterday for a few minutes.

The most strange thing about all this was that, until this morning, she felt that her emotions had always been in control, and that she had been contented and faithful that everything would somehow be all right in the end. Suddenly, today, she was feeling anger, disappointment, jealousy, frustration.

I talked to her about how slow it can be to make new habits and overcome old ones. I pointed out all the positives: twenty four hours earlier she didn't even know she had a heart connector, and now she could find it easily and work with it. Plus, despite her slowness that morning, she could not deny the fact that, for several minutes the day before, she *had* moved normally. I didn't mention that many people experience childish emotions when they first get their heart working again. I didn't mention it because we didn't yet know it. She was one of the first to demonstrate this phenomenon.

We talked for a long time about realistic expectations. I thought she was making great progress. Despite my reassurances, she was uncharacteristically emotional: wailing over the fact that she hadn't permanently retained her ability to perform loose, relaxed movement.

So, I had her get up on the table. I asked her how her legs looked from the inside. She said that various spots here and there still wanted some work, so I held those areas while she imagined every situation being worse than it was, and pretty soon she had calmed down.

I asked her if she wanted to try a habit-changing technique that works by mentally cauterizing the brain cells associated with a bad habit. She did it and then felt a warmth coming from the side of her head where the heart disconnect problem was located.¹ She also mentioned that she remembered this technique from a class I had taught a year earlier in St. Louis. I asked her if she'd ever actually tried it before today. She said she hadn't.²

¹ This technique and other helpful attitude-changing techniques are included in the appendices.

² I used to be astounded at most PDer's disinterest in doing the techniques that I share in this book and in my classes – even those PDer's who are certain that this program is the answer to their problems.

Sometimes PDer's come to our program from a long way away, even from other continents. They usually say they have read the material several times. They claim to be keen to “get started” with recovering, and can't wait for us to get to work on them.

But when we ask if they have tried to get started on their own, if they have imagined light in their body, tried to open their hearts, or done any of the attitude-changing techniques in the appendices, they usually say that they aren't interested in doing that stuff on their own; they want to work with us, in person, because we are “the pros.”

This attitude was puzzling. These people have usually been, for much of their lives, extremely “can-do:” so competent, so capable. But when it comes to actually changing their own negative or fear-based mental habits or learning positive ones, they often were not interested in initiating the work. We now understand that, with a dissociation response in place, the mind is hardwired to not visualize, not open the heart, and not imagine positive outcomes. These instructions are part of the survival mechanism that is part and parcel of the dissociation response.

Now, when working on changing a PDer's mental habits, it is helpful for us to behave as if we are working with a child, not an adult. The types of fears we often find at the root of the dissociation can, in some cases, suggest that we are working with people who have severely arrested emotional development, in terms of facing fears, even while they have overdeveloped mental aptitude for word-based logic and negativity. We now suspect that it is this childlike inability to confront a large fear, combined with an enormous mental ability to control one's own body processes, that spurs some people to consciously maintain a dissociation response, preventing it from turning off in the normal time frame.

We suspect that some PDer's wait until they are with us to start practicing having a heart because they feel, on some level, that we can best perform the role of supportive adult: the adult that a child clings to when he doesn't feel safe.

I asked her if she wanted to try moving in a loose way again today. She was uncertain, but finally decided to try it. Yesterday it had taken her some minutes, maybe five, to figure out how to integrate the base-of-the-spine “whip cracking” imagery. Today, it took only about two minutes until she was once again moving gracefully. As she practiced waving her arms and neck and torso gently side to side with her eyes closed, I pointed out that all the things she’d done yesterday she had done again today, and she had initiated them more easily. When she stopped forming ballerina moves with her arms, I pointed out that she had maintained the relaxed movement for about four times longer than the day before. I think that it was starting to sink in that she was, in fact, making steady progress, but that she was not going to switch from unhealthy to perfectly healthy overnight.

She agreed that her expectations had been unreasonable. She also agreed that, with her heart plugged in, she could do all the visualization work much more easily, her mind was calmer, and she could see how her recovery was accelerating. I left her on what I thought was a high note.

Only later did I begin to wonder at the sudden appearance of so many conflicting emotions in what had always been a person of calm self-control.

Lydia again

Lydia strode in next, looking great. She was still fairly rigid all through her body, but not painfully so. She was grinning from ear to ear. Through the two-hour session, she asked, at twenty-minute intervals, “Am I still smiling?” (Her husband was going to fly in to town to join her the next day, and she had not told him about her new smile. She was planning to surprise him. As an aside, when she had first seen her own smiling face in my office mirror the day before, she had been, at first, disquieted by the unfamiliar image. Then, as she kept looking at herself, the smiling face became “strangely familiar.”)

Lydia’s Qi was still running correctly. She had slept well; her legs had not slipped into that extremely painful rigid contortion. Her heart kept coming unplugged, but she kept plugging it back in.

I asked her how her legs were, if she could fill them with light. I also asked her to notice, as she did so, any way in which her body or legs differed from what “perfect” body and legs should look like. She started to fill her body with light but only got as far as the hip. There was a problem with the pelvic bone. In her mind’s eye, it was sticking out of the side of her body. I asked her to make it stick out further. She enlarged it so that, mentally, it was hitting the wall of my office, and then it shrank down and, to her mind’s eye, was unblocked and “correct.”

She wanted to know why there had been a problem in the hip today, even though she had gotten rid of the wall yesterday. Not only that, she could also “see” and feel other problems in her leg that she hadn’t noticed the day before. I had to point out that the wall had merely prevented mental and/or emotional access. Now that she could actually feel her own leg, she might be finally able to notice that there were lots of areas in the leg that were in need of healing.

We had seen in other recovering PDers that once access was gained into an area, a whole collection of long-forgotten, unhealed injuries often appeared: sprains; strains; and even bone breaks. Unremembered injuries or injuries that “hadn’t hurt” at the time they occurred are often exposed after the Qi starts to flow (after the primary Qi-blocking injury has been rectified). The events that caused these injuries are often clearly remembered once their pain kicks in.

Of course, as long as the area is mentally blocked off, and especially after Qi stops flowing correctly in the area, neither pain nor healing is able to manifest correctly. After Qi begins to flow correctly, the pain of old injuries can begin to register. Then healing can begin. Usually, the injuries will surface in a gradual sequence. One injury will make its presence known, and then begin healing. Sometime later, another one will show up.¹

Lydia had lived an unstoppable, dynamic lifestyle. Keenly aware of Lydia's long list of *remembered* accidents and injuries (none of which had hurt at the time) and suspicious that there just might have been some others that she hadn't even written up in her list of injuries, I gently warned Lydia: now that she had mental access to her leg, forgotten injuries were probably going to be calling for attention for some time.

Even though she could now imagine or pretend to visualize her body with ease, this did not mean that her body was completely healed from every past insult or injury. She was disappointed. I think she had imagined that she should be recovered completely, all symptoms gone, in a day or two, simply because the Qi was now running the right way. I had to explain that the pathological Qi flow pattern was gone, but that she now needed to heal.

We started working on some leg injuries that were now quite "visible" to her. They were also painful. It seemed that every few minutes she discovered a new ache or pain. Two days ago, she had been paralyzed with general rigidity and dystonias. Now, instead, she was feeling the pain at the places where the motorcycle had fallen on her legs (which hadn't hurt at the time). She addressed the problem areas one by one by focusing her attention on them until they seemed less bruised (to her mind's eye) or felt slightly less painful. Frequently, she started to tremor a bit and I would ask her if her heart was plugged in. She would replug the heart, the tremor or rigidity would stop, and we could continue.

She finally got frustrated with the heart becoming unplugged. She examined the wiring carefully and saw that there was a toggle switch near the heart that was causing the plug to be

¹ This pattern, in which the body almost always recognizes the most urgent pain, and can ignore lesser pains until the urgent one has stopped, has been long recognized in studies of pain perception. This may be why, in recovering PDer's, they are not able to notice all their injuries at once.

One recovered PDer, years after having completely recovered from Parkinson's, woke up one morning with the bridge and sides of her nose badly swollen and painful, with a faint darkness under one eyelid. She said that it felt as if she'd broken her nose. It was swollen and painful for several days. She had not bumped her nose recently. But she did recall a faint, junior high school-age memory of saying to someone, "Don't worry. I'm fine; it's no big deal: everyone gets their nose bashed once in a while." She could not recall the context of that statement, made 40 years earlier. But when the swelling of her nose subsided, there was a small, permanent discolored line across the dented place on her nose, the type of marking that one would expect from a broken nose.

Curiously, she had long been vaguely aware that her nose had a strange dent in the long ridge, as if it had been broken, but she had never had any awareness of having received a nose injury. A review of her school photos showed that the misshapen nose had appeared in junior high school.

Since recovering from Parkinson's, she'd had many similar incidents in which bruising and soreness showed up, and seemed to trigger the memory of some long-ago injury. She found the nose injury fascinating because she could see the proof of injury in her school yearbook photos. She was certain that she had never had any pain or swelling on her nose during those self-conscious junior high years when such a disfiguring bruise would have been noticed and mentioned by her schoolmates and herself.

The point here is that, after recovering from Parkinson's disease, many of her other forgotten injuries had "appeared" and then healed – all of them several years prior to her body deciding to spit on its hands, haul up its socks and get to work on the broken nose bone. We must conclude that the subconscious mind, left to its own devices, may pick and choose when and where it is going to heal the body's non-emergency injuries.

It is also important to note that she had completely recovered from PD even though she evidently still had an assortment of unhealed injuries. The anxious PDer can take heart from this: it means that a person can recover from Parkinson's even if there are still lots of unhealed injuries.

connected or not. She installed a metal bar over the toggle switch to prevent the switch from being thrown, and told me that, from now on, the heart would stay connected.

Habit being what it is, I was silently dubious, but hopeful. About five minutes later, while working on her leg, I noticed that she seemed taut and mildly shaky again. I asked her if her heart was connected. When she checked it, she found her heart disconnected in a new way; her habit of needing to shut out her heart (emotions, feelings) when confronted with physical problems had found a way to get around the toggle-switch stabilizer. In her mind's eye, a stick was now poking directly into her heart, preventing it from working correctly. The imaginary stick protruded out of her chest and up into her nose. Lydia actually thought it was pretty funny. She got rid of the stick and acknowledged that very possibly the habit of heart disconnection was going to take some time to unlearn.

I then held her feet for a while. They were still pink and healthy. The grey had never returned. Some of the foot bones moved a modest amount. She asked every twenty minutes if her smile was still there: the innocent, happy and excited tone of her voice reminded me of a child who keeps asking his mother if his tooth is loose enough to pull out yet.

Later on

That evening, I called my son, a PD-team member. When I expressed my surprise that Aggie had been so emotional, Clay chided me, "Mom! She has a heart again. She has feelings. She's not used to that. She hasn't really had feelings for who knows how long. Of *course* she had a meltdown."

"Oh." I replied. "Right. I hadn't thought of that. Tell her that tomorrow when you see her." We talked a bit more. I briefed him on the new heart-mind connection idea. I hung up and the phone rang again. It was Laura, with whom I'd spoken the night before. I will paraphrase her words:

"Omigod. I'm in shock. I didn't believe you yesterday. I didn't believe you. I didn't know why you were saying all those crazy things about Lydia, saying that the PD had already turned around.

"I have never seen such a change in a person. Her feet, I swear, the worst of the varicose blood vessels were disappearing beneath my hands. Maybe it's because she's so young and so healthy, but I've never seen anyone change so fast. Her face is normal! Her feet are healthy-looking. They are still a real mess, structurally, but there's life in them!"

Laura went on and on, describing what she'd seen. Then she wondered if possibly a heart-mind disconnect was the reason that so many PDers seem to get better in many ways and then get hung up on some part of the recovery.

Curiously, a partially recovered patient with whom I'd worked for two years had recently said, several times, "I'll never be able to recover. I know my foot doesn't hurt anymore and I can walk more easily, but there is something wrong with my *heart*. I can't access my feelings. I know that I'll never be able to feel emotions like a healthy person. Maybe this is why I'll never be able to recover."

Laura went on to say, "Won't it be wonderful if, by reconnecting with their emotions, *all* of them can recover in the same time frame as the easy recoveries, five weeks to a year?"

Listening to her excitement, I was relieved: I hadn't been imagining things when I'd seen Lydia's lightning-fast changes. Laura, too, had seen the same dramatic changes that I had seen. Despite my studied calmness when I'd spoken to Laura previously, I had been almost afraid to

credit my own observations. Laura said that I'd better call all the team members and inform them of what was going on. I agreed, and we hung up.

I was almost ready to be certain that we had found a magic key. But I still had to wonder what the next day would bring.

The third day

I did not see Gilbert, Aggie or Lydia on the third day. Instead, I got reports from two of the team members. Clay saw Aggie first, and called me on the phone afterwards to say, "Why is she coming here for treatment? There's nothing wrong with her."

I pointed out that this was his first session ever with Aggie; he'd not seen her a year ago when she first came. I also asked him if he had noticed that her walk was still hesitant and she was very, very slightly bent forward at the waist. He agreed on those points, but also said that he had done some slightly Yang (vigorous movement) Tui Na on her shoulder blades and that she had loved it. More importantly, the shoulders remained relaxed when he was done.

He had supported her leg and hip, and she had rotated her leg in huge circles. The rotations started up in the low back and were smooth and languorous.

He had done some craniosacral work on her neck and her neck loosened up and became nearly two inches longer – and stayed longer. He felt that she was no longer in need of our services; all that she needed from here on out was a little "clean up work."

As for Lydia, he reported, after seeing her for the first time, that she was a "real piece of work," but that her Qi was flowing well. He said that she was a mass of injuries, but he saw no reason why she wouldn't respond well. He went on to say that she was really determined to do the work. When she'd walked in, she had told him that her heart had stayed connected for three straight hours. She was upbeat, smiling, in a lot of pain, but not especially rigid.

Another set of opinions

Laura had also worked with Gilbert, Aggie and Lydia that day. She called me later that evening.

In Gilbert's case, she asked if it was possible that he had grown two inches taller. He was doing so well, she thought that maybe we didn't need to see him any more. He had been nearly recovered anyway, but she felt that he'd made some quantum leap forward in the last two days.

She also said that both Aggie and Lydia had been extremely discouraged: every time they went to check on the heart to see if it was connected, it was not. They were starting to feel that they could never overcome their habit of being disconnected.

Laura is a quick thinker. She had asked both of them, "Do you say to yourself, 'Uh oh, I'd better check to see if my heart's disconnected.' And then, when you've looked, it was, just as you'd feared, disconnected?"

They had both agreed that this was the case. So she had told each of them to never again ask whether the heart was disconnected. Instead, she wanted them, whenever they thought of the heart connection, to say, "Ahh. My heart is connected!" and then, having said that, they should mentally look to see the connection.

They both reported that if their connection check-up was preceded with "My heart *is* connected!" then, when they actually looked (with the mind's eye) to check, the heart was, indeed, connected. They played with this concept. When the thought that preceded the check-up was "Uh oh," or "I wonder *if* my heart is connected," the answer was always "no."

At this time, we incorrectly assumed that this thought repatterning, getting rid of the “uh oh,” was merely a matter of habit. We were wrong. As we were to learn eventually, so long as the dissociation response is in place, the brain will try to revert back to a disconnected-heart mode. Turning off a “stuck” dissociation response must happen at the heart, not at the brain. Although most sympathetic responses are regulated by the brain, the dissociation response goes much deeper; it shuts down the thrill of being alive – a heart function, not a brain function – and prepares one for death.

Even if the dissociation response was originally induced consciously, as it is with many PDers, the dissociation process, in a healthy animal, is designed to turn itself off in a short time, or when the highly *imminent* crisis is over and the animal feels safe. Feeling safe is a heart function, not a brain function.

A *consciously*-induced dissociation response in a human evidently does not necessarily turn itself off at the proper time – especially if the person wants to be free from emotional or physical pain. In such a case, the normal mechanism for ending the dissociation response is consciously instructed *not* to work. Despite temporary mental overrides, the heart dissociation response remains in place.

Overseas corroboration

Thinking that I’d discovered The Answer to something, I still needed to see what would happen if distant PDers tried to re-establish a heart-mind connection 1) via working from the printed page and 2) working with someone other than me. Replication of an experiment by another researcher is a crucial part of research.

By great good fortune, Chris Ells happened to be the Netherlands helping to christen the new “Yin Tui Na Centrum Amsterdam.” This meant that, in addition to me trying this technique on our local PDers, Chris could try it on his European PDer patients, quickly widening our subject base. Also, Chris would be working only from the emailed material I had sent him about my work on Gil, Aggie, and Lydia. Chris and I had not yet had a chance to discuss this new technique in person: Chris would have to try to replicate my results working from a written instruction. For scientific purposes, this made the experiment more valuable.

To keep the process somewhat objective, Chris did not share any expectations for the new techniques with his PD patients, explaining only that he wanted to do something new. Also, Chris made sure that the PDers he worked with were not aware of each other’s responses.

Here are his results from the four people that he saw the next day (the treatments each lasted two hours):

The first person did not see wire-type connectors going in and out of the area of excessive electrical activity; she saw doors and windows. One of the windows was stuck and could not be opened. She decided to throw a rock through it. This allowed wind to blow through the area: she had always felt most at peace when she was out in the wind. After “opening” the window, she felt calmer in her heart. Chris said that she seemed more radiant somehow.

The next two people on whom he tried it saw wires going in and out of the area of excess activity. However, there were no missing connections. On closer observations, however, they both saw that the wire coming from the heart was not in good shape, so they replaced the wire. After replacing the wire, they felt more peaceful.

The fourth person could see that all the connectors were in good shape. Even the connector to the heart was good. Chris asked her to look at the heart itself. The heart had blue spots on it that didn’t look right. She made them bluer and bigger. She became very quiet. Chris

asked her how it was going, and she said that the whole heart was blue. (Chris told me there was deep sadness in her voice when she said her heart was blue.)

Chris suggested to her that she relax her focus on her heart. Her mental image of her heart, instead of reverting back to a heart with blue spots, became one of a healthy heart, completely “the right color.” After the heart became the correct color, she felt very calm.

Ease of visualization

Possibly the most important development was that, in all four of Chris’s cases, their new relaxation and calmness was immediately followed by an significant improvement in their ability to feel and/or visualize their bodies. This new awareness then enabled them to direct Chris to areas of the body that needed work. For example, one person suddenly perceived a walled off area in his knee. Another realized that what her toe problem needed was some work on her ankle. Also, with their hearts “hooked up,” they were all able to respond much, much more quickly than usual to the FSR work that Chris then did on these body parts.

After years of having worked with PDerers who, typically, have no idea that there is anything actually injured and cannot really “feel” what is going on in their bodies, Chris felt that the transformation was stunning. Then Chris said to me, “It just makes sense. How could they have feelings about their body if they didn’t have feelings, period? Feelings come from the heart.”

Over the next few days, Chris worked with many more PDerers. Their responses were, for the most part, extremely gratifying. Chris also invented variations on the technique to fit the various PDerers.

One particularly noteworthy variation involved a PDer who was utterly unable to visualize. He was so adamant that he could not do this technique that Chris decided to try a different approach. Chris wrote to me in an email that he asked the PDer to place his hands on his heart and just “look around in the area of your heart.”

In Chris’s words, “This guy can’t visualize squat. He really tried. He could not form any visual image in his mind’s eye. His wife asked if he could try to visualize the painting that’s in their bedroom. He replied, ‘Why? I see the real one every day.’”

For the next hour, Chris held his foot. Every five minutes or so, Chris would say something like, “You’re doing fine, keep it up.”

After the PDer had “looked” at his heart for about forty minutes, Chris felt a sudden stillness in the PDer’s leg, right at the spot where Chris was holding. A moment later, the knee, in Chris’s words, “unsprang like a spring-loaded slingshot and the PDer blurted out a sudden, brief yell/cry.” For the remaining twenty minutes, Chris repeated a few words of encouragement every five minutes or whenever he sensed the PDer becoming “scared, anxious, inept, or in any other way intense.” This time was “characterized by some pretty cool (but way short of completely done) releases” in the PDer’s leg.

More experiments

Over the next few days, while Chris was doing this, I tried the heart reconnecting technique on several more PDerers. Some had results similar to Gil, Aggie, and Lydia. But many were still struggling with the idea of merely imagining a brain. Adding a heart to the picture was out of the question.

I’ll share the results of two of the cases that could *not* perform the heart-brain hookup.

Hope: no excess electrical activity

I asked Hope to do this exercise. Hope could see nothing wrong with her head. I insisted that she keep looking. She said that the electrical activity level in her frontal lobe seemed greater than in other brain areas, but it did not seem excessive. The heart appeared to be connected. She mentally looked at her heart, looked at all the connections, and everything seemed to be in place.

I stopped worrying about the mind-heart connection and took up where I had left off the previous week: working on her visibly torqued knee and hip. Interestingly, and possibly due to my suggestion that she be on the lookout for excess electrical activity, Hope suddenly became aware of something new. She “saw” an area in her neck that she had never been aware of. This area in her neck was definitely manifesting too much electrical activity. She could also feel, for the first time, that the place in the neck was sort of painful. As the session came to a close, she wondered if the neck place was connected with the old knee injury that we were working on.

Most significantly, Hope did not have a major breakthrough. The new technique was just one more exercise that she was more than willing to do.

But after several sessions with Hope, during all of which she had no problems imagining herself as beautiful and bright inside, I remembered her Mirror Image. She didn't want to look at the brain of the Mirror Image. When she finally steeled herself to do so, she saw a brain with an area of excess electrical activity, a heart that was black with barbed wire, broken glass, and nails around it, and a collection of brain areas that were walled off or hidden in caves. Hope was willing to do the work of getting rid of her mirror imagine, but she was understandably uneasy at first about connecting to her own body that agitated brain and that formidable heart. In fact, her Mirror Image's heart specifically told her not to do so. Hope's mind had to overrule Mirror-Hope's mind.

Over the next half year, Hope was able to do this exercise. It was terrifying and painful at times. I'm including Hope's story to make the point that connecting the brain and the heart was not a snap for all PDer.

Refusal to connect the imagined body and physical body

In that first, exciting week there was another PDer, Sarah, who also did not respond to this new technique with a new sense of inner calm, a cessation of internal tremor, and increased awareness of her physical body.

When Sarah first started looking at her brain hemispheres, she saw that the one on the left was a “complete mess.” It seemed to be a whirlwind of dust and chaos. Confronting it or exaggerating it did not change the mess in any way. Sarah was unable to imagine an area of excess electrical activity for quite a while. Finally, I asked her to just *pretend* that she was imagining it. With the mutual understanding in place that she was only pretending to imagine, she was then able to see the place.

In her mind's eye, she pictured her brain as brown and dried up. Connections were missing, but hooking them up did not help; the area was too brown and dried up. I asked her to look at her heart to see if the connections were OK at that end. Her heart was black. When she exaggerated the blackness, it stayed the same blackness as before. It never changed.

During the session, Sarah said that she felt much more calm and her tremor slowed down. She insisted that the ability to attain calmness was of no significance and that the tremor had, of course, slowed down because she was calm.

Deeply disconnected

When the session ended, Sarah mentioned casually that the images of her brain and her heart had not been taking place in her actual brain or heart. She had imagined both the brain and heart to be about three feet out in front of her body, a good safe distance away. I was disappointed.

Sarah and I had worked many times on her refusal to even try to integrate her mental image of her leg with her actual leg. I had told her how important it was, but she had never been interested in integrating them, though she frequently complained that I wasn't doing a good job of curing her Parkinson's. (She also complained to me that her anxiety was getting worse. When I asked her what mental exercises she was doing to overcome her anxiety – I had suggested many – she said that she wasn't doing anything. Fixing the anxiety was my job.)

Early in our working together, Sarah had told me, adamantly, that the reason she was so anxious was because of all the things going on in her life. Actually, her life was extremely uneventful. Her husband made a good salary. Sarah had very few interests or daily activities.

Still, Sarah was anxious about "everything." Many times, she tried to blame the Parkinson's on "all the things that are making me anxious." For example, she was anxious when her adult daughter, who lived on the opposite coast, drove to a bed-and-breakfast for a weekend getaway. Sarah called the bed-and-breakfast to make sure that her daughter had arrived safely. (Sarah said, "I didn't call my daughter's cell phone, I called the bed-and-breakfast directly because didn't want her [the daughter] to know I was checking up on her.") I repeat, Sarah blamed every external event in her life for "making her anxious." She was not interested in working on changing her attitude: *she* did not consider that she had a problem. Nevertheless, she wanted me to cure her anxiety.

I asked Sarah if she could possibly visualize her brain and heart inside her body. She was not interested in doing so.

I asked her again why she was refusing to work on the problem of integrating her mental leg-image and her physical leg: the leg that had been hit by a car when she was sixteen. She then made a revealing statement: "Well, I just felt so stupid. How stupid is it to step out into the street in front of a moving car? I felt like an idiot."

Evidently, she was not going to do the work of reconnecting her mental image of her leg and her actual leg because bringing up the subject still made her feel stupid. After a year of working with her, this was the first time she had informed me of the ego-based emotional content behind her disinterest in working with most of the techniques that I presented. She half-heartedly did the various mind-body reconnecting techniques with me, but she thought them stupid and pointless.

She really liked her own idea that I should be able to treat her with needles and make her anxiety "go away" without her needing to get personally involved. A few times, she told me to use needles for anxiety instead of using FSR or other PD treatment techniques. The benefits of these treatments were not long lasting. This idea of hers – that all she needed was acupuncture needles – had never panned out but, even so, she clung to it.

Although her foot injury was gone, her Qi was running correctly, and her facial expression and the energy in her foot had improved tremendously, she was increasingly

disappointed in my “system” due to her increasing anxiety and continued tremor. She was waiting passively for some acupuncture needles to “cure” her.¹

Meanwhile, while she was waiting, she was taking no steps to change her glaringly negative and cynical attitude about anything and everything. My sense was that she was waiting for the world to change so that she wouldn’t need to be anxious. The idea of doing the hard work herself of changing her attitude did not seem to be, for her, an interesting option.

At any rate, despite what I sensed was her determination that this technique could not work, she had, just the same, felt more calm and tremored less while doing this “failed” experiment.

MORE FRUSTRATION

Over the next few months, I had all my Parkinson’s patients try this mental imagining. Some PDers could not yet connect an imaginary brain to an imaginary heart. Many of those who could found themselves in physical pain or terrified. But some were able to temporarily improve their symptoms by using this imagery. We hoped that this new visualization technique would permanently cure the problem of partial recovery. It did not.

Over the course of the next year, the heart reconnection exercise was experimented with, refined, and simplified. As PDers shared the feelings and fears that welled up when the heart became connected, we compared these phrases and explanations with what the quick-to-recover PDers had said.

In people who recovered more quickly, there was less resistance to the idea of opening up to the heart to the potential joy *or* pain of the universe. The people who had the most difficult time opening the heart or keeping it open were also the people for whom the word “surrender” most stuck in the craw.

There was one phrase that we heard frequently as partially recovered PDers experienced the sensation of a “connected” heart. As the tougher cases learned to open their hearts, often with much anger, frustration, or tears along the way, it was not uncommon for them finally to calmly remark, as if remarking on the weather, “Oh. I just remembered when I decided to pretend to be this way.” However, remembering the origin of the mental game did not necessarily give a person the tools necessary to keep the heart connected.

Also, some found that the initial joy of connecting the heart and brain soon diminished. Also, the heart did not stay connected unless the person was consciously telling the heart to connect. Most significantly, most of the PDers were still unable to *feel* their own bodies. By pretending that their minds were connected to their hearts, they became able to activate their visual imagination centers in their brains. They were also able to visualize old injury sites and recognize areas in their bodies that were mentally inaccessible. However, they were still, for the

¹ She was not the first person I had met who was had decided that acupuncture was a cure-all. My students at the acupuncture college often feel the same way. It can sometimes take years before they come to realize that there is a place for all the different types of medicine. The best medicine for a given illness is the one that works: one that reverses the problem that is causing the illness.

A person who is *choosing* to have a closed heart can only reversing his condition by choosing to open his heart. There is no acupuncture needle big enough to change the mental posture of a person who is choosing to be emotionally shut down.

most part, unable to consistently feel the joy and body awareness that triggers dopamine-based movement.

During this time, we also tried other “heart opening” exercises, including the exercises developed by the Heartmath Institute. The PDers who had the hardest time imagining the heart being wholesome-looking and mentally connected to the brain were also unable to do the Heartmath visualizations that involve the heart.

Restoring healthy function to the five senses

As an aside, we were becoming increasingly aware of our partially recovered PDers’ inability to fully experience sensory function. As western doctors have noticed, PDers often have impaired senses of taste and smell. We had noticed years earlier that PDers’ sense of touch was greatly impaired. More recently we were starting to understand that even sound and vision, though usable, were nearly always interpreted by the minds of PDers in a negative way. It was as if all of their sensory function was somehow under the influence of inhibition or negativity.

We’d seen that fixing the foot injury usually restored energy to the nerves around the nose and mouth. This allowed for the return of sensory function of smell and taste in those PDers who had lost them.

But what about hearing and touch? In partially recovered PDers, their hearing, in many cases, seemed to still be stuck on “heightened alertness.” As for touch, many partially recovered PDers would readily admit that their proprioception was poor. And we knew that, during full recovery, PDers noticed profound changes in the way they saw the world. For example, they could suddenly see imaginary images in clouds, they could imagine seeing faces in the leafy shadows of trees. This type of vision had been unavailable to them while they had Parkinson’s, and remained unavailable to many people who were stuck in partial recovery.

To give a point of time reference, this was happening while we were still wondering why the professional musicians with PD had all recovered quickly, in a matter of a few months. There was clearly something going on between the way that a person with Parkinson’s used his sense of hearing and the way he communicated with the rest of their body. We discovered that answer to that story in spring of 2006, when we learned about the proximity of the brain’s frontal lobe area that tracks melody lines and the frontal lobe area that connects to a certain type of nerve signal from the heart. The professional musicians in our program, possibly by dint of constant stimulation of the melody line association area, had essentially forced their hearts to stay open and receptive to *nearly* every aspect of feeling with the exception being their dissociation from their foot injuries. This confirmed our idea that a healthy heart-brain connection was important for full recovery from Parkinson’s. But we had not yet figured out what process the PDers were using to inhibit this connection.

The worst fear: feeling

Lack of feeling and proprioception, we finally realized, were the biggest problems: some people whose bodies could function almost normally, who could taste and smell and use facial muscles and whose rigid bodies had softened and healed still could not really feel their bodies. Using the technique explained in this chapter they could eventually, sometimes after much work and the killing off of their alter egos, imagine their hearts becoming bright and beautiful. They could finally visualize light streaming through their bodies. But very often, they had no sense of what their bodies actually felt like or how their hearts “felt” while filling the body with light and

joy. Sometimes, if their eyes were closed, they still could not be certain where their body parts were located. If they still had bruises or injuries, they could not feel them.

When we asked, now and then, what it *felt* like to have light streaming through previously dark and murky legs, the PDer was most often stunned by the question. “What does it *feel* like?” “Feel?” “What do you mean, *feel*?” We might reply, “Does it feel good? Does it feel bad? Does your foot feel warmer or colder, or do you feel happy to have light in your foot? How do you feel when you do this visualization exercise?” And the PDer would say, “Feel? What do you mean, *feel*?”

A short case study about fear of the word “feel”

One PDer could be utterly relaxed while I did Yin Tui Na on his feet, only to break into a two-minute spate of tremoring every time I conversationally used the word “feel,” “felt,” or “feeling.” When I realized the connection between my words and his intermittent tremor, I did an experiment. Without explaining what I was doing, I started on a seemingly mindless verbal ramble. He was busy making sure that his heart and brain were connected.

I discussed the weather. When I said, “It *feels* to me like it’s going to rain soon,” he started tremoring. Two minutes later he was calm again. A minute or two later, I said, “I don’t eat a lot of eggs, but this morning I *felt* like having eggs for breakfast.” He started tremoring.

After half an hour of this, his wife, watching from the sofa, was trying to restrain her giggles. She saw exactly what I was doing. He never suspected a thing. I kept it up for most of the entire one-hour session. I would be talking gently of this and that, and every four minutes or so I would slip some form of the word “feeling” into a sentence. He would immediately start to tremor. After two minutes the tremor would calm down. I would wait until a few minutes passed, and then I would say a sentence with a form of the word “feeling.” The tremor showed up.

At the end of the hour, he was deeply confused. He said that he usually felt very relaxed after our sessions but that on this day, he had no idea what had been going on for the last hour. He had almost no recall of anything we had said or done.

I then told him what I’d been doing. He didn’t understand why I’d done it. I replied that he was adamant that he had no emotional blockages and that he was extremely sensitive and well-adjusted. I had done the experiment to demonstrate that his tremor was connected somehow with his inability to feel, his inability understand what I was talking about when I asked him questions such as “how does your foot feel when I hold it this way?” After I explained the above, he still had no idea what I was talking about. He asked again, “What do you mean, *feel*?”

Still looking for answers

We had to admit that we had not yet cracked the case. Most partially recovered PDers, even some of the ones who could imagine their hearts being connected to their brains while consciously working at it, were not able to feel their own bodies.

(Given that the heart-brain connection exercise wasn’t the final answer, why did I include so much information about it? Because getting a mental image of the heart turned out to be a necessary first step to turning off the dissociation response, and I want the reader to appreciate that this is not necessarily an easy step. Also, I wanted this chapter to do double duty as instruction in the technique, as well as sharing information about possible pitfalls.)

By now, we were deeply discouraged. We knew that PDers were mentally unable to do mind games or imagine themselves consistently having a *feeling* heart, and yet we couldn't get rid of these inhibitions. What we still didn't realize was that, in many cases, PDers had learned to mentally dissociate from anything that frightened them. In retrospect, we realized that PDers couldn't feel anything in their body because they had dissociated from their entire bodies.

Many PDers had already told us that, when they had received their diagnosis, their dominant thought was a variation on "my body has betrayed me." Betrayal is a horrible thing. When "betrayed" by their bodies, PDers, from years of habit, had dissociated from their traitorous bodies. Later, when we realized that we were working with expanded variations on the selective dissociation response, we could see that the very act of being diagnosed often expands on a PDer's ongoing preference for the dissociation response: even if, prior to the diagnosis, the dissociation had been limited to an injured area and a few mental areas, the entire body became an enemy, or at least a threat, when a person received a diagnosis of Parkinson's disease. The PDer, upon diagnosis, performs his usual method of dealing with his fears: he dissociates from his heart with regard to that particular fear.

And what is the core purpose of the dissociation response? Shutting down one's ability to feel. What happens if a person dissociates from his body? He cannot feel his body. Many PDers protest that they *can* feel their bodies, that they are very sensual or sensitive. They are wrong. We know that most of them have only the crudest sense of body awareness: when they do recover full feeling they are usually stunned that such an extent of proprioceptive self-awareness and feeling is even possible.



CHAPTER THIRTY-TWO

IMAGINATION AND DISSOCIATION

After a year of partially-recovered PDers experiencing only short-term benefits from “connecting the heart and brain,” *if* they could even imagine the heart at all, I grudgingly admitted that this exercise and the dozens of variations that we tried might never give anything but temporary benefit. Partially-recovered PDers seemed to have extreme difficulty in *maintaining* a sustained visual relationship with their hearts.

And worse, as they failed to fully recover, they increasingly manifested, in both frequency and severity, mood-related symptoms of Parkinson’s, which also happened to be symptoms that resembled dissociation: rigidity; difficulty in initiating movement; tremor.

Their constant *physiological* symptoms of idiopathic Parkinson’s might be waning or gone, but in partially-recovered PDers, their susceptibility to symptoms that came and went according to thoughts, moods, or environment – symptoms that looked similar to automatic dissociation – were getting worse.

Symptoms that come and go

Often, the partially-recovered PDers reported, during the weekly session, that they had “forgotten” to have Parkinson’s several times during the previous week. During these times, they “accidentally” had moved perfectly normally. When I asked if they might try to “forget” more often, I was often surprised by the anger with which my suggestion was received. People who, in their own words, “accidentally forgot” to have Parkinson’s once in a while were adamant that these were just mental lapses into a false condition: their TRUE condition was Parkinson’s. It was *impossible* for them to see it from the opposite side: that their periods of dissociation symptoms might be temporary, “false” conditions brought on by some mental incongruity and that their underlying TRUE conditions might verge on good health.

Similarly, during the actual treatment sessions, PD symptoms might come and go. During our treatment sessions, patients could sometimes induce these periods of normalcy. For example, when they were able to *imagine* themselves moving, or in some cases, just imagining themselves as having a heart, they were sometimes able to move perfectly normally. When they could imagine a connected, working heart and imagine themselves moving, they were often stunned at the effortlessness with which they could move arms and legs.

But the imagination and the image of the heart never lasted very long. It was as if their hearts ceased to exist as soon as they stopped focusing on them. When I encouraged them to continue, outside of the session, to visualize themselves moving, they tended to be of the opinion that moving via imagination was a false, short-term construct and they were absolutely not interested in maintaining it. Even though many of them could move *perfectly normally* if they let their limbs move in time with their own imagined images of their limbs moving, they had no interest in practicing this “false” style of movement. And they were not interested whatsoever in the *fact* that normal, correct movement *is* mentally preceded by imagined movements.¹

¹ In healthy people, visualization of motor actions is a crucial part of healthy, dopamine-based, parasympathetic system movement. Although almost no clinical (working with patients) neurologists seem to be aware that the *real* problem behind the movement initiation problems of Parkinson’s is related to PDers’ poor ability to imagine movement, brain scans confirm this finding. In the article “A Dissociation Between Real and Simulated Movements in Parkinson’s Disease,” Frak V, et al, *Neuroreport*, 2004, June 28;15(9):1489-92, a research team

As they kept getting worse, and their symptoms kept turning our minds to what we knew about dissociation, we considered some non-motor aspects of automatic dissociation. In particular, we zoomed in on the heart changes that occur.

Dissociation rule #1: the heart shall be as if dead

A key rule for dissociation, a “prepare to die” condition, seems to be this: *all* aspects of heart function should shut down as much as possible. Heart rate *and* the heart’s role in feeling resonance with sensory experience are *both* minimized while a person is in this condition. When the heart function is minimized, the brain’s ability to freely imagine, including imagining feelings – including imagining the sensations and appearance of movement – is inhibited.¹

reported, “In individuals with idiopathic bilateral Parkinson’s disease motor imagery is impaired and execution of overt movements is spared.” What this means in basic English is that, in PDers, the ability to imagine motor function is impaired: has diminished activity. Activity in this area occurs when dopamine levels in the brain are high enough. Dopamine is a neurotransmitter that is released when a person feels safe or feels glad to be alive. The brain area that controls the ability to actually execute movement (actually move) is *not* impaired: it can work perfectly well *if* it gets electrical signals from the imagery area. The reason that easy, normal movement fails to occur in a PDer is that the PDer did not *imagine* it occurring.

In “Motor Imagery in Parkinson’s Disease: a PET Study,” Cunnington, R, *Movement Disorder*, 2001 Sept;16(5):849-57, the researchers used PET scans to discover the PDers do *not* have any damage to their Supplementary Motor Area (SMA) [the brain area that helps fine-tune motor function], but have inhibition of pre-SMA function [movement imagining function]. In this test, PDers were asked to imagine certain finger movements. The PET scans of the brains suggested that they had difficulty *imagining* repeated finger movements on the more affected side. When imagining the less affected side, the PET scans were able to show that the SMA *proper* had correct function. Again, this means that PDers have healthy function in the brain areas that execute movement. They have deficits in the brain’s imagination areas: the areas that precede the activation of movement.

In the above study, the researchers took PET scans of the SMA (Supplementary Motor Area) portion of PDers’ brains while the PDers tried to imagine moving a finger back and forth at the rate of 1 Hz. The finger used was on the side of their body that was more highly affected with Parkinson’s. In PDers, the SMA area of the brain showed *no* signs of damage. However, the pre-SMA function of PDers is inhibited. In other words, the PDers were not able to *access* the SMA area of the brain. The brain area regulating movement works fine, but PDers cannot conjure up the mental imagery that is necessary to activate the movement execution area.

Another article with the same gist is “Motor Imagery in Normal Subjects and in Asymmetrical Parkinson’s disease: a PET Study,” Thobois S, et al, *Neurology*, 2000 Oct 10;55(7): 996-1002. This article was published in the year 2000 and the introduction to the article states, “Previous work in PD has shown that bradykinesia (slowness of movement) is associated with slowness of motor imagery.” So, even though most clinical neurologists in 2008 have no idea that the imagination is the hang up in PDers’ inability to initiate movement, this is *not* a recent finding.

This article’s research used PET scans to measure activity in the portion of the brain that is activated via imagination of movement. The subjects imagined moving a joystick. The conclusion of article states, “in patients with PD...brain activation during motor imagery is abnormal.

Another study, published in 2007, shows that *extra* areas in the brain are employed when PDers are confronted with a motor imagery task. These extra areas seem to be attempting to increase intellectual processing of visual input, to compensate for the PDer’s inability to visually imagine movement. The study used fMRIs while subjects tried to mentally rotate their own hands in response to an image of a rotating hand. The study concluded, “we infer that, in strongly lateralized PD patients, motor imagery of the most-affected hand exploits additional resources in extrastriate visual areas. These findings characterize the cerebral bases of the increased dependence on visual information processing during the generation of motor plans in PD, pointing to its compensatory role.” Cerebral Compensation During Motor Imagery in Parkinson’s Disease,” Helmich RC et al, *Neurophchologia*, 2007, Jun 11;45(10):2201-15. The findings of this study, in basic English, are that PDers use analytical, problem-solving visual areas of the brain to try to compensate for their inability to use the visual imagination area.

¹ The non-PD reader will recognize that visualization is a *feeling*: it is an operation of the visual *sense*. The inability to imagine the tactile, proprioceptive sensations of movement and the inability to imagine visuals are both forms of the sensory feeling inhibition that is characteristic of partially-recovered PDers.

A person does not have unrestricted access to imagination when he is dissociated. His thoughts must necessarily be negative, and his imaginations are limited to *problems* and analytical problem *solving*. While in this condition, he *cannot* imagine hoped-for events with truly positive outcomes.

Therefore, if dissociation had, over the years, become the dominant pattern in a PDer's brain, any attempts at positive visualization, including imagining having a good looking, hooked up heart, might be difficult or short term.

Oppositely, during the "accidental" moments when the PDer was moving freely, he could evidently *imagine* freely. He was, therefore, not dissociated at these times.

But evidently, even if a PDer managed to temporarily rid himself of the dissociation, he usually soon slid back into dissociation. Very often, he slid back into rigidity or tremor in response mentally noticing that he was doing well – as if doing well was some sort of risk. As soon as he dissociated, he could once again only move in the PD manner: trying to *mentally overpower* his own fear-based compulsion to making him rigid.

Come-and-go movement ability suggested come-and-go dissociation ability. But how could that be?

Movin' easily when it's safe

Partially-recovered PDers tended to "accidentally" move easily or "forget" to have Parkinson's during one or several narrowly defined situations that, through some quirk, they had *subconsciously* deemed safe. Anything that wasn't "safe" had subconsciously been labeled "not safe." There was no neutral ground.

I've already mentioned examples of safe activities and safe times. Some more examples of these "safe times" show the range and seeming arbitrariness of individuals' "safe" conditions: watching an opera; dealing with public safety issues (as opposed to personal safety issues);

Telling research on this subject is included in *The Boy Who Was Raised As A Dog And Other Stories From A Child Psychiatrist's Notebook*, by Bruce Perry. He recounts how a young girl who taught herself to dissociate during her once-a-week rapes by her step-father found that she could go numb by dissociating and going "outside" of her body to an imaginary place. However, she was unable to create the visual mental fantasies that she *wanted* during her dissociation episodes.

During dissociation, her imagination's color choices were limited, as were her options for events that took place in her imagination. She could exercise some degree of control over the events that transpired in the imaginary land that she went to during the rapes, but she could not imagine *good* scenarios or *happy* outcomes. In her dissociation imagination, she could only imagine dark images consistent with being in sympathetic (danger) mode: dangerous, ugly situations that she could resolve using force and power. Her dissociative imagination was severely limited in other ways, as well. For example, during her dissociative events, she wanted to imagine herself as a bluebird but beautiful blue colors were not available to her mind. She could only be a black bird: a powerful, frightening black bird.

If asked, she might have said that she *was* able to imagine. I would argue that she had a limited ability to imagine, and that *full* reign over her imagination feelings was not possible while she was dissociating: in the dissociated condition, she was numb to positive outcome or beauty – just like most of the partially-recovered PDers would be numb to what it might feel like if they recovered.

Many PDers told me that they intellectually or even intuitively *believed* that they could recover. However, they could only *imagine* their PD symptoms worsening. They were *unable to imagine* that they might be recovering. And when they "accidentally forgot" about Parkinson's and moved normally, they could not imagine that this had been a good sign – after they re-dissociated. At the time of easy movement, they might feel certain that they were getting better. But as soon as the good phase passed, they then imagined, every time, that the brief period of ease of movement had certainly been the very last time that they would ever move easily – no matter how many times it happened.

washing dishes; performing in theatricals; making love; after eight forty-five p.m.; and so on. I have already sprinkled other examples throughout this book. And what was safe for one PDer was not safe for another. Some PDers were *more* anxious than usual during the evening hours or while attempting to make love, or while performing. The safety zone was a seemingly random, highly personalized choice.

Safe in the evening

Quite a few PDers' tremors ceased after some pre-determined hour in the evening. The actual time varied from one person to another. They tried to explain to me that this was perfectly normal because, after their specific time arrived, they were "done for the day," or they had "finished everything that was supposed to get done that day." The implication was that the rest of the evening was free time – so there was no need to tremor.

No amount of logic could convince them that, if they applied this "free time" attitude to the entire day, they wouldn't need to tremor at all. They dismissed my idea with explanations that were always pretty much the same: "I *do* have Parkinson's. Why should I pretend I don't? If I stop having symptoms when my day is over, that's because I'm not *really* doing anything any more. As long as I'm actually *doing* something that *needs* to be done, I do have Parkinson's. It would be futile and it would be a lie to pretend that I don't have PD.

It would be years before I realized that I could substitute the words "at risk" for their words "doing anything." So long as these people were "having to do something" that was "important" or "real," they behaved as if partially dissociated. My eventual translation of these words became "so long as they thought they were *at risk* of criticism or possible error, or at risk of *anything* that might lead to the slightest physical or emotional discomfort, they dissociated."

No heart: no movement; no heart: tremor

But I did see, based on our experiments, that partially-recovered PDers were not going to be able to sustain a healthy mental connection with their hearts so long as they were afraid of or not interested in having their hearts. And if they didn't have a heart, they couldn't *feel* their hearts. The heart performs the core processing of sensory perception, or feeling. The ability to feel safe is necessary for imagining the feeling of movement. If they couldn't imagine movement, they couldn't move. No heart connection = no movement

Also, when the heart doesn't perform its job of sending sensory information to the brain, the brain has to assume that the heart is struggling to come out of a dissociative state or a state of shock. The brain therefore institutes tremors, as it typically does when, following a shock, the body's autonomic nervous system doesn't receive the full complement of neural exchanges between the brain and the heart. In an otherwise healthy person recovering from shock, tremor can be helpful in restarting the heart: the involuntary tremor movement makes the person slightly more alert, causing him to take a deep breath, feel the inspiration of life pouring back into his chest, *feel* the "good to be alive" sensation, *feel* safe, and then, because of that feeling, *wham*: he snaps out of his heart numbness, the heart communicates a full message to the brain, the brain communicates with the heart, and voila! The autonomic (automatic) heart-lung-blood pressure regulatory system is back on line and running correctly. No more tremor. But as long as there is no heart connection, a person can be prone to tremor.

This means that tremor can have two sources: the brain shift that occurs in response to backwards flowing Qi from the foot injury, and from selective dissociation from the heart.

As noted earlier in the book, we came to realize that many PDers actually had two forms of Parkinson's: injury-induced idiopathic Parkinson's *and* psychogenic parkinsonism that derives from dissociation from the heart.

We'd seen people recover simply by having their foot treated. But many PDers also needed to re-establish a lasting connection with their hearts if they hoped to recover.

As these realizations broke over our heads, I started to understand the answer to my prayer: "She has to do it herself."

Basically, my heart had told me that, if a person has shut out God (heart resonance), God will not override that person's decision to be apart from God. God, or Love or Wisdom, if you prefer, is a perfect respecter of free will. If a person is making a decision to be apart from God, God will honor that.

In other words, if a person was selectively dissociating from his own heart, God wasn't going to step on his toes and force him to feel heart resonance. If a person wants to stop being dissociated from his own heart, he has to do it by changing his mind and deciding to have a heart again.¹

We were able to understand, finally, that there was no needle treatment or herb that could change the mind of a person who had decided to selectively dissociate from his heart. So long as he kept making that decision, we couldn't help him. But on the other hand, when we realized that our patients were selectively (mentally) dissociating, a pathology that was well known and well studied, we were relieved, in a way. At least our partially-recovered patients had a *recognized* mental problem: dissociation. They had a problem with a *name*.²

¹ I tried to research these two uses of the word dissociate to learn which came first. I was not able to discover the dates. But I did learn that another, more literary usage dates back to the 1700s. This meaning of dissociate also means to "be apart from," but it refers more to dissociation from one's customary social group or country.

I came across an essay from the 1930s in which a yogi used the word dissociate with this "social" meaning when he advises people to dissociate from their pain. The rest of the text makes it clear that he is not advocating numbness, but rather a lack of self-identification with the pain.

A person might be identified by the society (associations) he keeps. If he ceases to belong to those societies, he will no longer be identified by them. He will have "dissociated" from them. The yogi was using this word in this "social" manner. What he meant was, if you find yourself succumbing to pain and imagining that *you* are pained, stop and remember that you are the immortal soul. Your soul is not in pain – only your body. "Dissociate from the pain" means, don't identify with the body; do not imagine that your true identity is pained. When you remember what you really are, you will no longer identify with the pain. You will then be able to use your soul's energy to focus on the painful place, calm the erratic energy in the area of the pain, neutralize the nerves signals so that they convey mere sensation instead of fear and pain, and then mentally process the experience and instruct the body to institute any necessary healing mechanisms. This is what a great spiritual teacher means when he says "dissociate from the pain." He does *not* mean "make yourself numb" (automatic dissociation) or pretend it never happened" (selective dissociation).

² When I say "mental" (meaning "not structural or chemical") I have to note that the brain is extremely plastic and able to undergo physical change in response to thoughts. Psychological behaviors *can* cause brain change, which means that PDers' brains *can* be structurally affected by their pathological thinking patterns. For example, people with purely psychogenic parkinsonism (parkinsonism that starts in response to a terrible trauma and which goes away after the person processes the trauma) may still produce brain scans that show decreased dopamine-receptor activity – a temporary *physical* brain change – while they are in the depths of their parkinsonism.

The brain is extremely plastic: it changes in response to usage. Because many people were taught in school that the brain serves as an unchanging switchboard, and that brain cells don't change or grow, I will make this an extended footnote.

THE TREATABILITY OF SELECTIVE DISSOCIATION

I wrote earlier that people can recover from selective dissociation. I don't want to create false hopes that treating dissociative disorders is easy, or to imply that there is a standardized protocol for treating these disorders. In the most severe form of dissociation, multiple personality disorder, there is *no* consensus as to whether this condition is actually treatable. Some psychiatrists say yes, others claim that any improvements are due to coping mechanisms. As I searched for success rates for treatment of dissociation from specific body parts such as the foot or the heart, I found *nothing* in the readily available literature. There *is* an enormous amount of literature addressing the psychological pathologies that can arise from sexual abuse, which can include dissociation from the abuse *memories*.

As I studied hundreds of these cases and their treatments, I became increasingly certain that treatments used in these cases of sexual or other forms of abuse would *not* be helpful for my PDers. Based on thousands of hours of psychological work with PDers, I knew what most of them could and couldn't do in terms of mental exercises. I was digging into the materials about selective dissociation in 2008. By that time, I'd already spent nearly a decade doing psychological therapies with hundreds of PDers, and I had a very good sense of what they could and couldn't do. They could *not* do the treatments suggested for dissociation from events.

As an example of the brain's plasticity, studies show that the language portion of a bilingual's brain is much larger than the language area in the brain of a person who only knows one language. Not only is the grey matter (the portion that "knows" the words) larger, the white matter (which links words to other areas of the brain) is *also* larger in bilinguals and trilinguals. This is an example of a brain area increasing in size in response to use.

The brain also *declines* in response to *non-use*. An excellent study used juggling to demonstrate that both brain growth and brain decline occur in response to activity or non-activity, respectively. In this study, healthy volunteers had brain scans (MRIs) before being taught how to juggle. After much daily practice, they were able to keep three balls in the air for at least sixty seconds. A second round of MRIs at this point showed their brain's gray matter had increased in those areas related to this skill (in the temporal lobes of both hemispheres and the parietal lobes of the left hemisphere). Three months after of stopping the juggling, a third MRI showed that "the gray matter gains were reduced." (Both the bilingual study and the juggling study are written up in *The Wisdom Paradox*, by Elkhonon Goldberg, PhD, p. 255. The juggling study was cited as coming from "Neuroplasticity: changes in gray matter induced by training"; Draganski, B., Gaser, C., Bush, U., Schuier, G. Bogdahn, U., & May, A. *Nature* (2004), 427 (6972), 311-312.)

For some light, fascinating reading on the subject of brain plasticity, I recommend *The Brain That Changes Itself*, by Norman Doidge, MD, and *Mozart's Brain And The Fighter Pilot*, and *The New Brain*, both by Richard Restak, MD.

My point here is that the brain is plastic. So when I say that a problem is psychological instead of physiological, the reader has to understand that changes in thought patterns, mental behaviors, and physical behaviors will *create* physiological brain changes. In the case of PDers, psychological habits can affect dopamine production and dopamine receptor activity. However, these dopamine-decreasing changes do *not cause* a PDer's dopamine inhibition – these changes come about *because* of a PDer's constant dopamine inhibition. This inhibition can be both electrical *and* mental. I'll be redundant (again): *all* PDers with the "classic" look of idiopathic Parkinson's (asymmetric skin and muscle changes along the Stomach channel) have the electrical (physically caused) inhibition of certain brain functions that is a correct "shut down the body so healing can occur – as soon as the injury is actually recognized," result of a severe injury. This brain shift will cease when the injury heals. In addition to this electrical shift, *some* PDers and people with psychogenic parkinsonism have, to varying degrees, *mental* inhibitions (selective dissociations) that shut down the heart's ability to feel and thus inhibit dopamine release.

Based on modern understanding of the brain's plasticity, we hypothesize that both of these types of inhibition will, over time, cause measurable decrease in certain brain structures and functionality. Healing the injury *and* getting rid of any mental dopamine inhibitors *plus* experiencing the feelings that release dopamine will cause an *increase* in dopamine-related brain structures and functionality.

For example, treatments for dissociation from sexual abuse often involved creating a “safe, non-abuse” zone where they could have their usual, normal full range of feelings, and then teaching the person to expand that safe zone. This process would not work with a person who felt safest when numb or who didn’t know what the word feeling even meant. These treatments required people to modify the settings that triggered explosions of painful feelings.

With regard to painful experiences, my patients tended to not actually *have* feelings.

Dissociation from feeling as opposed to dissociation from an event

An abused person tends to dissociate from specific *experiences* so that he won’t have to feel his violent physical and emotional reactions to the event. Sexual abuse survivors were still very much able to feel, enjoyed feeling, and were constantly traumatized by their inability to stop their revisitations of painful feeling. They dissociated from painful *events* so that they could continue to have feeling for all other facets of life. Their problem was that the brain made linkages to other events – keeping them perpetually traumatized.

PDers dissociate from their ability to feel, period. The event may or may not be forgotten, and it may or may not be considered a problem. The problem, for many PDers, is *not* that a painful event occurred. The problem for many PDers was that they felt at risk of *feeling* a strong response.

PDers don’t necessarily dissociate from the memory of terrible *events*. Some do, but some don’t. However, all of our partially recovered PDers had, to some extent, dissociated from the ability to *feel* the experience. The negative *experience* wasn’t the problem; the problem was the ability to *feel* a physical or emotional response.

PDers were dissociating from feeling, period. When a person dissociates from his ability to feel his own physical or emotional pain, he does this by dissociating from the signals that are generated in his chest, in his own heart. Whether or not he consciously thought about not having a heart, a cessation in heart sensory function is the mechanism employed when a person decides that he is impervious to pain.

We now recognize that most, if not all, of our partially recovered PDers had dealt with unpleasant events by dissociating from their ability to feel. Many recalled doing this consciously, at some point in childhood, in response to an emotionally or physically painful situation. For most of them, they were now able to do it subconsciously, when any situation arose that might potentially result in criticism, error, or anything that could cause physical or emotional pain.

As I read books on treating selective dissociations, I knew that PDers would not be able to respond to the treatments that were being used. We’d actually tried some of the techniques already. We were going to have to come up with a treatment technique specific to the problem of fearing *feeling*.

Dissociating from the pain of diagnosis

When we figured out a way to treat this problem, we discovered that one of the most common emotional pains that actually needed to be specifically treated (as opposed to the pains that could be treated in batches, by focusing on generalities) was the pain of receiving the diagnosis of Parkinson’s. We saw an enormous beneficial shift in PDers when they were able to

correctly neutralize the pain of having been diagnosed with Parkinson's disease. This helped us answer a puzzling question.

You may recall reading in chapter xxx that the people who didn't have a diagnosis of PD tended to recover quickly. They didn't slide into partial recovery.¹

In our limited experience, the people who had not yet received a diagnosis of Parkinson's had only dissociated from the ability to feel their foot pain. But it became evident, in the last stages of our project, that people with a diagnosis of Parkinson's had dissociated from their ability to feel the emotional pain of bad news. This turned out to be a much harder dissociation to treat: it seemed as if it linked up to other emotional pains. When we starting working on the pain of Diagnosis of Parkinson's disease, it often brought forth other old, buried issues, as well. These other issues in and of themselves might not have been strong enough to create the symptoms of Parkinson's disease. But joined by the enormous pain of diagnosis, these various dissociated emotional pains seemed to assume the power of a small cabal, a gang that was, on top of everything else, highly susceptible to negative suggestions (the placebo effect).

After we learned how to teach people the necessary steps in re-associating with their hearts in general, the dissociation from the pain of diagnosis very often needed to be specifically addressed.²

We finally had a hypothesis that answered all our questions about partial recovery.

Next, we had two jobs. First, we had to *prove* that our hypothesis was valid. This proof would have two parts: we had to prove that PDers were, in fact, in a state of dissociation *and* we had to prove that they had been able to put themselves there by simply telling themselves not to feel.

¹ The fact that *none* of the twenty-five people who had PD symptoms but no official diagnosis were dissociated from their hearts is probably due to our small sample size. We have many PD patients who have told us, during or after recovery, that they had definitely shut down their hearts in childhood, and had never felt much physical or emotional pain ever since. Had these people been in the "no-diagnosis group," they may have experienced a brief respite from their symptoms when their feet healed and then, over time, continued to manifest symptoms of psychogenic parkinsonism. This would have altered our statistics, so that we would only be able to say *most* people who have not yet received a diagnosis recover very quickly.

A much larger sample size, involving thousands of PDers, might yield more variables, as opposed to the 100 percent finding of our small sampling.

² The PDer reading this section may well say to himself, "I don't remember feeling any pain when I was diagnosed." My reply would be, "My point exactly." Not all PDers are the same, of course. Some did recall feeling horribly betrayed by the body at that moment of diagnostic horror – but they didn't necessarily feel physical pain. Others were certain that they hadn't felt anything at all. An emotionally healthy person might respond to a terrible diagnosis of "incurable illness" by feeling as if he'd been kicked in the solar plexus, or feeling a knot in his throat, or feeling a knot forming in his stomach: some sort of physical pain.

When we developed a simple, safe, and pleasant method for processing these dissociated emotional pains and focused this method at the pain, gently reawakened, that *had* in fact occurred at the time of diagnosis, PDers have been able to feel the physical sensations of the emotional pain that actually occurred at the time. These pains were not anything special or rare. They were usually things like a lump in the throat, a gnashing or sinking feeling in the gut, a twist in the stomach, shortness of breath, etc: the usual responses to emotional pain. These are perfectly normal pains and can be processed in the normal way *but* only if a person knows that they exist. If a PDer had averted these sensations away from his conscious mind, then he had never felt them. But they were still sitting there in the body, unfelt, and they were also still sitting in some dark cubby-hole of the mind, sending fear signals that encouraged the PDer to keep making use of his skill in dissociating.

Second, we needed to come up with a treatment. We had to figure out a way to teach our PDers how to consciously induce *feeling* at will. After teaching them to feel, we had to teach them how to feel and heal from their buried pains without being overwhelmed or frightened by them.

And then we realized that we had already done the first job. One of our many experiments, “A Day at the Beach” had proved that partially recovered PDers were in a state of dissociation. Another experiment, the Heart-in-a-Box technique, had yielded proof that PDers had, in fact, induced this state intentionally, by deciding to be impervious to feeling.



Riddle: Pretend you're trapped in a steel box with no doors or windows, no way in or out, and no tools to cut the steel: how do you get out?"

Answer: Stop pretending!

- as told by a nine year-old neighbor

CHAPTER THIRTY-THREE

TWO PROOFS

PROOF #1: A DAY AT THE BEACH

The mystery of the toe spasms

Toe and/or foot spasms and toe and/or foot dystonias (long-lasting, even semi-permanent, spasms) are not uncommon symptoms in medicated and unmedicated Parkinson's disease. Researchers have also determined that the dystonias and dyskinesias (uncontrolled movements) of Parkinson's that are brought on by excess medication most often develop *first* in the feet and toes.¹

We noticed that many partially-recovered PDers continued to have problems with toe spasms, toe curling, and other toe-related problems even after their feet injuries had healed. These toe problems had various triggers. For example, in some PDers, the toe problems worsened in response to a short walk. For others, the toe problems *diminished* in response to a short walk.

One partially-recovered PDer thought that wearing boots was the cause of her toe cramping. She started wearing only loose shoes and her feet felt better for a while. Within a few months her feet cramped up when she wore loose shoes. She started wearing only sandals. Again, she got a few months relief but the foot cramping started up again a few months later – if she even *thought* about footwear.

That's when we all realized that the problem was not just physical: something psychosomatic was going on with her and possibly with the other people with foot and toe cramps. After all, once the foot cramping habit started, it got worse – and it usually got worse in exactly the way the PDer expected it to. And if a PDer “accidentally forgot” to have foot cramping during his “normal” foot cramp situation or time of day, the foot cramping did *not* occur.

For example, one person got toe cramps if he walked exactly ten minutes or more *but* on the day when a visiting friend went with him for a thirty-minute walk, he “forgot” (his own word) to have toe cramps.

It seemed to us that, *if* the foot cramping was coming from a fixed, physical infirmity or a fixed neurotransmitter shortage, it should not come and go in response to expectation. But even if the trigger was psychological, the mechanism might be detectable. We wondered about the

¹ Vidailhet M et al, “Do parkinsonian symptoms and levodopa-induced dyskinesias start in the foot?”, *Neurology*, 1994 Sep;44(9):1613-6. Winkler AS et al, of King's College and St. Thomas' Medical School, “The frequency and significance of striatal toe' in parkinsonism, *Parkinsonism Related Disorders*, 2002 Dec;9(2):97-101. Harris ED, “Pseudorheumatoid deformity of the feet associated with parkinsonism,” *Journal of Rheumatology*, 1987 Aug; 14(4):855-6. Uhrin Z, “Rheumatoid-like deformities in Parkinson's disease,” *Journal of Rheumatology*, 1998 Jan 25(1):177-9.

physical mechanism that the body was using to generate this fairly common, painful, mind-related, come-and-go problem.

An experiment

One day, while describing to me in great detail her toe cramp problems, really getting into it, a partially-recovered PDer experienced a foot cramp while she was in my office. This was a rare opportunity. I used my hands to feel the Qi flow pattern in her foot while the cramping was occurring. The normal Qi flow pattern was not happening.

I was puzzled. Usually, when I treated her, the Qi was flowing correctly in her foot. Her foot injury had been treated and had responded to treatment. Energy was no longer flowing backwards in her leg. Since the foot had healed, whenever I held her foot briefly and then checked to see how the Qi flow was doing in her leg, the Qi flowed perfectly. But right now, with her foot cramping up, the Qi was *not* moving correctly.

The Qi of the Stomach channel was moving strangely in her leg, almost as if it was moving back and forth. Stranger still, at ST-42, at the center of the *top* of the foot, the Qi was shunting into KI-1, at the center of the *sole* of the foot – and vibrating back and forth between top and bottom of the foot. To my hands, the vibrating current in the center of the foot seemed to be giving off static even though it was almost standing still. It felt as if an electrical charge was moving back and forth very quickly between ST-42 and KI-1, as if it were trapped in the center of the foot, unable to flow.

I felt the ST channel current higher up on the leg. It too was moving in a strange, “standing still” manner, sort of vibrating back and forth but not actually going much of anywhere.

It seemed to me that the electrical pull of the “standing Qi” between ST-42 and KI-1 might be strong enough to pull the toes in towards to center of the foot.

I puzzled over this for days. I recalled to mind the image of a mouse that dissociates after being clawed or nipped by a cat. I could picture the mouse’s toes and feet curling tightly inward as the mouse lies rigid, as if dead. I wondered if dissociation might cause this alteration in Qi flow.¹

¹ Beginning acupuncture students are taught only one pattern of Qi flow for each channel. This idealized pathway represents the Qi flow pattern when a person is awake and in parasympathetic (relaxed) mode. Dr. Ju-Yi Wang, a National Treasure of China and a Master of channel theory, told my doctoral degree class that this ideal situation only occurs seventy percent of the time, *at best*, in a human. When illness or pathology is present, the odds of Qi flowing in the ideal pathways decreases further. As it says in the *Nei Jing* (221 BC), a pre-eminent classic of Chinese medicine, “Life exists by virtue of the twelve channels. The beginner thinks it [channel theory] is easy. The master knows how difficult it is.” One reason for the difficulty in mastering channel theory, which is to say, using channel flow to diagnose and treat illness, is that the flow patterns of the channels shift quickly in response to environment, thought, injury, or illness. Another, more modern, reason for difficulty in mastering channel theory is that channels have officially been discarded by the Chinese government.

During the latter half of the twentieth century, Dr. Ju-Yi Wang had kept pretty quiet about his ideas on channel theory, even while using channel findings to make diagnoses. The political climate in China has changed a lot since then: when I talked to him (via a translator) in 2008 he was considered a master even though his specialty was channel theory. I asked him if the current government was becoming more supportive of channel theory. He replied (via translator) that in the past, focusing on channels was wrong, and then, later, it was merely suspect. But more recently, it was tolerated because nobody really cared anymore. He explained further by saying that, nowadays, if I wanted to continue my studies in channel theory at any medical school in China, I would *not* be able to find a sponsor who would be interested in anything having to do with channel theory, but on the other hand, no one would be sent to prison camp for having talked with me about it.

I devised an experiment to simulate dissociation so that I could explore channel Qi changes, if any, during this fairly rare neural mode. I named the experiment “A day at the beach.”

The dozen subjects included healthy people and partially-recovered PDers.

A Day at the Beach: the technique

Part one: “Imagine that you are lying on a sunny beach, and the air is warm and silky. The air feels so wonderful that you focus on feeling the air with your skin. You are almost reaching out with your skin to feel the air.”

The subject would lie still, imagining this for a moment, while I felt the Qi flow in the Stomach channel. The Qi in healthy people and in *some* partially-recovered PDers flowed correctly: the Stomach channel Qi got to ST-42 and then bifurcated, with some Qi moving to the toes and a small amount of Qi flowing over to SP-3, on the medial side of the foot. On the torso, Stomach channel Qi flowed down from the clavicle and over the breast, in the classic pattern. This matched the “correct” pattern of Qi flow that is taught in the schools. This Qi flow pattern occurs when a person is in parasympathetic mode. In this mode, a person has the maximum ability to experience to the fullest the five senses. Also in this mode, steady energy flow to the stomach helps drive the gastrointestinal tract and the digestion of food.

But some of the partially-recovered PDers, people whose foot injuries were healed, told me that they couldn’t imagine feeling the warm air. A few even said “I don’t know how to do that.” The Qi in their Stomach channels seemed to be standing still.¹

The Stomach channel Qi flow in PDers whose feet injuries were not yet healed flowed backwards, as always – uninfluenced by the instruction.²

Part two: “As you are lying there on the beach, a tiger suddenly jumps out from the tall bushes bordering the sand. He sinks his teeth into your chest and starts pulling your arm off. Feel the sensations of having his teeth sink into your chest. You are starting to bleed heavily. You may die.”

In response to this mental image, the Stomach channel Qi flow in all subjects was the same: the Qi ceased flowing to the toes. It performed the ST-42 to KI-1 standing wave pattern through the center of the foot. The Qi in the leg was barely perceptible, but what there was of it

But my point is, the amounts and pathways of channel Qi vary. The pathways that we learn in school are not fixed, rigid roads. They are paths of least resistance, and they are influenced by, and resistances can be formed by, internal and external thoughts and events.

¹ In these partially-recovered PDers, Qi in the feet *could* flow correctly if they were relaxed. I suspect that the fear and tension induced by trying, unsuccessfully, to imagine something pleasant caused them to shift into dissociative mode.

² Prior to healing the foot injury, some PDers’ Stomach channel Qi is undetectable in the foot or lower leg, as if there is no Qi at all. In order to feel the backwards-flowing Qi of the Stomach channel on these patients, the practitioner must sometimes feel as far up the leg as the knee before he can detect the Stomach channel – which is running backwards.

was more “standing still” (going back and forth) than flowing down towards the feet. On the chest, the Stomach channel Qi flowing down from the clavicle disappeared down into the chest as it approached the third rib. When it did emerge again, below the waist, it was only present in the “standing still” pattern. The perverted pattern of Qi flow on the feet was strong enough that in a few subjects the toes pulled in gently, in a manner similar to that of the toe cramping in PDers.

This pattern conformed with the fact that, during dissociation, blood and energy shunts deep inside the body. The skin becomes cold. The limbs become lifeless and the toes may curl in.

After a few moments of this, the instruction continues: “Aha! It was only a dream. There was no tiger. You are not hurt. You are fine. You are still lying on the beach, enjoying the warm air. You want to put your energy into your skin, reaching out with your skin to feel the glorious warm air blowing gently over your body, even over your feet.”

Within seconds, I could feel that the Qi had resumed the pattern that it had followed in Part one.

Part three: “As you gaze over at your little sixteen month old niece playing happily in the sand, you suddenly see an evil-looking man sneaking up on her with a baseball bat. He looks deranged. You realize that he is going to whack her with the bat. You have to jump up and save her!”

Then, I quickly felt the feet and legs. In most of the subjects, Qi was pouring down the Stomach channel and coursing over to SP-3. No Qi was going to the toes: it was all gushing into SP-3. The Qi in the Stomach channel of the chest was not flowing over the breast. It dropped down into the heart at around the third rib and re-emerged just below the waist, flowing with vigor.

Many of the subjects even volunteered that they could feel the soles of the feet getting warm or throbbing with energy.

This pattern corresponds with what happens during a “call for action” sympathetic system response. During sympathetic mode, energy flow to the heart and the skeletal muscles increases. Energy surges into the feet or whatever muscles are going to be called on for action. At the same time, flow to the stomach decreases. Channel Qi (electrical energy) that would normally go to the gastrointestinal tract is diverted to the heart.

I performed the experiment many times. Sometimes I asked the subject to describe what their feet and legs felt like during the experiment. Except for the partially recovered PDers, many of whom did not experience any changes – and whose legs and feet remained in a dissociative mode, throughout – most subjects noticed a weird absence of feeling in the legs during the “death by tiger” (dissociation) phase, an increase in energy on the sole of the foot during the baseball bat (sympathetic) phase, and a feeling of relaxation in the chest during the warm air on the skin (parasympathetic) phase.

This experiment showed several things. It showed that the body could assume the Qi flow patterns consistent with certain neurological modes merely by *imagining* evocative conditions. Transition into the Qi flow patterns of sympathetic, parasympathetic, or dissociative mode could

be *mentally induced*. The changes took place in a second or less: as quickly as thought. The speed of thought seemed to be the determinant for the speed of conversion from one phase to another. No chemical or fluid transfer time is necessary for electromagnetic brain waves to influence electrical currents.

The experiment also offered a mechanism for the toe cramping: the column of standing Qi in the center of the foot very possibly created an electromagnetic pull on surrounding tissues. In the presence of that electromagnetic standing column, foot tissues were pulled towards to the center of the foot. Painful hammer toes or toes and feet curling under and in towards the center of the foot can be normal symptoms of automatic dissociation. This condition is very different from the limpness in the toes and feet that occurs moments after actual death, when the Qi truly ceases to flow.

We had already seen that, when a partially-recovered PDer engaged in fear-based thinking including thinking about risk, pain, or worsening of his symptoms, his toe cramping – if he had any – might be triggered or worsened. Even subconscious thoughts appeared to play a role.

For an example of subconscious thoughts triggering toe spasms, consider the childhood of the person whose feet started to curl whenever she thought about shoes. Her extremely self-centered mother, who had never hid her dislike and disdain for her children, had a collection of hundreds of pairs of shoes, many never worn. The PDer remembered thinking bitterly to herself as a child, “A mother should love her children more than her shoes.” Forty-five years later, the pressure of shoes on the feet and then, eventually, as her brain grew more negative links and habits, the mere *thought* of shoes was able to induce a subconscious negative association – and toe spasms. It got so that when she even *thought* about shoes, her mind immediately performed its usual trick for dealing with negativity – a selective dissociation response in which she dissociated from her ability to feel. The selective dissociation from her ability to feel would then trigger various symptoms of automatic dissociation – including, in her case, toe curling and tremor.

This experiment showed other things, as well. It showed, yet again, that some of our patients who were stuck in partial recovery had no ability to imagine various feelings. No matter which part of the experiment they were in, their leg Qi flowed as if they were dissociated – barely moving, with a faint sensation as if the energy was vibrating back and forth. Again, these people could experience correct Qi flow if they were relaxed. That’s a prerequisite for being considered partially recovered. But evidently, when I asked them to imagine the positive sensation of warm air on the skin, the discomfort of this unpleasant task or maybe the inability to perform the task of positive imagination induced enough stress that their Qi flow reverted to dissociation during the course of the exercise.

In patients who were just starting treatment who still had an obstruction in the foot, the leg Qi flowed backwards, in an injury pattern, regardless of the mental imagery. The Qi flow pattern in these people was unaffected by the experiment.

My mistake

For years, I had assumed that, since Qi *could* once again run correctly in PDer’s feet, it would always run correctly: it would run in the parasympathetic pattern.

I had been wrong. Partially-recovered patients evidently had nice, correct parasympathetic Qi flow patterns when I held their feet or when they felt relaxed, but it quickly shifted into a dissociative pattern in response to a negative thought. They might have Qi flowing normally in my office, but when they went about their daily routine, their minds shifted in and out of selective dissociations. This, in turn, meant that their bodies shifted in and out of Qi flow patterns that corresponded to automatic dissociation.

Evidently, for some of them, the shift into dissociative mode caused a strong enough channel Qi shift into automatic dissociation mode that they experienced toe and foot cramping.

Tiger bites feel normal

Many partially-recovered PDerS were unable to *imagine* a good feeling, such as enjoying the feeling of sunlight on the skin. The people who could not imagine this tended to get into a dissociative pattern and stay there while they were instructed to imagine feel-good scenarios. Many of them did not want to imagine feeling the skin, and told me so. They had trouble imagining and wanting to imagine warm air on the feet.

However, true to their pattern of being able to imagine bad scenarios but not good ones, *all* the partially recovered PDerS were easily able to imagine the death-by-tiger scenario.

And here's the nub: alarmingly, some of them volunteered that, in terms of energy and emotion, they felt *normal* when I suddenly told them that they were being attacked by a tiger. This statement startled me so much the first time I heard it, that I paused in the middle of the experiment to ask questions. The replies suggested that the person felt in his comfort zone, he felt *normal*, when the tiger was killing him! Death-by-tiger automatically invoked in him a familiar sense of overall numbness and readiness to die, and this felt *safe*. Statements to this effect were offered by *several* of the subjects who were stuck in partial recovery.

As for the sympathetic system response, some of the partially-recovered PDerS felt confused by the baseball bat situation, uncertain as to what they were "supposed" to do or what they were supposed to feel.¹

¹ Regrettably, I did not have any patients at this time who were professional musicians with untreated Parkinson's. I suspect that such a person would not have the same imagination inhibition as a more repressed PDer. If a PDer in the experiment were a professional musician and therefore had maintained a brain link to positive imagination, he would probably have some portion of a correct sympathetic system response during the imaginary baseball bat part.

The reader may protest that PDerS are supposed to be able to move normally during a real emergency. He may wonder why the PDerS in this experiment weren't able to generate a sympathetic Qi flow pattern. I suspect that the reason my unhealed PDerS were not able to manifest sympathetic system Qi flow during the experiment is that they were unable to truly imagine themselves in the situation. They might be able to move normally in a *real* emergency but they were unable to invoke a healthy response to an *imaginary* emergency.

Our musician patients, and other PDerS who recovered easily, seemed to have *only* dissociated from their injured feet. They had *not* dissociated from their overall ability to feel pain or pleasure or the linked ability to control the imagination. For example, they were still able to feel strongly – and frequently – the thrill of the swelling sensation in the chest that occurs in response to a situation of great beauty or grace.

At this point, if the reader is in a hurry, he can go back to the text. But is he doesn't object to veering off on a steep tangent, he can stay with me while I point out that Paramahansa Yogananda affirms that perfect concentration and pure feeling are associated with the ability to control the imagination. Yogananda makes the point in one of his lectures that a great spiritual master such as Jesus can concentrate his imagination on the idea of an apple and have the apple appear, spontaneously, in his hand. A Master's supreme ability to concentrate is connected to his ability to maintain an ego that is absolutely unruffled by external and internal stimuli while *feeling* (as

Proof

This experiment had shown that people could instantly trigger the electrical flow patterns that occur during automatic dissociation merely by *thinking* about a life-ending situation. Electrical instructions to the body (channel flow patterns) could rapidly come and go based on thought patterns.

When we came to realize that partially-recovered PDers were selectively dissociating, and that their negative thoughts could come and go, we recognized that the shift in channel Qi that instantly accompanies a change in thought might be the mechanism behind the come and go symptoms of rigidity and tremor.

We originally conducted this experiment merely to help understand the mechanics of the toe cramping. But when we hypothesized that selective dissociation might be the mechanism for triggering the on and off symptoms of automatic dissociation in partially recovered PDers and needed to prove that a rigidity-inducing shift in channel Qi could be induced by thought, this experiment provided that proof.

Additional proofs

I had to wonder about the strange statements from many partially-recovered PDers about the *normalcy* of “going numb” and the inability to imagine a positive scenario of feeling sunlight on the legs and feet. Their verbal responses to this experiment indicated that sliding into a dissociative mode felt normal, safe, and *good*. I will repeat this: people who became stuck in partial recovery derived a feeling of safety by mentally inducing, to varying degrees, the pre-death, somewhat rigid state of automatic dissociation.

opposed to thinking about) all states of energy (internal and external stimuli) to the fullest – a characteristic of supreme parasympathetic mode.

My work suggests that the opposite is also true: when a person becomes unable to access the parasympathetic mode, he loses the ability to be the master of his own imagination. He may be able to concentrate, but as his contact with his parasympathetic system wanes, his imaginations will be increasingly limited to sympathetic system, brain-based problem solving and avoiding danger. In this mode, attempts to access *positive* imagination may even be perceived as unpleasant, even dangerous.

As an example of imagination impediment in a partially-recovered PDer, I recall one who, whenever she tried to imagine being embraced and cradled by the loving arms of Mother Mary – a comfort that she keenly desired – she was immediately fearful that it wouldn't turn out to be Mary: it would be a murderer who was going to stab her in the back with a knife. It took several years of trying to let Mary comfort her before she was willing to accept the idea that, since it was her *own* imagination creating either the Mary or the dagger, *she* was in charge of whether or not, in her imagination, she got stabbed in the back. In an act of enormous bravery, she finally decided to let herself imagine being embraced by Mother Mary, and to use her own calm will power and loving concentration to prevent the image from converting into that of a murderer. When she successfully conquered this mental terror, she suddenly remembered witnessing a murder as a very young child. (Later, a few phone calls to older family members confirmed her memory. Also, the patient could suddenly make sense of her mother's frequent and even angry statements that no one could remember anything that occurred prior to age four and that all memories prior to age four were not true; these statements had occurred every time that the patient had started to mention a memory from early childhood. The patient also realized, suddenly, that she had been the only child in the family who was instructed that she could not have childhood memories.) When she remembered the murder and her shock and her terror, her lifetime of dissociation ceased. She was engulfed in a wave of feeling, followed by a wave of relief. She quickly shed her last, few symptoms of Parkinson's disease.

I provide this example in case any readers are starting to think sneeringly of PDers for having “brought this dissociation problem upon themselves.” I can attest that many of my patients were absolutely correct in having dissociated from what was, at the time, a horrendous event, an event that they were utterly incapable of dealing with at the time, and from which dissociation was the best possible choice at the time.

“Not safe” is the default condition

We had shown that a healthy person, when imagining death by tiger (dissociation), manifested a specific set of channel flow changes. These dissociation-type channel flow patterns are extremely rare: they should only occur when a person is in shock or at risk of immediate death.

And yet, these very rare channel flow patterns are the ones that *predominate* in partially-recovered PDers. Evidently, any time that they aren't feeling utterly safe, their bodies operate in dissociated mode.

So how often do they feel safe?

It varies, of course. Some usually felt safe while someone was holding their healed feet in the acupuncture office. They also were able to feel safe in highly specific situations that, for various reasons, they had deemed “OK.” As soon as the “safe” situation was over, however, they immediately reverted back to “not safe” (needing to be dissociated). In other words, the default setting for these people was “*not safe*.” And “safe” was a *narrowly* defined condition.

In a healthy person, the default mode is “safe.” In times of wariness, stress, or emergency, a healthy person switches *temporarily* over to sympathetic mode. A healthy person only shunts his electrical system into dissociative mode while experiencing a near death situation or imagining one.

Comparing fast-recovering PDers and partially-recovered PDers' neurological states

People with Parkinson's with untreated foot injuries are in “badly injured mode.” In this mode, a person's brain is trying to make the person go to sleep. Dopamine is not available when a person is in “badly injured” mode. The foot fails to heal however, and the “go to sleep” signal is ignored, because PDers have selectively dissociated from their feet injuries. However, some people have dissociated from their feet injuries *and*, during those moments that they deem potentially harmful, have learned to dissociate from their ability to perceive the feeling that accompanies expansion in the chest, also known as the feeling of “open heart.”

The fast-recovering PDers seem to have only had a foot injury: they do *not* have a mental habit of frequently inducing dissociation from their own ability to feel. For fast recoverers, numbness does not feel “normal” or “safe.” With the fast recoverers, when the foot injury is treated and the dissociation stops, they recover rapidly from Parkinson's – over a period of several weeks or a few months. When the foot is healed, they quickly feel a surge of well-being: they are no longer in a suppressed condition of injury. They do not need to learn how to reconnect with their hearts in general, because they have not made a lifework of practicing dissociation from their ability to feel anything they don't like.

The people who get stuck in partial recovery, like the fast recoverers, have also been practicing selective dissociation from their ability to feel their injured feet. However, when these people recover from their feet injuries and cease to be in “badly injured mode,” they start vacillating back and forth between parasympathetic and dissociative modes.

Partially-recovered PDers, once the foot injury is gone, are once again able to have normal energy flow and feel quite good *when they feel safe*. However, when they imagine that they are *not safe* (which, for many, is an steadily-increasing mindset), they dissociate.

We came to realize, after various mind experiments, that the defining characteristic of our partially-recovered PDers appears to be this: their default mode is dissociation. Although, in rare

moments of narrowly defined safety, they are able to revert, temporarily, to the parasympathetic system, the rest of the time, they feel most comfortable, they feel *normal*, if their channel systems are in prepare-to-die mode.

Prior to healing the foot injury, most PDer's do *not* vacillate between *parasympathetic* and dissociative modes. They tend to vacillate between *sympathetic* mode, deeply injured mode, and dissociative mode. They may have also have periods or fleeting moments of parasympathetic mode, depending on the degree to which they are able to relax and feel their internal sensations.

When the foot injury heals, the subconscious sense of urgency and injury decreases: access to the sympathetic mode and deeply injured mode declines. This is the point at which a person will either start being able to enjoy parasympathetic mode again, or will slide into partial recovery, spending increasing amounts of time in a dissociative mode.

Looking ahead a bit, we eventually learned that, for many PDer's who eventually become stuck in partial recovery, the trauma of being diagnosed with Parkinson's disease did in fact contribute largely to their plunge into body-wide selective dissociation, in which they dissociate from their ability to feel their entire body. This helps explain why PDer's symptoms often accelerate after receiving a diagnosis of Parkinson's. It also helps explain why our patients who have never received a diagnosis of Parkinson's recover so much more easily in response to the mere foot treatment: they have not dissociated to the same extent as those whose response to their dire diagnosis was to perform a body-wide selective dissociation.

Using the sympathetic mode to get going

After the foot heals, some partially-recovered PDer's experience a far faster *increase* in PD symptoms than might be expected in ordinary, idiopathic Parkinson's.¹

We recalled that some PDer's, prior to getting the foot injury fixed, had enjoyed invoking a sense of emergency when they needed to "get things done." Their lives had often been lived with an intensity of purpose and ferocious will – conjured up by invoking the sympathetic mode. They used their ability to concoct mental emergencies, thus shifting themselves into sympathetic mode, when they needed to "get going."

Because their dissociative, increasingly rigid condition was not based on a real pre-death condition, but only a mental one, they could over-ride it at any time by being in an emergency.² But in general, for the basic business of living, they were most comfortable and felt the safest while they were dissociating: while they were pretending to be nearly dead.

¹ I had a PD patient whose MD changed his original diagnosis when this shift *away* from sympathetic mode occurred. His original diagnosis was idiopathic Parkinson's. However, as soon as his foot injury healed, removing his source of adrenaline, this *extremely* emotionally frozen patient plunged headlong into severe dissociation: he was nearly immobilized. The doctor, stating that idiopathic Parkinson's never develops so quickly, changed the diagnosis to parkinsonism. The doctor, in this case, may have been thinking of psychogenic parkinsonism, which *can* appear virtually overnight in response to a severe trauma. There are no hard and fast rules on the rate of development of psychogenic parkinsonism: psychogenic parkinsonism develops slowly in some people.

² Prior to recovery, many PDer's had reliable "emergencies" that they could draw on to get themselves moving. Examples of these include "If the in-laws come over they will see the dirty dishes in the kitchen and they will think I'm bad," and "If I'm late to the appointment I will be bad" (or, "everyone will hate me). These and other "emergencies" usually consisted of situations that might show the PDer in a poor light. While these situations were not actually life-threatening, they served as a real enough risk to the PDer's sense of well-being that a PDer could actually rouse his own sympathetic nervous system by focusing on such fears. By contrast, the imagination techniques that we proposed did not actually provide the PDer with any sense of risk.

When the foot injury was healed, people who got stuck in partial recovery found it far harder to invoke their mental “states of emergency.” Very possibly, the existence of a genuine, painful injury had helped PDers invoke their sympathetic response. When the injury was gone, it became much harder to conjure up a sense of emergency.

Because the injury was gone, a safe time would be a genuinely safe time – full blown parasympathetic mode could actually occur, enabling a much richer level of sensory awareness and calm. But, at the same time, if the partially-recovered PDer was accustomed to dissociating from anything, even thoughts, that seemed to veer towards the negative, he would very likely find himself increasingly suffering from rigidity and tremor during those “negative” times.

In other words, during calm times, he would feel much calmer and might be able to move perfectly normally – because he was no longer badly injured. But during negative times, he would feel even more negative because he had lost some of his ability to “snap out” of dissociation by shifting into sympathetic mode.

This hypothesis certainly matched up exactly with the much greater intensity of come and go symptoms of rigidity and tremor that manifested in the partially recovered patients.¹

As we pondered the collection of neural modes that PDers slide in and out of – sympathetic, deeply injured, parasympathetic, selectively dissociated from a body part, selectively dissociated from the ability to feel with the heart – combined with the infinitude of psychological triggers that PDers have determined to be risk factors, we began to appreciate why no two PDers have the same collection of symptoms or symptom prompts.

¹ Some clinical neurologists may imagine that *all* PD symptoms come and go or imagine that symptom swing is a normal manifestation of Parkinson’s. To some degree, this may be because they are accustomed to working with medicated patients. In medicated patients, symptoms come and go because of drug dosages coming on and wearing off. But even prior without taking drugs, many PDers have noted that some symptoms come and go, depending on thoughts and moods. This fact, in and of itself, should have suggested to researchers long before now that a mental element was present in Parkinson’s. However, because of the high intellect, intensity of purpose and powerful will power shown by PDers, together with their very correct (harm avoidance) behaviors, it might be difficult for researchers to suggest that PDers have a mental illness. MDs have researched the Parkinson’s personality for years, but they are unwilling to take a step that would seem to “insult” people with Parkinson’s: suggesting that these people are contributing in some mental fashion to their own illness.

And possibly, it is correct to avoid the term “mental illness.” We tend to use the term “mental illness” for those people who cannot relate to the world in the same manner as most people. PDers can usually relate very well, very carefully and correctly, to the world. It is to themselves that so many of them cannot relate correctly. Since this personal relationship is well hidden from most of the world, PDers do not fit the usual understanding of “mental illness.” However, many PDers’ spouses will quickly volunteer that the PDer, however compassionate, logical, and well-intentioned, does *not* have a healthy way of dealing with his *own* feelings or his ability to feel his own body.

Hundreds of spouses have tried to explain to me the “something very wrong in emotional ability” that they sense in their PDer spouses, and they struggle for the words. So maybe it would be better to say that PDers have an emotional illness instead of a mental illness. Maybe we can say that they have an emotional illness that is kept in place, that is not allowed to heal, by virtue of a well-meaning mindset. I will leave these decisions to the future researchers.

But the main point of this footnote is that, even prior to healing the foot injury, the mentally-induced symptoms tend to come and go somewhat. But if a person has the dissociation from heart mind-set that spins him into partial recovery, he will find that his *physical* symptoms are gone even while the severity of the *mentally* induced symptoms is increased. When his foot injury is gone, the symptomatic swings and the come and go nature of the symptoms become far more distinct and dramatic.

I've wandered far from the main point, however. So getting back to the subject at hand, which is proof, this experiment proved that a person *could* mentally, quickly, and at will, invoke the Qi flow patterns, and therefore, the physical symptoms, of automatic dissociation.

This led to another question: could a person invoke symptoms of automatic dissociation by pretending that he couldn't feel pain? Certainly, none of my patients invoked their come and go symptoms by regularly imagining tigers. We suspected that what they did was more subtle: they merely told themselves that they couldn't feel pain.

So the question was this: could a person invoke symptoms of automatic dissociation by pretending that he couldn't feel pain? Could we prove it was possible?

Happily, I had already done another experiment, more than a year earlier, that answered this question. Our heart-in-a-box experiment had already provided proof that a person could invoke some symptoms of *automatic* dissociation merely by *telling* himself that his heart was numb.

But before I can describe the heart-in-a-box experiment, I need to explain how sensory perceptions vary depending on neural mode. Assessing changes in perception, changes in feeling, can give a clue to the neural modes that a person is experiencing. The next section serves as a very brief introduction to this subject. After that, we can jump into heart-in-a-box.

HOW DO I FEEL ME? LET ME COUNT THE WAYS...

The following list gives a very general overview of how different neural states respond to sensory stimuli and thoughts.

1) A *sympathetic system* response causes stimuli to have a negative, or "warning" connotation. The signals from the stimuli that enter the body via eyes, ears, tongue, nose, and tactile nerves are interpreted via the brain. The brain initiates responses based on instinct, habit and learning.

2) A calm, *parasympathetic* response to stimuli provides the richest possible resonance with the stimuli. In this mode, the heart serves as the primary interpreter for sensory stimuli. Information enters the body via the eyes, ears, tongue, nose, and tactile nerves, but this information is interpreted via the heart *before* being processed by the brain. The electromagnetic field of the heart resonates, or not, to varying degrees, with the sensory input. The heart transmits to the brain its responses to the stimuli, and the brain then responds based on the heart's instructions.

3) A complete state of *automatic dissociation* renders a person numb and seemingly dead. Both the brain and heart may be partially unresponsive or completely unresponsive to stimuli.

4) A *partial* form of automatic-type dissociation response may render a person somewhat numb. A person may have *some* sympathetic nervous system responses, but may not have full access to normal sympathetic feeling.

For example, a person who is "in shock" might be in this partial condition for a short while. He may be able to see and hear, but he may not have normal access to a full range of sensory input. His peripheral vision may be limited, and his hearing may be acutely tuned in to sounds of danger. If his autonomic nervous system has not yet fully stabilized, his heart's physical pumping action, as well as his heart's ability to receive sensory information, may also

be somewhat unsteady. In this condition, he may tremor and/or experience poor temperature or blood pressure regulation.

5) *Selective* dissociation from stimuli *might* produce any sort of reaction to the dissociated stimuli, ranging from amnesia to numbness to multiple personality disorder. The specific possibilities are endless, and depend on what direction that individual's brain chooses to go. As noted in the "numb from the waist down" example in the previous chapter, it is nearly impossible to guess what the brain might do with items from which it has selectively dissociated.

And making it all the harder to deal with, a person who has selectively dissociated from an event, or even some portion of an event, may not remember the event or he may not remember some portion of the event. After all, making the problem "not exist" is the whole purpose of selective dissociation. This means that, when symptoms of selective dissociation appear, they may *seem* to be spontaneous – unrelated to any consciously known trigger.

The above categories are merely an introduction. In real life, the distinctions are not so all-or-nothing. Most people are usually in a blend of sympathetic and parasympathetic modes. And most highly focused people have some number of subconscious selective dissociations going on at all times. When they tune out distractions, they might not even realize that they are doing it.

Examples of variations in sensory function

Smell

Consider a person who is in sympathetic mode when he smells wood smoke in the air on a summer evening. He might immediately wonder if he should call 911. He may even worry that he has forgotten to recharge his home's fire extinguishers. On the other hand, the same person, if he is in parasympathetic mode, if he smells the *exact same type of smoke*, might smile to himself and recall the summer barbeques of his youth.¹

Hearing

A person who is listening to music while driving will almost always turn the music off if he becomes lost or becomes uncertain of his directions. Getting lost usually triggers the sympathetic system. In sympathetic mode, the ear strains to catch sounds that might suggest danger. *All* other sounds are considered potentially dangerous distractions. Even if the driver has no thoughts whatsoever of danger and the driver really loves his music, his *perception* of his previously enjoyable music becomes altered when his heart-mind relationship shifts into predominantly sympathetic mode.

In sympathetic mode, despite his conscious love of music and despite all logic, the music *must* be perceived as a distraction, an annoyance, or even a genuine problem. Most lost drivers turn off the music and tell the passengers to stop talking until they figure out where they are and

¹ Studies show that people with post-traumatic stress disorder have a significantly decreased ability to smell. (Dileo, J, "Loss of Smell Linked to Post-traumatic stress disorder," *Psychological Medicine* April, 2008.)

On the other hand, people who are severely depressed retain their ability to *identify* smells, but they lose their ability to *feel* the emotional impact of various smells. ("Smell, Emotion Processor in Brain May Be Altered in Depressed Patients", Science Daily, March 2003: based on study done by Pause, Bettina, dept. of psychology, University of Kiel, Germany, published in *Psychophysiology*, March 2003.

their sympathetic mode surge of adrenaline is neutralized. As soon as a driver's neural system shifts back to parasympathetic, he will want to hear his music again.

Sense of touch or, more specifically, sense of internal touch

During an emergency, a person might not be able to feel the pain of injury: he may not notice that he has been severely injured.

During dissociation, a person may feel depersonalization: he may feel as if he is *observing* his body from the outside, rather than *feeling* himself inside his body. This is a form of numbness to internal sensation: numbness to "internal sense of touch." When internal touch sensations shut down, a person tends to use his visual sense and visual (negative) imagination to take the place of his internal touch sense. His proprioceptive sense, the "locator" aspect of internal touch, may also be inhibited.

A person who has experienced a severe shock, a condition that can neurologically resemble dissociation, may "feel numb inside." This person may well be able to feel with his index finger the difference between velvet and sandpaper, but he may be numb to his own sense of how he feels inside his own body. His body awareness, that is to say, his sense of how he feels his own life-force and vitality surging around inside himself or not, or his ability to feel a physical sensation of vitality or joy concurrent with inhalation or chest expansion – the most basic of all sensory feelings – may be numbed.

People with Parkinson's often insist that they are not numb because they can detect external textural differences. However, their ability to detect their own internal sensations is usually highly inhibited. Their sense of proprioception is very often declining. Usually, they do not even realize the extent of their internal numbness until they start to recover.

Examples abound of the ways in which responses to sensory input vary if the recipient is in parasympathetic as compared to sympathetic. And if a person transitions into dissociative mode, the change in sensory perceptions can be even more extreme, as demonstrated in chapter xxx in the case of the woman who went numb from the waist down due to dissociation from an emotionally painful childhood episode.

This quick look at the various states of perception relative to the neurological modes of the body is the merest of introductions and might raise more questions than it answers. An expanded explanation is included in appendix xxx: The Adrenaline-Dopamine Relationship. This introductory section was inserted to point out that the type of sensory perceptions a person is experiencing can be a good indicator of a person's neural mode.

Now we can consider how these sensory shifts relate to our heart-in-a-box experiments.

PROOF #2: HEART-IN-A-BOX

Back in early 2007, when we first starting asking our patients to do the heart-in-a-box experiment, we merely thought that this technique might be able to "unstick" a person who was stuck in a dissociative state. By pretending to duplicate the conditions that had first triggered the dissociation and then pretending to turn *off* the condition, we hoped they might be able to actually turn off the dissociation that they seemed to be locked into.

For the experiment, we asked them to imagine their hearts sealed up in an impenetrable steel box so that no sensory input could register with the heart. They mentally affirmed several times that *no* sensory feeling could penetrate the heart. They also affirmed that all sensory input

would be processed via the brain. Then, they gazed out the window looking at the trees, sky, and clouds, listened to ambient sounds, nibbled food bits, and smelled flowers. If they wanted, they could walk around or perform simple motor activities.

About four minutes later, we had them remove the imaginary box and perceive all the same sensory inputs as though the sensory events were traveling to the heart, instead of the brain, while affirming that all sensory input was being felt via the heart. All motor functions, including speech, should be performed as if they were originating in the heart instead of the mind.¹

The technique did work, after a fashion. The trick allowed most PDers to experience a spurt of enhanced, parasympathetic-type sensory awareness when they got rid of the box. Some PDers even enjoyed this novel surge of parasympathetic-type sight, sound, taste, smell, or tactile awareness, and the calmness or happiness that accompanied it.

The more incredible thing was that they didn't struggle with this technique, the way that they had with previous exercises in visualization. Some even did their "homework" and practiced the technique after leaving the office.

We were pleased with this unexpected level of compliance until we found out why they were able to do this exercise so easily. They could do it without a struggle because it felt so *normal* to shut the heart in a box. For some PDers, doing the exercise even felt *good* because they were shutting their hearts off even more rigorously than usual.²

As for the shift in perceptions, some of them enjoyed the fleeting, novel experience of parasympathetic perceptions when the box was removed. Others were deeply confused by the shift in sensations. A few felt no difference. One told me, with unshed tears in his eyes, that he had felt "No difference." When I pressed him (on account of the tears), he grudgingly said, "No difference: only happiness." We were just beginning to realize that, for many PDers, happiness is a potential risk situation.

Of all our psychological experiments, this one was probably the most educational for us. The positive and negative responses to this technique led us to the research that, in turn, led us to the hypothesis that selective dissociation from the ability to feel was triggering symptoms that resemble partial automatic dissociation.

In retrospect, it also provided proof that, by pretending to shut down the heart, by merely imagining his heart to be inaccessible, a person can provoke a shift in sensory responses that corresponds to a shift in neural mode. When a person pretends that his heart is in a box, vision,

¹ This experiment has a prerequisite: The PDer needs to be able to imagine that he has a heart. Some PDers worked for nearly a year before they could even imagine themselves having a functional, vibrant heart or a heart that was as radiant as that of a "good" person. Before a PDer was fit to do the heart-in-a-box experiment, we asked him to imagine the heart of a "good" person, a loving person that he admired. Then, the PDer had to practice imagining himself having a comparable heart. This was *not* an easy or pleasant task for many PDers. But until they could imagine themselves having a heart, there was no point in them trying to do the heart-in-a-box experiment.

² Of the dozens of partially recovered PDers who did this experiment, only two partially-recovered did not *like* shutting the heart in a box. These two people said that putting the heart in a box was scary. Both of these people were extremely guarded even when they weren't imprisoning their hearts. For both of them, putting the heart in the box was perceived as yet another source of danger. However, they still had no idea what we meant when we asked them to perceive their sensory feelings via their hearts. In other words, they didn't like to have their hearts visually imprisoned, but they didn't *use* their hearts even when the hearts were *not* imprisoned.

The small sampling of non-PDers and recovered PDers who did the experiment didn't necessarily *enjoy* putting their hearts in boxes, but they weren't scared or threatened by it.

hearing, taste, smell, and touch all become narrowed, limited, and are analyzed rather than actually felt. When a person pretends that his heart is *not* in a box and that all sensory input must go through his heart instead of going through his brain, his sensory functions yield richer responses and the responses are felt more than analyzed.

For example, with the box gone, colors are brighter, peripheral vision is expanded. Ambient sounds are interesting instead of annoying. Flavors are stronger and more stimulating to the palate. A new sensation, a feeling of hunger, might be felt in a person who never feels hunger and only eats “by the clock.” And so on.

More to the point, nearly all of the PDerS who were stuck in partial recovery felt that the boxed-heart sensations were normal and safe. Unboxed-heart sensations were powerful and startling. This confirmed for us that PDerS were not accustomed to perceiving the world via the parasympathetic mode.

In addition to asking our partially-recovered PD patients to do this technique, we ran an experiment in which three groups of people did this technique. The first group was professional musicians who have never had any symptoms of Parkinson’s. The second group was people who had fully recovered from Parkinson’s disease. The third group was partially-recovered PDerS.

Telling the whole story of this experiment and all the results would impede the pace of this book. But because the individuals’ responses were so powerfully telling, I’ve included a write up of this experiment in appendix xxx.

This chapter will only share a few crucial findings and statements from the subjects: statements that, in retrospect, we consider to be proof for our dissociation hypotheses. I will start with the most surprising statement.

“I remember doing that”

One of the subjects was a fully recovered PDer. He had never had an MD’s diagnosis of Parkinson’s, and he never suspected that I was treating him for Parkinson’s disease. He had come to see me at his wife’s insistence. He definitely had early stage Parkinson’s. (Details of his PD symptoms are included in appendix xxx.)

Following my policy for working with people who do not have a doctor’s diagnosis of Parkinson’s, I had never used the words “Parkinson’s disease” with him. I didn’t give him a diagnosis, but said only that sometimes a dragging foot can be caused by some structural displacement in the foot from some old, long-forgotten injury. I did not mention the significance of his increasing difficulty in initiating movement, his shrinking handwriting, his reduced vocal ability, his increasingly slow response time during sports and the many other PD symptoms that he told me about, or which I asked about and he confirmed, or which were visible to the naked eye. He attributed all of his slowing-down symptoms to “the normal process of aging.” He was in his mid-forties.

I did not discuss my Parkinson’s work with him. So far as I know, he never read any of my articles or books. He’d had a bad foot injury when he was eleven years old. He didn’t remember the foot injury until our second session. I worked on his foot injury five times; each session was one hour long. All his “normal symptoms of aging” cleared up over the next few months.

More than seven years after recovering from early-stage Parkinson’s, he came back again to my office to ask if I could help with his psoriasis.

During one of his psoriasis treatments, I asked him to do the Heart-in-a-Box experiment. I figured that he was a great candidate for the “recovered PDers” group because he did not know he’d had Parkinson’s and he did not suspect any outcome from the experiment. After doing the Heart-in-the-Box experiment, he made the following observations.

“It’s easy to do [shut the heart in a steel box]. Maybe *too* easy! (Laughter). It seems so familiar; I remember doing it when I hurt my ankle [when I was eleven years old].”

I asked if he had noticed any sensory shift going into the box.

“No, no awareness of sensory shift because [with the heart in the box] I was trying to *shut out* sensations. When I stopped [got rid of the box], it was great. I *really* enjoyed hearing and seeing everything.”

I was so stunned at his statement that he had done this when he hurt his ankle, a subject that we had not broached in years, that I questioned him. “You did this when you hurt your ankle?”

He replied quickly, “Oh! It was obvious immediately.”

I repeated his words to myself silently as I jotted them down. He had said, “I remember *doing* it.”(!) He *hadn’t* said that a similar set of sensations had spontaneously occurred, of their own accord, in response to his ankle injury. He remembered *doing* it. *Doing* a conscious game of pretending to have his heart shut down.

My jaw was hanging open. In terms of my research, this was like winning the lottery. Without any reason to suspect a specific desired outcome on my part, this man had spontaneously told me that, when hurting his ankle, he had mentally invoked a condition in which he’d pretended that his heart was shut down so that he’d been unable to feel, in his heart, his physical pain.

The implications were glorious: in our search for the cause and correct treatment of idiopathic Parkinson’s disease, we were definitely on the right track.

Prior to this, other fully recovered PDers had suddenly, calmly, *during* recovery, stunned themselves and us by making a statement to the effect of, “Oh. I remember when I *decided* to start being this way,” and then went on to tell that they had decided not to feel pain because of some situation that they hadn’t liked or which terrified them. They had completely forgotten about this decision until, suddenly, while recovering, it had all come back to them.

For research purposes, because these people were intentionally looking for some way to understand their Parkinson’s disease, their memories were suspect.

But the psoriasis patient was not trying to throw his mind back to remember anything in particular. And yet, in response to this experiment, he had laughed and said, “I remember doing it when I hurt my ankle.” We could now add his excellent, unprompted evidence to that of other recovered PDers. His response to the experiment, added to the responses of other recovered PDers strongly suggested that, once they recovered, ex-PDers could remember having *chosen* to set their numbness in motion.

Proof

This experiment provided proof that a person could alter his ability to perception of sensory events towards sympathetic- or dissociative-type perceptions by simply telling himself to not feel via his heart.

The responses of all the healthy participants (the musicians and the fully recovered PDers) showed that their movement style, thoughts, and sensory functions were clearly inhibited or altered when they put their hearts into a box and affirmed that they could not perceive sensations via the heart. These changes were consistent with dissociative and/or sympathetic mode: wariness; negative, analytical or evaluative perceptions (as opposed to sensory feelings); and even depersonalization – the hallmark indicator for dissociation. When their hearts came out of their boxes, their movement, thinking patterns, and sensory functions surged back, with feelings of relief, towards parasympathetic-type behavior.

And it proved our boldest hypothesis: a person can quickly and temporarily alter his *manner* of movement and his *perceptions* of sensory input by simply telling himself that his heart is unable to feel sensory input. A person can shift his body into a different neurological mode by pretending that his heart doesn't feel.

Expanding on that idea, a person of strong mental focus could, conceivably, shift his body *for the long term* into a pattern similar to automatic dissociative mode by selectively dissociating from his heart's ability to feel pain at all times – except for those times when he felt absolutely safe.

Putting it another way, a person who trained himself to shut down his heart whenever he felt unsafe might manifest symptoms similar to those of Parkinson's disease every time he selectively dissociated from his heart. And these symptoms could become somewhat fixed or they might come and go as quickly as thoughts come and go.

CHAPTER SUMMARY

This chapter shared just two of the many experiments that contributed proofs that such mental choices and such results were possible. Not only were they possible, but many recovered PDers volunteered that they had at one time made these mental choices.

While it might bolster our arguments if we could experiment on thousands of PDers, we don't really need to. All we needed, at this relatively early stage in our research, was to prove that mentally-induced selective dissociation from the ability to feel *could* set in motion neural changes corresponded to symptoms similar to those of automatic dissociation. The two experiments written up in this chapter and dozens of other smaller experiments, not yet written up or published, contributed to our proof that this was indeed possible.

Next, if we could also bring about a *cessation* of mood-related PD symptoms in partially-recovered PDers by training a PDer to stop using dissociation as his default system, that symptom cessation would serve as proof that we had, in fact, discovered the cause of the “mental blockage” that some PDers and all people with psychogenic parkinsonism were struggling with. A permanent cessation of symptoms, that is to say, a lasting cure, would be proof that we had correctly identified the underlying cause of Parkinson's disease and had learned how to cure it.



“In this drama of life, your love must be greater than your pain”

Paramahansa Yogananda

CHAPTER THIRTY-FOUR

THE CORRECT WAY TO DEAL WITH PAIN

We figured the PDers dissociated from their ability to feel pain (dissociated from their hearts) because they did not know how to deal with pain. We realized, with alarm, that they might not even know how an emotionally healthy person deals with pain. We were going to have to teach them how to deal with pain.

Emotionally mature people deal with pain by applying a neutralizing “good feeling” to the pain. The “good feeling” is the same feeling, or very similar to the feeling that I’ve referred to as “the feeling one experiences from expansion in the chest. PDers who are stuck in partial recovery usually don’t even know what is meant by the “the *feeling* that accompanies expansion in the chest.”

We realized that, in order to help PDers be able to re-associate with their hearts, we would have to figure out how to describe this wordless process of pain neutralization, one that uses indescribable feelings, so that they could learn how to deal with pain. The following is an attempt at explaining how to correctly process pain.

The infant deals with pain

A young child “deals” with pain by crying. When he cries out in pain, a loving adult is supposed to hold him, cradle him. When this happens, the child can feel the pain and fear (which is caused by chaotic electromagnetic forces surging through his body, sending “error” messages to his brain), *but* he also can feel the greater electromagnetic field of the loving adult. The adult’s electromagnetic heart field, moving correctly in the “human” pattern, is physically larger than the child’s suddenly chaotic electromagnetic field. The adult’s larger field is able to influence and restore to some semblance of normalcy to the disturbed, even deranged currents running through the injured child’s body. The adult can easily comfort the child because the adult’s healthy electromagnetic pattern is *physically* larger than the child’s chaotic pattern.

As the child’s electromagnetic fields and currents begin to resonate with the much larger fields of the adult, the child calms down. In response to the corrective influence of the larger person’s field on his much smaller field, his “error” signals, which cause fear, are shut down. Instead of fear and pain, he is left with mere “pain” sensations. These pain sensations can peacefully exist in the context of his once again electromagnetically “human” body. As soon as the child’s electrical system is stabilized by the adult’s larger, stable, harmonious electromagnetic system, the child is able to *feel* the pain without being terrified of it. The pain is no longer threatening: it is just a sensation. The body knows how to deal with injury-based sensations.

As soon as the heart and mind recognize the injury-based sensations and initiate processing the damage that triggered the pain, the brain calms down. The pain is no longer compelling and no longer causes fear. The injury-based sensations become merely a mild signal that serves as a gentle reminder that the injured body part needs special attention. When the body’s currents are stabilized and the brain is satisfied that corrective steps have been initiated,

the fear-based *pain* has served its purpose: it can now stop. The brain no longer registers the nerve signals coming from the injured area as “pain.”

At this point, when fear-type pain converts to mere sensation of injury, we say that the pain has been “processed.”

“Processing pain” occurs when the body calms down enough that it can turn off the sympathetic nervous system (the flight, flight, or freeze system) and switch back to parasympathetic (calm) system. Healing only occurs when the body is in parasympathetic mode. In this calm, non-fear condition of parasympathetic mode, the body can calmly use the non-threatening sensation of pain to assess the problem and get to work on healing the injury.

Growing up

As a child grows up, he soon gets too big to be completely cradled in his mother’s arms. When he is big and he gets injured, he may need to settle for a hug, since he no longer can be picked up and held. The larger child can no longer be “surrounded” by the adult’s larger field. Instead, he must learn to *tune in* with the comforting feeling of being *adjacent* to a stable electromagnetic field from another person’s heart. When the larger child gets a hug, or a pat on the back, he is able to use that close contact with the other person’s heart field to restabilize his own.

As he does this, he is supposed to learn how to lean on the harmony in a supportive adult and supplement that harmony by *invoking* harmony in himself – despite his pain. This is how children are supposed to learn how to comfort themselves after being destabilized by physical or emotional pain.

Even when the child has grown too large to be literally surrounded by a harmonizing field, he can learn to use a nearby electromagnetic field (from a friend or parent’s heart) to stabilize himself, calm himself down, and bring his heart back to parasympathetic mode. As soon as he can restore his body’s electromagnetic system and his electrical currents to some semblance of order, he can *feel* his pain without being overwhelmed by it.

As long as the field of correctly moving electromagnetic energy covers a larger area than the physical area of the pain, the pain is bearable. The pain will cease to be perceived as a terror or a mortal threat.

When the child becomes an adult, he is supposed to expand further on the above principle. He needs to learn how to draw on his own heart’s electromagnetic field. When he is injured, he can conjure up the harmonizing, comforting feeling of the heart’s field by “expanding” or “opening” his heart. By focusing on the feeling associated with the expanded heart, he can then surround and de-terrify his pain by making his “heart feeling” larger than his “pain feeling.”

As an adult, we are supposed to know how to draw on our self-contained sources of electromagnetic harmony (our opened heart). An adult should be able to use his own heart’s electromagnetic field to deal with pain. If an adult experiences a jarring physical *or* emotional pain, either of which can destabilize the body’s circuitry, he is supposed to know the correct method for making the pain bearable: he can open his heart: he can allow himself to feel, inside his chest, that “feeling of expansion” that healthy humans experience during moments of great peace, gratitude, or inspiration.

Then, he can use that expanded chest sensation to counter the destabilizing pain. By mentally imagining a physical increase in size of the expansion-in-the chest feeling that

ordinarily occurs when content or in the presence of great beauty, he can make his “heart” sensation is large enough to encompass the area that is in pain. Then, the pain becomes bearable.

As the self-comforted body then switches from sympathetic (panicked or fearful) over to parasympathetic, the healing can begin. The pain can then be processed by the body’s healing faculties.¹

In my limited experience, most of our patients with Parkinson’s who have become stuck in partial recovery have no idea what I am talking about in the above paragraph. In particular, PDers that get stuck in partial recovery have no idea what I mean when I refer to a physical sensation of expansion in the chest that is supposed to occur when during moments of relaxation, peace, and inspiration. If the reader has no idea what I am describing, just bear with me. This sensation, and its significance, will be explained in extreme detail later on.

But the PDer should know that millions of people *do* understand what I am talking about. They may use different terms, and many will use spiritual terminology to explain the process, but they do understand what I mean about dealing with pain by drawing on a power *larger* than the pain: a power that is filled with calm. This is *very* different from dealing with pain by becoming numb.

Many of our patients who have become stuck in partial recovery also have no idea that it is possible to experience real pain – really *feel* it – and not be threatened by it. To them, the words “pain” and “threat,” even “mortal threat,” are synonymous. They have no idea what I am talking about when I say that one must surround the pain with love until the pain becomes mere *sensation*. After the pain becomes reduced to mere sensation – without the fear – it can begin to heal.²

For many PDers, a major goal of life has been the avoidance of pain. But pain must be processed so that it can heal. There is a right way to deal with pain, and thousands of wrong ways. The right way is to give a *feeling* of stable support to the body until the pain becomes mere sensation. One *wrong* way of dealing with pain is making oneself numb. Other wrong ways are nursing the pain, blaming others for it, dwelling on it. These styles of dealing with pain are excellent for developing self-pity, anger, resentment.

A history of injury and/or emotional pain is not the real trigger for Parkinson’s: incorrect *management* and *treatment* of the pain is the root of the problem.

If a person receives a terrible injury and deals with it by *shutting down* his ability to feel pain, when can he turn his feeling capacity back on? Never. If and when he gets around to turning his feeling ability back *on*, he will necessarily feel the pain that he has temporarily suspended. If he does not know how to process pain, and his goal is to Not Feel Pain, he will

¹ Of course, I am writing about how a “mature” adult deals with pain. There are plenty of immature adults who do not process pain correctly: they are the “poor me” people. We all know someone like this. These are the people who are fascinated with every little insult or injury and think that everyone else should be, too. These “adult children” focus on their pain, tell everyone about their pain, and cultivate their pain. When I refer to “adults” learning to deal with pain correctly, I am referring to mature adults, not the ones that cultivate self-pity.

² People in the southern United States have an expression, “sweet pain.” Many PDers, though very intelligent, have no idea what *feeling* this phrase is describing. “Sweet pain” refers to sensations of pain that are bearable because they are filled with love, harmony, or understanding. Another example of pain that is not a mortal threat is contained in Shakespeare’s phrase, “Parting is such sweet sorrow.”

never be able to turn his heart back on with regard to that particular injury – because turning his heart back on with regard to that injury will cause the suspended pain to once again be *felt*.

Here is the rub: the way to terminate a spate of pre-death dissociation is to experience a wave of feeling.

Many a PDer does not *want* to experience a wave of feeling. If he does, he will then feel the pain that he has been at great pains to avoid. Therefore, he tells himself to *not* experience a wave of feeling. But, since turning the dissociation completely off *and* switching the heart back on *only occurs* in response to a wave of feeling, the PDer is stuck. If he turns the heart back on, he risks feeling his pain. If he leaves his heart turned off, he is stuck in a physical condition in which he cannot move very well – *unless he uses adrenaline to temporarily override his dissociation*.

How can emotion create a “feeling”?

Many PDers truly do not know that emotional pain can cause physical sensations or even physical pain. I will suggest a few of the more common physical feelings that can accompany emotional pain: lump in the throat, painful twisting of the stomach, nausea, a “sinking feeling” in the gut, and pain in the solar plexus as if one has been kicked in the gut.

Many PDers have assured me that they do not have any painful events in their past because they have “mentally processed” and “come to terms with” their negative events. However, because they have most likely dissociated from the *pain* of the event, they have never physically processed the *physical* sensations of the event. If they dissociated from the *pain* of the event and created a mental story to put the event in a “logical” place, that dissociated pain is still sitting somewhere in the brain, laying low, waiting to be processed.¹

For years, we had assumed that keeping the heart open only required a shift in mental attitude. We didn’t realize that, for some deeply blocked PDers, another step had to come occur: these emotional infants and young children had to learn how to correctly process pain.

Were all of our years of heart experiments pointless? No. Figuring out how to recognize the sensory shifts that occur when the heart is opened *did* turn out to be a helpful first step. Some PDers need to go through all the exercises that we have shared in the preceding chapters in order

¹ Semantics became a real problem. Despite our careful definitions, most of them responded to our questions about sensory numbness and “shut down heart” with denials. They insisted repeatedly that they were *very* sensitive people. To prove their point, they usually gave the example that they were deeply concerned about others. We kept insisting that we were talking about physical sensation, and they kept saying that they were selflessly concerned about others. We can only assume that, because they are so profoundly numb to their own bodies, they truly do not know what we are talking about when we say, “physical sensations in your own body.” As a rule, they have a *very* hard time understanding the words “sensory feeling” or “physical sensation.” We finally learned that, for many of them, “physical sensation” is a term that is synonymous with “pain.”

In response to our repeated explanations, in which we tried to make the point that we were talking about basic physical sensation, and not emotionisms, they repeatedly insist that they are not numb in the heart: they care for others; they are deeply concerned for others. As I say, semantics problems. I finally learned to ask, “Is your ability to see somewhat inhibited? Is your ability to hear somewhat inhibited?”

One PDer’s spouse even got angry with us when we started asking questions about his wife’s heart responses. He insisted that she was the kindest person he’d ever met. When we explained to him that we were talking about physical sensitivity and physical numbness, he understood what we meant right away. She, however, did not.

to get to the point where they can learn how to process pain. So, all of the stages that we went through did turn out to be helpful, in the end.

All that remained now was teaching people with no sense of the “chest expansion feeling” how to experience that feeling. And then, we would have to teach them, step-by-step, how to use it to process pain. Then, they would be able to stop dissociating from their hearts.

We still needed to encourage PDers to visualize their hearts. If they couldn't understand what benefit might be garnered from having a heart, we had them put their heart in the box. If they insisted that they had no dissociations, we had them spend a “day at the beach” and observe the different feelings that arose in the body. We still use the techniques shared in the previous chapters. Also, PDers who have come to be treated in our program after having read about these preliminary techniques seem to be far more ready for the final healing techniques that were not yet in print, techniques that we had to teach orally.

The two things that PDers had to learn, it turned out, was 1) how to feel safe and 2) how to correctly deal with pain.

They were able to learn the feeling of safety either by practicing a gratitude exercise that we developed or by creating their own exercises in gratitude. By practicing unconditional gratitude, they were able to develop mastery over their negative thinking, and feel safe – or at least as safe as a mouse. Mice, when they feel safe, experience a surge of dopamine in their midbrains.

They were able to learn how to correctly process pain by following the instructions in Part IV, the Treatment Techniques part of this book.



PART IV

TREATMENT TECHNIQUES

“Like a bridge over troubled water, I will lay me down”

- Paul Simon

CHAPTER THIRTY-FIVE

DEMYSTIFYING TUI NA

I have already referred in passing to an extremely Yin style of Tui Na, Forceless Spontaneous Release, that we use in assessing and treating our Parkinson’s patients’ foot injuries. This chapter is a quick introduction to Tui Na in general, the difference between Yin and Yang Tui Na, and a short bit explaining how I came to learn Yin Tui Na. The following two chapters will teach the FSR technique. The three chapters after that will teach the even more Yin variation of FSR that we use for the most stubborn blockages.

Tui Na: a definition

The words Tui Na refer to any scientific method of applying hands on a patient in a therapeutic manner. Tui Na (pronounced “tway nah,” as if to rhyme with “Hey! Ma,”) is represented in Chinese writing by the two characters Tui and Na. It is nearly impossible to translate literally from Chinese into English, but an attempt at defining these words yields the following: Tui means push or shove,¹ and Na means hold or take.² Tui Na is a generic term, not a specific technique. For example, both of the following are Tui Na: the vigorous technique of shoving an athlete’s displaced upper arm bone back into its shoulder socket and the gentle technique of stimulating an infant’s nerve centers alongside his spine by gently rolling the back-skin. Both of these techniques use the hands in a prescribed manner to help stimulate healing, and, therefore, both of them are types of Tui Na.

Some people might incorrectly translate Tui Na as “massage.” Massage, to most English-speakers, involves some sort of rubbing or kneading. While some types of Tui Na include rubbing or kneading, most do not. Therefore, even though the title of the official Chinese government’s English translation of the official Tui Na textbook is *Chinese Massage*, the title’s translation actually does the text a disservice.³ Most of the techniques described therein bear little or no relation to massage, as we understand massage in the west.

Some examples of western techniques that fit under the heading of Tui Na and which are clearly *not* massage are some of the more forceful therapeutic techniques of chiropractic, certain osteopathic treatments, Rolfing, and the replacing of displaced joints by allopathic (western) doctors using an insightful, but powerful, push or shove. These are strong, manipulative techniques. They are *not* massage, but as they are scientific health treatments that use the hands, they fit under the heading of Tui Na.

Examples at the other end of the forcefulness spectrum of western techniques that fit under the umbrella term “Tui Na” are the light-touch therapies. These light-touch therapies include various types of craniosacral therapy, Unwinding therapy, Zero Balancing, Healing Touch therapy, Positive Touch therapy, Bowen therapy, and hugs, to mention just a few in an

¹The Pinyin Chinese-English Dictionary. Commercial Press. Hong Kong. 1979. p.698

²Ibid. p.486

³*Chinese Massage*. Publishing House of Shanghai College of Traditional Chinese Medicine. Shanghai.

explosively growing field of light-touch therapies. These are all types of Tui Na; they are hands-on, scientific techniques for promoting healing.

Yin and Yang styles of Tui Na

The names “Yang Tui Na” and “Yin Tui Na” do not describe specific techniques. Yang Tui Na is a general term referring to those techniques of Tui Na that feature strong, forceful, and obvious movements that are visible to the naked eye. Yin techniques are so subtle that very often an observer might think that nothing is happening. Some light-touch, Yin types of Tui Na employ almost invisible levels of force. Some use no directed force at all, but work on the principle that manual support of an injured person or body part initiates or accelerates the ability of an injured person’s body to heal itself.

The two phrases, Yang Tui Na and Yin Tui Na, are generalized descriptive terms that explain where a technique should be placed on the huge continuum of hands-on techniques that range from rough and rowdy to meek and imperceptible. On that Tui Na continuum, those techniques that are the most vigorous are referred to as being the most Yang. The most gentle techniques are referred to as being the most Yin. The hands-on therapies that are moderate in force and intent, including many therapies that we in the west call “massage,” are placed somewhere on the middle of the continuum: neither extremely Yin nor extremely Yang, they are possibly moderately Yin, moderately Yang, or even just Somewhere-in-the Middle Tui Na techniques. All medical techniques that are hands-on can be placed somewhere on the continuum that ranges from very vigorous to extremely gentle.¹

Choosing a treatment modality

When a patient comes to an Asian doctor for treatment, the doctor must decide, based on the diagnosis, which treatment is most appropriate: herbs, acupuncture, Tui Na, dietary counseling, or energetics (Tai Qi, Qi Gong). The age and constitution of the patient, as well as the nature of the problem, will help determine what type of therapy is used.

Choosing Tui Na

Certain medical situations recommend themselves for Tui Na treatment. For example, infants often receive a special type of Tui Na instead of foul-tasting herbs or acupuncture needles. At the school of Asian medicine that I attended, nearly all infants were treated with a gentle skin rolling Tui Na technique. This technique would be considered somewhat Yin when compared with the vigorous arm-twisting or neck cracking techniques of Tui Na, and yet, because the skin rolling does use some force and intent, it should probably be placed on the Yang side of the Yin-Yang continuum.

¹ Nearly all of the techniques in the recent (1980s) official Chinese medical school Tui Na texts are either pediatric Tui Na or the very Yang techniques: powerful, bone moving techniques similar in many ways to the spine-cracking techniques of original chiropractic. (Modern chiropractic is usually much gentler.) In centuries past, the official Chinese medical books described information on many Yin, or gentle, types of Tui Na. Now, the official books do not describe any techniques of Yin Tui Na. The official writing only refers to Yin Tui Na in the introductory chapter as being used in the opposite situations as Yang Tui Na. The Chinese government’s revisionist policies in the twentieth century included the altering of medical lore, removing those aspects of medicine that might provoke scorn in the west or encourage spiritual practice in the populace. Now that “Yin” techniques are becoming common in the west, it is rumored that the Chinese practitioners of Yin Tui Na, those who have been doing Yin Tui Na in secret right along despite political disapproval and public disavowal of these techniques, are starting to come back out of the closet.

In addition to being used on infants, Tui Na is very often the modality of choice any time that tissues, including muscles, bones, tendons or ligaments, are displaced. Tui Na can also be used for other types of physical problems, as well as some types of emotional problems. The following paragraphs give a few examples.

Broken bones: an example of overtly distressed tissue

As an extreme example of displaced tissues, consider broken arm bones that stick out through the skin. Which healing modality should be selected? Herbs to increase blood flow and provide calcium? Acupuncture to stimulate the electrical currents of healing? While it is true that acupuncture aids in the healing of broken bones, often reducing by 75% the length of time required for the bones to knit, it would be ridiculous to stick acupuncture needles alongside of bones that are sticking through the skin and expect, somehow, that those bones will spontaneously set themselves back into place in response to acupuncture needles.¹ In a case like this, it is most likely that some type of Tui Na should be the first modality to be applied. The health practitioner should select a type of Tui Na that will bring the broken ends of the bones together, or bring them at least close enough together that the bones can knit. (After the bones are in position, it might make sense to use acupuncture and herbs to encourage healing.)

More discretely distressed tissue

Even if nothing is obviously sticking through the skin, many physical injuries still involve displacement of various tissues. In these cases, techniques of Tui Na that allow the body parts to straighten themselves out can be appropriate treatment modalities. Again, Tui Na is often the first modality of choice for injuries in which some body part is twisted, jammed, pushed or pulled out of place, broken, or broken-and-displaced.

Distressed emotions

Tui Na may be useful in more subtle health problems. Very often, emotional tensions can be relieved by Tui Na therapy. As an obvious example, a person in the throes of an emotional trauma might be comforted out of hysteria via the healing “technique” of a comforting hug. A person who is stifling and/or holding on to an emotional trauma may be able to release some of that trauma in response to the supportive hand placement or hand movements of some (usually Yin-type) Tui Na.

Suppressed emotions

People with emotional traumas very often hold tension in their neck, lungs, diaphragm, liver or heart area, to just name a few holding spots. By applying hands-on support to these and other soft tissue areas, a therapist using Yin-type Tui Na methods can often initiate healing of physiological problems such as asthma, insomnia, indigestion, or other maladies and pains that are related to the traumas being retained in the soft tissue.

¹ As a teacher of Asian medicine, I frequently meet gung-ho students who are determined that acupuncture should be the solution for every ill; I use the above broken bone example a lot.

Choosing which type of Tui Na to use

When a health practitioner has decided that a particular problem might be best addressed via Tui Na, as opposed to or in addition to other types of medical modalities, he then needs to decide what style of Tui Na should be used.

Generally, the following rule applies: Yang techniques are used when an injury is recent, painful, and obvious. Yin techniques are used when an injury is still unhealed, but old, painless, and hidden or forgotten. These Yin injuries can cause pains or problems near the point of injury or distant from the point of injury, but these problems usually appear to be unrelated to the actual injury. The underlying, unhealed injury itself may be forgotten or concealed even while setting in motion other problems.

If a health practitioner has decided that an ailment should be treated with Tui Na, he must decide where, upon the long continuum of human-ailment causes that range from painful to forgotten, a person's malady originated. Having decided on the nature of the underlying cause of the problem, he can then pick the appropriate type of Tui Na.

People with Parkinson's have Yin injuries

The few case studies described in chapter nine featured people with old, painless, hidden injuries. These injuries may be contributing to painful, obvious problems with muscles and movement, but the causative injury itself is laying low. Consistently, the foot injuries that we have seen in hundreds of people with Parkinson's have also been old, painless, hidden or forgotten injuries. PDers may, of course, also have a collection of more obvious injuries, but they do, in our experience, have some hidden, forgotten injury in the foot that is instigating problems hither and yon in the body. The obvious problems seem unrelated to the old, forgotten injury. These foot injuries are Yin: hidden or forgotten, painless and old.

The types of Tui Na that we have found most effective in treating PDers' injuries are Yin. One type of Yin Tui Na that we use is referred to in this book as Forceless, Spontaneous Release (FSR) Tui Na. The other type is a derivative of FSR.

The main types of Yin Tui Na

In the vast panoply of light-touch therapies, it is nearly impossible to say where one named technique leaves off and another begins. In the last thirty years, it seems as if dozens, maybe hundreds, of therapists have been busy developing some new "unique" version of light-touch therapy and slapping their own names or a copyrighted trademark on some variation of touch. As an interesting legal point, techniques cannot be copyrighted. Names for techniques and the text used to describe them can be copyrighted, but, actually, the act of touching a person in a therapeutic manner cannot be copyrighted or patented.

For example, holding a person's hand can be considered a form of hands-on healing. There are many ways to hold someone's hand. An inventive person could, if he wanted, make up a specific name for a variation of hand holding that involves, say, interlocking the fingers. He could name this position after himself and copyright that name. This would *not* mean that this person discovered or invented the interlocked finger position. He could, however, publish books on the subject and hope that people would, from then on, refer to themselves as doing the popular "Wilson" or possibly the "MacGruder" or the "Spongworth-Hugeusson" Technique whenever they interlocked their fingers.

I suspect that most of the exciting and new techniques that are flooding the field of light-touch therapy are, in truth, nothing more than the normal, intuitive touching and responding that

emotionally healthy humans can do automatically. I am certain that if we modern humans, yes, even doctors, spent more time practicing touching in an intuitional, healing manner, the way that most of us rub, pat, and hold our pets, we would realize there is nothing new about the “miraculous” new healing techniques that recently are being expounded. Not only that, I suspect that we all would intuitively know when and how to do them.

However, since we tend to touch very little and feel even less, our generation of health practitioners finds itself in the ironic position of having to take extra classes to learn how to touch, how to feel, and how to support with our hands. Most of us have never learned how to touch and hold our patients’ injured or insulted body parts. Most of us do not even know how to recognize those maladies of our patients that might best be treated by some type of hands-on therapy.

Happily, many of the researchers who are experimenting in this field are doing a brilliant job of writing about those techniques that work for them and publishing case studies. Writing about this realm of light-touch therapy can be challenging: it can be as difficult to describe in words just what a touch technique should feel like as it is to describe in words the flavor of an orange.

Also, many of the people who are promulgating these new techniques are working hard to make their writing available for free or at low cost. Some have done an excellent job of promoting light-touch work, offering frequent workshops and promotional material to “get the word out.” Others have worked hard to explain, using terms from biology, physics, metaphysics or by making up new words, just how these therapies work. I do not think I am exaggerating the numbers if I say that millions of people who have not responded to conventional (allopathic) medicine have benefited from some of the new light-touch therapies. This is an exciting time to be practicing “new” medicine.¹

Forceless, Spontaneous Release, a type of Yin Tui Na

Despite my general resistance to using proprietary, trademarked names for modern, light-touch techniques that are, essentially, nothing more than directed holding, the techniques we use for treating PDers now have a name attached. I actually have grown to like the name used for this technique, mainly because it is literally descriptive of what to do and what happens when you do it.

This technique, as the name implies, uses no *detectable* (by the patient) force in the hands of the practitioner. The practitioner may actually be using a fair amount of energy, but the patient should not be aware that there are forces at play other than secure support. The practitioner uses as much force as is necessary to support the part of the patient’s body that he is working on. The patient, however, should not feel as if any part of his body is being subjected to a directional force. Also, there should be *no* healing thoughts being directed at or imposed on the patient by the practitioner.

As for the “spontaneous release,” there is not an agenda or specific protocol for setting a release in motion: the responses of the patient determine entirely where the practitioner will put his hands, what he will do with them, and for how long. If and when the patient’s body decides

¹ A study undertaken in the mid 1990s revealed, much to the astonishment of the allopathic medical world, that one third of the people in the US had used “non-traditional” medicine. The alarming thing was that a majority of these people had never told their doctors for fear that their doctors would respond with anger. Of all the “alternative” modalities, acupuncture is the one most requested from people seeking alternative medicine coverage from their health insurance companies.

that, in response to being held or having been held a few days or weeks ago, it now feels safe enough to let go of some micro or macro muscle tensions, start processing the associated trauma, and/or restore displaced tissues back to their correct position, it will do so on its own. The practitioner cannot determine when these healing changes will occur, nor does he try to accelerate them beyond their own natural pace.

Releases will occur when the patient is darned good and ready, and not sooner. Although releases often occur during the FSR treatment, it is not uncommon for releases to occur later on, hours or days after the therapy session has long since ended. Therefore, the releases that occur give the impression that they have occurred spontaneously. Because the practitioner's job is to give intention-free support that feels forceless to the patient, and the patient's body will spontaneously respond in its own good time by surrendering up or releasing the retained mental and physical energy of the insult/injury, this technique is well suited to the name Forceless, Spontaneous Release, or FSR.

The next chapter explains how to perform this technique.

A CHAPTER APPENDAGE: THE HISTORY OF FSR

I am asked so often about the origins of FSR that I am going to tell that story here. If you are in a hurry to get to the more practical aspects of the Yin Tui Na technique that we use in treating Parkinson's disease, you can skip this section. But if you don't understand what I mean by "support, support, support," you may wish to read this bit. I will keep it short.

Shinzo Fujimaki

When I was getting my master's degree in Traditional Chinese medicine, classes in Tui Na/massage were required. I was fortunate to have classes with one of the most brilliant "massage" therapists in, I think, the world. I put the word massage in quotes because the class that Shinzo Fujimaki Sensei was teaching was officially titled "Shiatsu Massage." However, what he taught us was nothing like the usual acupuncture prodding that is normally associated with Shiatsu.

Master Fujimaki was so famous for his massage therapy that his appointment calendar was always booked at least three months in advance. His clients testified that his treatments had removed chronic pain, tumors, cancers, asthma, and a long list of other ailments.

As a teacher, he worked very hard to convey to us the essence of what he was doing. A majority of my fellow students did not like his class. Their complaint usually ran something like: "He is wasting our time telling us about his ideas. I don't get it. I just want to learn where to push to cure which problems. Fujimaki never tells us anything we can use." A few students, myself included, considered our classes with Master Fujimaki to be some of the most important foundation-stone hours of our entire school career.

Shinzo Fujimaki was a man with a radiant smile. He was also an aikido master. His energetic style of walk evoked images of tigers and horses. To best honor what he taught, I will simply quote to you, as closely as I remember, the words he told us, over and over. It will be up to you, as it was left up to us, to see if you can find anything helpful in his words.

Support, support, support

"Support, support, support.

"If a patient is lying on the table, and you push down hard on them giving acupuncture or massage, or push hard when you are feeling for the right place to put the hand or the needle, his

body will automatically push back against you. There will be a fight going on. How can a person relax, how can he begin to heal, when he is fighting? If the patient is lying on his stomach, do not push his back down into the table. Instead, put one of your hands under his chest and your other hand on top of his back. Position the upper hand directly over your hand that is underneath. Now when you push on his back with your upper hand, resist that push with the hand that is underneath. That way, you are doing all the work; you are doing the pushing and the resisting. Your bottom hand is supporting the patient, holding him strong against your push. Support, support, support. You give the support; then the patient doesn't have to work at resisting you or work at supporting the weight of your hand. The patient can be peaceful, he doesn't need to resist you; you are resisting yourself with your opposite hand.

"The patient cannot relax if you are pushing or poking him. If your goal is to allow the patient to relax so that he can let go of his problem, do not hurt him. Give him support. Support, support, support.

"If you are going to have one hand on [on some body part of the patient], your other hand should be on the other side [of the body part], catching the power of your first hand, protecting the patient from your active hand. If you are not doing any pushing, if you are just resting your hand on a patient, still, his body will have to worry what to do about your hand. His body will be pushing back on your hand, especially if you are touching a part of his body that is scared.

"But if you support the patient by putting your other hand on the opposite side of his body [part] to support the patient, and use that other hand to catch the energy from the first hand, then the patient can relax. Sometimes both hands are active and both hands are supporting. It doesn't matter. The only thing is this: the patient should not have to do extra work because you are there. The patient should be allowed to relax. Support, support, support."

Have fun

The master continues: "My attitude when I am giving treatment is that I am having fun. I learned that I gave best treatments after I had already worked about eight hours. After working eight hours without a break, I start to feel hungry, tired. I cannot stay focused on my work even if I try. I begin to think that I cannot survive if I don't stop working. My mind becomes distracted from my work. I want so much to stop working that I cannot think about what I am doing. To keep myself going, I imagine that I am looking up at the blue sky. I imagine that I am at the beach.

"I love to go to the beach. When I go to the beach, I imagine that I am a red horse, a red pony, and I run in and out of the waves. When I am finished running in and out of the waves, I lay on the sand and look up into the blue sky.

"When I am starting to get so tired from treating clients, after about eight hours, but I can't stop because there are still more clients with appointments for several more hours, here is what I do: I think that I am lying on the beach, looking at the sky. I have learned that during this time, when I am exhausted and looking at the sky, when the sky exists and the patient is no longer the center of my focus, this is when I begin to give good treatments. After a few more hours of still working hard giving treatments, when I am *in* the sky, when I *am* the sky, when the patient doesn't even exist anymore, then I am starting to do the best treatments. I learned this.

"So now, whenever I am working, I put my mind on the idea that I have been working eight hours already. I think that I can no longer keep going. I must start to imagine that I am looking into the blue sky. I must work very hard at it because my idea is that I am so completely

drained, I am so tired, I cannot think anymore about the patient. I can only survive if I am, in my mind, looking up at the sky with all my love and energy.”

Shinzo-san often worked twelve and thirteen hour days without taking a break. His point, however, was *not* that he gave his best treatments at the end of a long day. His point was that he had learned that, no matter whether he was just starting his day or was starting on his twelfth client, his mind must always be as desperately seeking transcendent joy as a drowning man seeks for air. When he could hold his mind in this state, the treatments – no matter when they were scheduled – more or less took care of themselves. Meanwhile, what were his hands actually doing? Support, support, support.

Where was he placing his hands? Very often he would start with the hands on the part of the patient’s body that was having pain. But just as often, as he gently pushed, vigorously pushed, or let his hands rest on the patient’s skin, his hands would move, with almost no thought or motive, to some other part of the patient’s body that seemed to want to be held, pushed, or prodded. The patient never felt pushed or prodded, however. The patient usually didn’t feel much of anything, except safety and relaxation, because the actual work of Shinzo-san’s hands was somewhat undetectable to the patient’s reflexive tendency to push back. Why? The support, support, support that his hands were giving each other.

Control your thoughts

Another point that Master Fujimaki made was also very important, although I think most of my fellow students only thought that he was relating a funny story.

“In Japan, we have a massage tradition that the patient leaves his clothes on. When I first came to this country, I was surprised that people remove their clothes for massage therapy. I was not used to working on bare skin.

“After I had been working in this country for about a month, I felt very bad about the way that my American patients behaved towards me. After every treatment that I gave, *every* treatment, the patient told me that he wanted to have sex with me. I thought that this was very bad. Young men, young women, old men, old women, they were all the same. After the massage, they all wanted to talk about having sex with me.

“One day I decided to learn why this was happening to me. I realized that I had a cultural difference about bare skin. To me, because of my Japanese background, bare skin suggested having sex. I must have been conveying my cultural ideas to the patients. So I made an effort to understand that in this country, bare skin was not a statement about having sex. I never again made this wrong idea about bare skin during massage. Ever since that day, when I changed my attitude towards bare skin, not once after a treatment has finished has a patient wanted to talk about having sex with me, not once.

“When my mind was on sex, every patient thought about sex. Now I think about the red pony and the blue sky, and my patients think about whatever they want; and they recover from their pain and their sadness.”

I could write volumes about this “Shiatsu” class that taught us nothing about classic shiatsu. However, I think the above examples make the two points most important to our work with Parkinson’s patients. First, the patient must be supported. No matter how much or how little energy the health practitioner is applying to the patient’s body, the patient should not feel the need to fight back or resist any of it. The patient should not need to push back unless he, for some reason, wants to. The support, support, support that Shinzo-san insisted on created a

pressure-free, supportive environment for the patient's body, as if the therapy, no matter how vigorous or how firm, was somehow forceless.

The other important point is that the mental sojournings of the practitioner are important. The best results occur when the practitioner is not trying to give undue influence to the patient. If the practitioner's mind is focused on something, the patient can pick up on it and even misinterpret it. Even focusing on healing the patient is usually inappropriate: if the practitioner is focusing on healing the patient and the patient is holding back for some reason, an unspoken conflict ensues. In the throes of this conflict, the patient cannot let himself go, he cannot relax. The patient cannot attend to the business of healing if he is busy fighting the practitioner or defending himself, however silently and invisibly.¹

But when the practitioner forgets about trying to heal the patient and plunges himself headlong into his own joy or inner peacefulness, the patient is less threatened. The patient can let his guard down. When this happens, the patient's body may very well start doing what it was designed to do: heal itself.

My 1989 class was the last group to have Shinzo Fujimaki as a teacher. The school administration, after receiving too many complaints that: "Shinzo doesn't teach us anything real," replaced him with a teacher who told the students, right out of the texts, where to push and how hard.²

Dr. Paul Lee

Fulfilling another course assignment, I was privileged to take a class in Medical Qi Gong from a Qi Gong Master. Dr Paul Lee, recently arrived from China, taught a brilliant class in which he taught us very specific techniques that patients could perform on themselves to stimulate their own healing energy in various body parts.

By way of introduction to Dr. Paul Lee, I will describe one of his projects. His work in China on self-applied eye massage had been adopted by the national government and was being taught to Chinese school children. The government had wanted a solution to the problem of poor vision becoming rampant among children at the seventh and eighth grade level. As students were doing increasing levels of book-work, they were starting to need glasses. This is considered perfectly normal in the west, but in China, where the government is the supplier of eye exams and eyeglasses, this trend towards "student's myopia" was considered a problem.

Dr. Paul Lee had devised a quick and easy program of Qi Gong (energy control) that included gentle eye socket massage and using the energized palms of the hands to push and pull energy into and out of the eyes. Starting in sixth grade, students did these quick exercises every day at school. They subsequently did not develop myopia and did not need glasses, even as they progressed through the later school years.

¹ When I wrote this sentence just now, I realized that it sums up, very well, the problem that Parkinson's patients are dealing with. *A person cannot relax and cannot let go if he is busy defending himself, however silently and invisibly.* Keep this phrase in mind as you work on your PD patients.

² This material was redundant; as second- or third-year acupuncture students, we already knew all the point locations and their applications. The replacement Shiatsu teacher simply demonstrated that these points could be stimulated by hand as well as via needles, and spent the whole semester doing it. I suspect the reason that most students liked this format was that they didn't have to learn anything new. They could spend the class practicing acupressure on points they'd already studied.

This type of Qi Gong exercise, in which the patient learns how to focus on a body part and move energy through it in a soothing, healing manner, is the essence of Medical Qi Gong.¹

This class taught me crucial lessons in the role that the patient plays in healing himself. If I could summarize the essence of this class, it would be this: the best doctor is one who sees where or what the source of the problem actually is, and then shares helpful information, even including specific exercises, to help the patient to change himself. The good doctor may advise on diet, exercise regimen, movement patterns, or instruct the patient in how to recognize where energy is moving incorrectly and how to correct it. The point of the treatment is to help the patient learn what he was doing wrong that made him susceptible to the illness, and how to

¹ Some regrettably deluded students have embraced a recent import from Asia, a version of “medical Qi Gong” in which the doctor uses his own energetic power to force healing onto a patient. While this may sound appealing to some people, this powerful work does not improve a person’s health in the long run. A person who allows his body to be manipulated in this manner actually suffers a weakening of his own will power and sense of energetic direction.

When the treated malady returns (and it will, sooner or later), the patient will be even less able to activate his innate healing energy than he was before. His body will passively wait for the next blast of healing energy from the healer rather than doing its own work. This type of healing, in which a charismatic person refers to himself as a Healer and forces the energy in a patient’s body to move in an unnatural (not according to the patient’s will) manner, is considered very bad form by traditional Qi Gong practitioners. This type of work can be dangerous to the ego of the practitioner and does no long-term good to the patient.

Great souls from time immemorial have done miraculous healing work. However, these souls performed their healings by removing first the causal (ideational) problem that set in motion the unhealthy energetics: the unhealthy energetics that manifest as the illness. Therefore, these great souls actually do remove the entire illness. More importantly, they only perform these miraculous healings when their cosmos-attuned intuition tells them to do so. They have no vested interest in whether the person heals or not. For the most part, if they have a preference, they prefer that others seek the Truth and Love that will enable them, the patients themselves, to cast out their own demons instead of passively waiting to be healed.

Patanjali, a contemporary of Socrates and one of the greatest Hindu writers on religious philosophy, makes his point in his Yoga Sutras. He explains that a sign of spiritual advancement is the ability to remove illness, including the underlying wrong thinking and past karma that caused the illness. But he also makes the point that a truly advanced soul may have this ability and, because of his wisdom, will choose the more difficult path: not using his spiritual powers to force an alteration in a person’s chosen life direction. Except in rare cases when commanded by God, the truly great soul understands the roles that sickness and health play in this worldly drama of cause and effect.

However, some modern medical Qi Gong practitioners ignore this wisdom from the past. These well-meaning people, finding that they have the ability to temporarily alter a sick person’s energy, go ahead and do so, imagining themselves to be spiritual healers. Even worse than the inevitable return of the illness in the original patient, these would-be healers often become deeply sick themselves despite their magic mantras, dramatic hand gestures, and bowls or gimcracks for “catching the bad energy.” If this type of Medical Qi Gong healer does get sick, then when his “healed” patient’s problem inevitably resumes, there are then two people sick with the same malady. From a larger standpoint, the world is worse off than before. Even if they do not get sick, these would-be healers are perpetrating the false idea that they, and not the patient, are the driving component of the healing process.

Only a Self-realized master can truly remove from the cosmos, through exercising his will in accordance with Divine instruction, the wrong energetics in another person’s body, mind, and heart. However, each one of us has the right and the ability (usually undeveloped) to instantly or gradually heal ourselves from the results of our own wrong thinking, the wrong thinking that is our own source of our emotional, mental, and physical health problems.

In the new testament of the Bible, Jesus celebrated a teaching moment when he pointed out, insistently, that he was not responsible for the healing of the woman who clutched at his robe and was instantly healed. He emphasized that she, and not he, had worked the miracle. The miracle came about through her faith, through the change in her thinking as she willingly tapped into the Love that Jesus personified. Jesus was trying to make the point that all of us have within ourselves the capacity for “miraculous” healing.

correct it. The burden of recovering and staying recovered is on the patient. The job of the doctor is to non-judgmentally, in a kindly manner, figure out the source of the problems in the patient and suggest to the patient a direction that will reverse the problem. The goal is relieving patient suffering through patient education and empowerment, which will usually include the patient learning some energetic (Qi Gong) exercises. A further outcome is the confidence and positive attitude the patient develops as he learns how he can confront his own weaknesses and change them.

Techniques used on PDers

The many classes that I took in Asian bodywork, including some of the teaching in the Medical Qi Gong classes, all contributed to my understanding of Tui Na. Some of the techniques I learned had names, some did not. The result of taking these many classes, combined with the other college classes, including five semesters of Asian medical theory, was that, by the time I graduated with a degree in traditional Asian medicine, I had learned, at least on a beginner's level, how to use my hands in a supportive manner. When I got my license and started practicing medicine, if I did include Tui Na in the treatment session, I never bothered to mentally define which, if any, particular technique I was using on a given patient at any given moment of hands-on therapy. Everything I was doing was the sum of all the things I had learned. I imagine that this is true for all bodyworkers. Although a good hands-on therapist may study dozens of named techniques, when he actually starts working, he will do whatever combination of techniques seems to be appropriate at the time for that particular patient.

When I started working with Parkinson's patients, I used very Yin techniques of Tui Na to both assess their physiology and to treat it.

Early on in my research, a Chinese colleague gave me Chinese words to describe what I was doing, words that meant not using force, letting the patient let go of the problems by himself. These words were descriptive of the Tui Na that I was doing. If people asked what I was doing and wanted a Chinese description of the technique, I could use those words.

Putting the Parkinson's Tui Na techniques into writing

When I wrote my first article for publication in the *American Journal of Acupuncture*, the editor had a problem with my description of the techniques I was using. The article was about Parkinson's; it was not the time or the place for a lengthy discussion of theory and description of techniques. On the other hand, I couldn't just say that I was doing Tui Na, because Tui Na covers ground all the way from pummeling someone's back to holding someone's hand. She needed a quick description of the type of Tui Na that I was doing. I told her it was Yin Tui Na. How Yin? Well, so Yin that the patient doesn't feel as if anything is happening, and when the problem areas release, they just spontaneously do it on their own.

The editor paraphrased by stating that the Tui Na was a forceless, spontaneous release type of Tui Na. I concurred. So she had me refer to the Tui Na I was using as a forceless, spontaneous release technique. She and I also agreed that I could refer to it in the article as a Yin form of Tui Na, and include a few details about the techniques to make it very clear that the work was forceless and not directed at any particular response from the patient: if, how, and when the patient responded, it would be some sort of spontaneous healing event on the part of the patient, not a change in response to anything *actively directed* by the practitioner.

The intent of the editor was not to create a trademarked technique. Nor was there an intention of implying that I had ever learned a specific, rarified technique of this name, passed

secretly from master to master, through the ages. The editor and I were merely looking for a way to describe, as clearly as possible, exactly what it was I was doing. What I was doing was a Yin type of Tui Na, one that was pretty much forceless and intention-free, and which resulted in patients having some sort of release whenever they were ready to do it.

Again, I did not invent this technique. I learned everything I know from my teachers. However, they did not always have a name for everything they taught.¹ A mere two years after that first article was published, enough people were using the phrase Forceless, Spontaneous Release Tui Na that the descriptive name used in the journal article had become a free standing name. I heard people referring to FSR as if it was some sort of “official” technique.

By the time I web-published the fifth edition of *Recovery from Parkinson's* in 2000, even I was referring to the various techniques used on PDers as FSR. Now, seven years after that first article was published, when I hear other people saying the phrases “Forceless, Spontaneous Release” and “FSR,” my mind’s eye sees the capital letters where there used to just be plain old adjectives. Somehow, this technique has turned into yet another named therapy! And yet, as you read on, you will realize that this is not a mysterious therapy from the misty past or the distant shores of Asia, but a basic method of using hands to work with an injured person. FSR is not a specific, exacting technique. FSR is just a way of providing support, support, support.

For the die-hards, even more information about Tui Na

Because I am frequently asked questions about Tui Na, and because there is not much available yet in the bookstores, I am including the following interesting tidbits about Tui Na, what the words actually mean, and a bit of history of Tui Na. If your interest is only in treating Parkinson’s disease, you can certainly skip this section and not feel the loss.

Asking for a better definition

While I was still in medical school I asked the Chinese doctors and teachers at my California acupuncture college for their definitions of Tui Na. My teachers were all practicing acupuncturists. One teacher, an MD in pediatrics from Shanghai, said, “Tui Na means pediatric finger massage.” An MD and Ph.D. in Chinese medicine from Guangdong said, “It means all forms of Chinese massage.” An MD from southern China said, “It cannot be translated. Tui Na means Tui Na.” An MD from Shanghai said, “It means bone medicine.” Another MD from Shanghai said, “It means bone massage.”

Sue, who was an accountant in southern China and now runs a restaurant in California, gave this non-medical translation: “Tui Na is a doing word, it is a word that means you do something, and then there is a result. It means moving, doing, and then it brings something out that wasn’t there before. So then you have something. Because you did something, this way.” She moved her hands in a slow, open and shut, back and forth pattern to demonstrate.

¹ I sometimes think my role in this Parkinson’s project, and my role as an acupuncturist, isn’t one of discovering new things, but that of putting English names on things that already exist and might even be intuitively understood in Asia but which, because they are, in English, nameless, or not described in technical terms, are not accessible to us in the west. I honestly feel that I personally have never discovered or invented anything new, but I have certainly put a lot of English words together while working on this project.

For that matter, and let me get this off my chest, even if, when I started working on this project, there was no known treatment for Parkinson’s disease, this lack was only due to our failure to understand the disease. The cure for this illness was always floating about in the ether. The cure was always simply this: reverse the problem that causes the illness. I may have tapped into an answer or a solution, but I never invented it. My only accomplishment, that I can see, is that I have written up what I have seen. And I did it with the help of hundreds of people.

*...Bearing thy heart, which I will keep so chary; as tender nurse her babe from
faring ill.*

- William Shakespeare, sonnet XXII

CHAPTER THIRTY-SIX

FORCELESS SPONTANEOUS RELEASE, A FORM OF YIN TUI NA

INTRODUCTION TO THE SECTION ON TECHNIQUE

This chapter will provide instruction in performing Forceless, Spontaneous Release therapy, or FSR. The next chapter will guide you through a very specific practice session in which these techniques are applied to legs, ankles, and feet. The chapter after that will offer an explanation of how FSR works. Several chapters that explain the very still, resting variation of FSR that we use on Parkinson's patients follow these chapters on FSR.

This chapter will start by explaining why we have to learn and use both the standard FSR and the Parkinson's variation.

Comparing the technique of FSR and the Parkinson's variation

The technique of Forceless, Spontaneous Release, FSR, is extremely simple. It consists of holding a body part between two hands in such a supportive way that the patient doesn't really notice that he is being held. Undetectable, even purely mental nudges in one direction or another may be performed, if necessary, to determine whether or not the area being held is capable of reflexively responding to either the support or the minute suggestions of movement. The hands are removed after the body part being held *seems to relax or otherwise responds in some way*.

This technique is different from the FSR variation that will be used in the locations of the more stubborn injuries that are seen in PDers: with certain PD-initiating injuries, no matter what you do, how correct your pressure is or how long you wait, certain injured areas, particularly certain areas in the feet, will not respond for a long, long time: maybe months – certainly not minutes.

Because the injured feet of people with Parkinson's do not respond to touch in the normal manner that is seen in FSR, we need to use a variation of normal FSR technique when we work with Parkinson's patients. This variation, referred to as "resting FSR," consists of assuming the same type of supportive position with the hands that is performed in normal FSR, but then keeping the hands as still as possible, remaining fairly motionless for as long as necessary (usually the length of the session), while actively working to keep one's (the practitioner's) mind uninvolved in whatever the patient's injury might be doing. There is a bit more to it than this, but for now, this description of resting FSR will be enough to differentiate it from the regular, more diagnostic type of ordinary FSR.

Both “normal” FSR and the PD variant are used in treating PD

FSR is a diagnostic tool as well as a treatment tool. We use FSR to determine where the PDer needs the resting variation to be performed and to assess whether or not the area is starting to change. A PD therapist must first learn how to perform FSR, and then the PD variation: resting FSR.

FSR as a diagnostic tool

Normal people have an automatic, reflexive response to being touched or held. The injured areas of PDer's, in stark, clearly perceptible contrast, do not have a normal, automatic response to being held. When, by practicing FSR, a practitioner learns to quickly recognize what the normal range of responses of a healthy person feel like, he will be able to recognize the pathological response that he gets when he homes in on the injured area(s) of a PDer.

Another reason for learning FSR is that, eventually, as the PDer starts to improve, the fairly Yin FSR technique may appropriately supplant the extremely Yin PD variation in order to do the fine-tuning.

Referring to a health technique as both a diagnostic tool and a therapy is somewhat uncommon in the western medical realm. However, when applying assessment methods that indicate whether or not a person can pay attention to a given part of his body, the very process of assessment can be an attention-garnering act. As the body brings its attention or curiosity to an area, it may also bring healing capability to the area: the assessment becomes the therapy.¹

In order to discover which areas of a PDer's legs and feet do not respond with a normal reflexive movement, FSR is first used on the legs and feet of PDer's. When the practitioner comes to an area of the foot or leg (and later on, possibly an arm, hip, shoulder, neck, or cranial bone) that does not respond normally, this non-responsive area is where the practitioner will begin

¹ Years ago, when I was studying Zero Balancing, a very Yin type of body stretching, the teacher kept saying “Gently move the patient's foot (or neck or whatever) in direction X or Y and then assess what happens.” Over the course of two days, I got increasingly antsy with the “assessment” process. I was eager to find out what technique we would do on the areas that had been “assessed” as needing more work.

At the end of the two-day workshop, it suddenly occurred to me that the actual work of Zero Balancing was the moving of the patient: the assessment process was the technique. The verb “assess” had been used, very wisely, by the originator of Zero Balancing to prevent students from thinking that the imposed movements were supposed to do anything to the patient. By asking practitioners to “assess” what happened when moving the patient, the practitioners very carefully tuned in with what was going on in the patient's body, but didn't try to actively do anything. The benefit that was observed by patients was “spontaneous:” it occurred on its own, while the practitioners were very gently moving the patient around, trying to make an assessment.

I realized, several years later, that a major challenge for founders of various schools of light touch movement comes in writing up the instructional material. If the founders use verbs that imply any sense of doing on the part of the practitioner, most of the students will completely misunderstand and use some, and therefore, too much, force. On the other hand, if the writer says “assess” or “apply a few grams of pressure” (a gram being less pressure than most humans can even feel – a gram of pressure is less than the weight that a dime imposes on a tabletop), the student correctly goes about his work of touching in a non-forceful manner. Despite, or because of, the lack of force involved, the patient responds in a beneficial way. Therefore, the assessment and the therapeutic work are very often one and the same.

The best analogy would be that a mother's kiss works best if she first really focuses on the child's little injury, looks at it, clucks her tongue, and then gives the spot a kiss: assessment and gentle treatment. If a mother merely blows a distracted kiss to the crying child from across the room and hollers, “Don't worry; you're still alive! Ha, ha, ha!” a therapeutic benefit will probably not occur. The careful assessment by the mother, drawing the child's own loving and attention to the area, is a crucial part of the healing work.

doing the PD variation on FSR. In comparison to normal FSR, this variation is extremely motionless and intention-free: extremely yin.

Setting standards for diagnostic tools: determining what is normal

One requirement for using a touch technique as a diagnostic tool is familiarity with what constitutes a “healthy” response. In my weekend seminar experiences, I learned from those practitioners who had already been working on PDer, using only text as their FSR learning tool, that most of these practitioners had never bothered to practice first on healthy people. They also were the most baffled as to why their patients were not improving, considering that, according to these practitioners, their patients’ blockages were all gone.

Ignoring the repeated textual admonitions that these techniques should be first practiced on healthy people, these practitioners had assured themselves that the tiny, random, small electrical cellular responses coming from their PDer were “normal” tissue responses. Concluding, based on this wrong, presumed evidence of “movement,” that their patients’ blockages must be gone, the therapists had gone on to prematurely and/or unnecessarily use acupuncture needles or physical therapy on people who still had blocked injuries and backwards-running Qi. These practitioners then wondered why their patient was not getting better. In fact, their patients were *not* yet making normal responses to touch. They still needed Tui Na therapy. But the therapists didn’t know what was a healthy and normal response and what wasn’t.¹

If a person has no training in FSR, he may not realize if a PDer is having a normal response or not. Therefore, I will state as forcefully as possible: the following techniques should be practiced on several healthy people before they are used on PDer. After practicing these techniques on several or dozens of healthy people, a therapist may have enough sense of the normal range of healthy response that, when he comes across a strangely unresponsive area on his PDer, he will know immediately that there is something deeply wrong. He will also know when the injured area is starting to feel healthier, closer to normal.

FSR TECHNIQUE

Positioning the hands

The following is directed to the health practitioner who is learning FSR. The word “you” in the following section refers to the FSR student.

Place the palm side of your hand flush against some part of the forearm of your learning partner. Then place your other hand on the opposite side of the forearm. (See fig. 13.1.)

¹ In many training classes, I have had a student or two who had thought his long-time PD patient was responding fabulously because he thought he could detect subtle movement deep inside the skin. In the classroom setting, when these students begin working on healthy fellow students, they are astonished. I remember one exclaiming to the room, “Oh my gosh! Is this what a normal person responds like? My PDer doesn’t feel anything like this!” This same practitioner had been writing to me for months telling me about her FSR progress with her PDer. She had said that her PDer was responding beautifully to her touch, and yet wasn’t seeing a change in her PD symptoms. When this practitioner exclaimed this way, I asked her point blank if she had ever tried these techniques on a healthy person (my previous editions made the point very strongly that one should practice first on several healthy people). She told me, “No, I assumed I didn’t need to. I’m a licensed acupuncturist, and a massage therapist. I had thought I knew what it felt like to touch a person.”

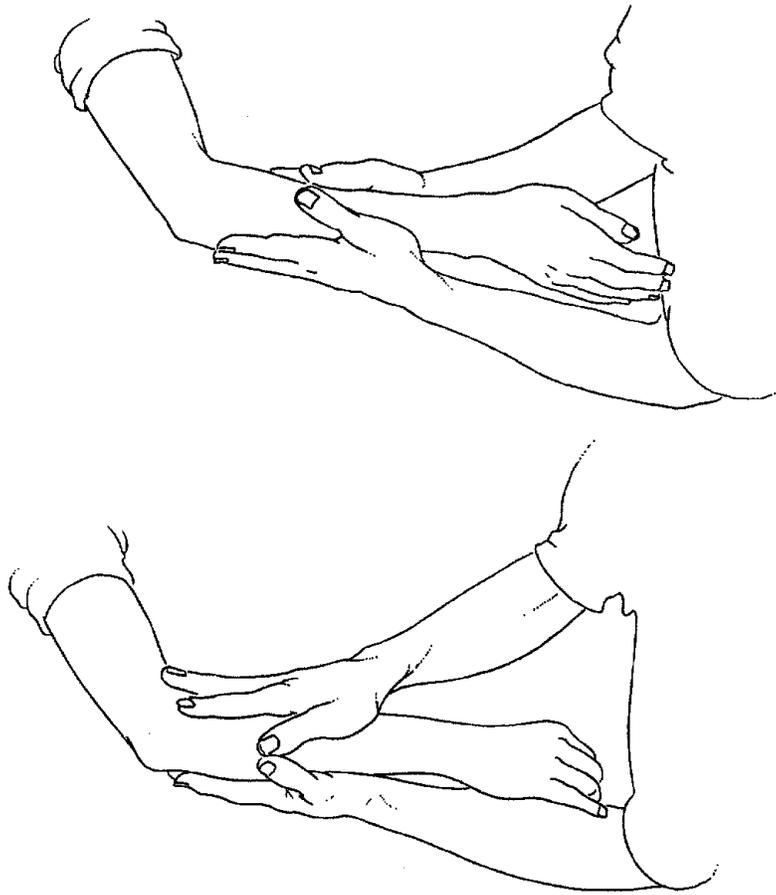


Fig. 36.1

Two examples of placing your hands on opposite sides of a body part. It does not matter exactly in which direction you are holding. The important thing is that your two hands are more or less opposite each other.

Your own physical comfort is an important factor in deciding where your hands and arms need to be, both in relation to the rest of your body and in relation to the contours of the patient's arm. The exact location of your hands during this practice – and whenever you are doing FSR – is not critical. Your ability to be relaxed is much more important than the exact placement of your hands: you may need to sit fairly still for a long time. Slumping back in a soft chair may seem like a comfortable way to sit, but this type of “comfortable” is not easy to maintain for very long. You will be able to work longer and better if you can learn to sit upright, with good posture, while your arms suspend softly from the shoulder, and your hands have no tension at all.¹

¹ This footnote is directed to those people who worked with the 5th edition of this book. In that book, I wrote more details about what the practitioner should do during FSR. We have learned a lot during the last five years. One of the main things we learned was that the level of expertise and attunement with the patients' skin, legs, and bone doesn't matter nearly so much as we thought it did. We also learned that the role of the patient is much greater than we'd thought it was.

Pressure

The amount of pressure that your hands exert on the partner is very important. You should make fairly complete skin-to-skin contact without actually trying to push the partner's skin around. The goal is to hold the patient with such complete support that he cannot feel your hands. Because the completeness of the support is more important than the exact, precise location of where you are holding, it is important that you find a way to nestle your hands into the contours of your partner's forearm, even if such a holding position is not exactly the location of holding that you originally had in mind. When you have gotten your hands nestled into a place that feels comfy, your partner, curiously enough, will often volunteer that you have put your hands in "just the right place."

Now let's back up a bit and look more closely at what you are doing, looking at one hand at a time. Let's assume in the following explanation that you are putting one hand on the upper side of your patient's arm, and your other hand underneath his arm.

Upper hand

If you are sitting in a comfortable position, you will be able to let your upper hand drop gently from your shoulder and come to rest on the patient. The pressure of your upper hand should exactly equal the force that gravity is using to keep your hand resting on the patient. This is a fair amount of force, by the way. If you are using any muscles to prevent your hand from pushing too hard onto your partner, you are holding too lightly. Your hand should be resting like a dead weight, with the full weight of your hand plopped down on your partner.

If you aren't sure what I mean by "dead weight," you might want to abandon your partner for a moment and try this practice exercise: sit in an armless chair. Let your hand flop down onto your thigh. Let your hand just sit there, held in place by gravity. Don't push your hand into your thigh as if you were trying to leave an imprint of your hand: that would be too much pressure. Don't rest your hand gingerly, as if your thighs were sunburned: that would be not enough pressure. Let your shoulders relax and sag down. Let your hand rest heavily on your thigh. That is the exact correct amount of pressure for your top hand. This amount of force could be calculated mathematically as the force of gravity times the mass of your hand. Now, take this hand off your thigh and respectfully allow it to plop back down with the same degree of weight onto your practice partner's arm.

Lower hand

Use the exact same amount of pressure with the lower hand that you use with the upper hand.

If you want, you can abandon your partner again for a moment and practice holding your thigh again, but this time you will use both hands. Let your first hand flop down onto one side of your thigh, and place your other hand on the opposite side of your thigh. Imagine that your thigh is a mound of bread dough. Use as much pressure between the hands as you would need to use to keep the lifeless bread dough from dropping to the ground. Do not hold the limp "bread dough" gingerly; it will slip through your fingers. Spread your fingers apart so that the "bread dough"

If you first learned FSR from the old book, you will notice that this edition spends a lot more time on FSR technique, but has also eliminated some of the old material. This does not mean that the instruction is less complete. It means that we've figured out the critical elements and spent more time on those, and left out the things that turned out to be not important.

doesn't sag. Have a firm grip on the two sides of the "bread dough" of your thigh, but don't be leaving imprints of your fingers in your flesh.

Either one of your hands acting alone could not hold up the soft bread dough from the side. But the two hands, one on each side, can firmly (but gently, without squishing the dough) support the lump of dough if both hands press towards each other with just the force of gravity.

Try placing both hands on your partner's forearm again, using the same amount of support that you used to support your thigh.

More about how much pressure to use

Again, how much force should be coming from your two hands? You should use as much force as you need to make the patient feel perfectly supported. If you are holding with approximately the force of gravity, the patient will be able to stop fighting the force of gravity in that particular body part. This unconscious work of combating gravity (on the part of the patient) can cease. The patient can relax.

However, the actual body position of the patient will not have changed, and if your hands are merely supplanting a force that was already there, the net change in applied forces will be zero; the patient's body soon will be unable to tell that anything like force is being applied by your hands. The patient will feel as if, for all intents and purposes, your hands are not even there.

Again, do not hold gently; nothing can be more annoying. Get a nice, solid, supportive hand position on the area that you are going to work on. Place your other hand opposite the first one and have the two hands together hold the body part with such support that even if the table on which your partner is resting was to be pulled away, your holding would prevent any falling down of that part of the patient that you happen to be holding.

Don't be trying to physically *or mentally* manipulate the limb you are holding. At this stage, when you are practicing how to hold without undue physical or mental pressure, consider that any intention on your part for the good of the patient is a form of psychological pressure. So don't be imagining any particular outcome as a result of your support. Have no intention in mind for how the practice partner should respond.

Pressure without intention: the harried mother example

I frequently use the following example to demonstrate what I mean by solid support without intention.

Picture the scene: a harried mother is trying to cook dinner. She is standing at the stove, stirring food in two pots. One pot has almost come to a boil and needs to be watched closely. The other pot is bubbling away and needs frequent stirring. Just out of arm's reach, her four-year old child is pestering her two-year old child. The younger child is just starting to scream in frustration. The mother cannot reach them because she is stirring the dinner, plus she is talking to her friend on the phone; the phone is the old fashioned kind, attached by a cord to the wall. She is not using her hand to hold the phone, the phone is cradled between her ear and her raised shoulder. She is alternating between telling the youngsters to stop fighting and trying to arrange a babysitting swap with her friend for tomorrow. Meanwhile, she is also holding a baby on her hip; it is not her own baby; Mother is, at this moment, babysitting for the other neighbor, who should be home shortly. Until that neighbor gets home, the mother has the neighbor's baby wedged up against her left hip and she has her left arm wrapped around the baby. Baby is stuck between the firm left hip and the snug left arm. Baby is in solid. Baby is going nowhere.

With her other arm, mother is now alternating between adding some spice to the dinner and stirring it. Mother is still on the phone, on hold, and is now pleading with her young children to stop fighting over the stuffed weasel that they are trying to tear in two.

Here is my question: who is the most contented person in the room?

If you guessed the neighbor's baby, you are absolutely right. The baby is looking around, taking it all in, reveling in the fact that he doesn't have any social interactions going on. Baby is being held so closely that he doesn't even notice that he is being held. Baby has such complete trust in that firm support coming from the hip and the embracing arm that baby does not notice the pressure from the mother's hip and arm. Also, the mother is not paying any attention to the baby with her eyes or words. The baby is physically relaxed and comfortable.

Of course, when the baby's mother returns, baby will probably go into his regular routine of crying or cooing at his own mother, doing all the things it has already learned to do to fulfill his mother's expectations. But while the baby is being held tightly on the neighbor-mother's hip with no one looking at him, no one cooing at him, no one expecting anything of him, he is able to take it all in with wide-eyed wonder, amusement or contentment, and his body will be physically at peace.

The way that the above mother is holding the baby, that's the way you hold a person with Parkinson's disease.

As a member of the PD Team often says, "The biggest mistake therapists make is that they hold the PDer as if he was *their* baby. You should hold the PDer as if he was *someone else's* baby."

Comfort for the practitioner

In the example above, the mother is doing whatever she needs to do to be comfortable. No doubt she has one hip swung way out to the side to support the baby. But the hip is not putting undue pressure on the baby. The pressure that exists in the baby-hip contact is the pressure from gravity acting on the baby. The hip is not trying to force itself onto the child to support the child. The hip is just there. What with gravity and the additional lateral support provided by the left arm, the baby is nice and snug. The amount of force that occurs in the hip-baby contact is the amount of force that your hands should be using on your partner/patient.

This amount of pressure should be comfortable for you. If you find that you are getting tired arms or sore hands, possibly you are using way too much pressure or your chair is not at the right height. If you are not relaxed, it will be hard to give perfect support to your patient.

Now you have mastered two parts of FSR. First, you know that the hands are opposite each other, supporting the body part in between. Secondly, you know to use just as much pressure as you would need to counteract gravity: enough pressure to provide support.

Practicing position and support

Practice the above positioning of the hands (one hand opposite the other) and applying just the right amount of force on a practice partner. Place your hands on your partner's forearm and experiment with positioning your hands until you find a pose that is very comfortable for you. Have your partner tell you if your hands feel too pushy, too light, or if they feel just right. Have your partner then try the same on you. Take turns seeing how it feels to hold someone's forearm. If you think you are comfortable with the forearm, try holding the upper arm. Try holding the partner's thigh or lower leg. Play with this. See what it feels like to hold supportively but without expectation, and how it feels to be held.

As you get more familiar with this type of holding, try pretending that your confidence level has increased to the point that, as soon as you set your hands on your practice partner, you are instantly applying just the right amount of pressure. In other words, you don't want to spend five minutes figuring out exactly how much pressure to be using. Practice resting your hands firmly and opposite to each other until you get to the point that you know, even before you set your hands on your partner, just how much pressure you will be wanting to use. From the moment you start to place your hands on your partner, do it confidently, with the correct amount of pressure.

Movement occurring in your partner in response to your holding

When you practice holding your practice partner's forearm, you will notice, eventually, that at the moment when you place your hands on your partner using exactly the correct amount of support and pressure, an immediate and perceptible change occurs in the position of those muscles in your partner which are immediately under your point of contact. This comes from the immediate, localized relaxation of the patient's body part in response to your touch. The change may even be visually perceptible to you (and your partner) because your hands, making firm contact, will perceptibly move even as your partner's forearm relaxes.

Because of your commitment to supportive contact on your partner's skin, when his skin/underlying muscles move, your hands must move along with him. Your hands may find themselves resting in a slightly different position than they were when they were very first placed on the partner's arm.

You might ask, "What if my partner is already relaxed? If so, he will not relax in response to being held." Don't worry. If your partner is fighting gravity, he is doing work and, therefore, is not perfectly relaxed. One can safely assume that all conscious patients/partners are not in a state of perfect relaxation and will relax somewhat in response to being supported.

When your hands are applied to his forearm (or any body part), your hands will supplant some of the patient's inherent tension; the touched area will relax its share of internal muscular grip accordingly. This small release of muscle tension will create a movement in the skin and underlying muscles, such that the practitioner's hands will find themselves resting on the patient in a slightly different position than when they began. This change might not be perceptible to the partner/patient if he has his eyes closed. Because he felt no vector of force being applied, the partner will most likely think that nothing has happened except that he briefly felt the contact from your hands. If the partner sees that your hands have moved in the first moment after you placed your hands on him, he will most likely assume that *you* have initiated some movement that caused your hands to move.

However, if your partner's eyes are closed during the time of contact and subsequent movement, he will probably not detect that anything has happened at all other than the fact that you are supporting his arm. However, you may have noticed, especially if the angles of your wrists and arms had to move in order to follow the movement of your hands, that your partner's skin, by relaxing, has carried your hands to a different position from your starting position.

Although you will have done nothing but support, something will have changed in the partner/patient. Sometimes enough relaxation can be inspired just through this type of brief holding that a significant release of tension or repositioning of displaced bone or tissue will occur. Sometimes – and this is the point – healing can begin to occur in an area that previously, due to tension, was resistant to healing.

This simple holding and the immediately subsequent following of the response with your hands are the basic events of FSR. Practice it on someone else. Practice it a lot.

Delicate touch, heavy touch

Though I risk redundancy, I repeat that touching, if done too lightly, is an irritant. Oppositely, when touching is done with too much pressure, it generates a pulling back response. The type of touching used in FSR is the confident, firm gentleness with which a mother holds someone else's sleeping child. FSR requires a supportive, a full hand touch which does not impose, but which conveys confidence and assurance.

More practice exercises for understanding position and pressure

Very possibly you understand exactly what I am talking about and are ready to move on to the next chapter. However, the most common requests I get are for more information about how much pressure to use or where to put the hands. The following is a rehash of the above. It may prove helpful.

“Forceless” touch

Most often, the beginner is far too delicate, employing an irritating, “gentle” touch. The problem is that he is trying to be “forceless.” Therefore, I repeat myself: the word “forceless” applies to the perception of the patient, not to the amount of pressure used by the practitioner.

We are surrounded by forces. We are unaware of most of them. Air is pushing against our skin at all times. Gravity is always exerting a force. Our skin is holding in the contents of our insides. Blood vessels are putting pressure on the blood. There are many acts of pressure and force at work on our bodies at all times. We cannot perceive these forces because we have become used to them. We can even become used to unnatural forces; when we wear clothes and shoes to which we have become accustomed, we don't actually feel them. Within a few moments of putting on a snug pair of old shoes, we have no awareness of the shoes pressing against our feet. This level of force, a force comparable to the perfect snugness of favorite shoes, is the level of force that you should use when supporting your patient.¹

Again, gentle touch is annoying. Full support, in which the patient feels completely safe and supported, is just the opposite of annoying; such support quickly becomes imperceptible to the patient. Again, the word “forceless” in the name of this technique does not describe the amount of pressure used by the practitioner. “Forceless” describes the amount of pressure that is perceived by the patient. The art is in learning to make contact that is firm but which is soon imperceptible to the patient's consciousness.

¹ One excellent FSR practitioner that I know says that, when he sits for an hour not moving, with his hands cradling a PDer's wounded foot, he feels like a human cast. Actually, that is a very good analogy. A plaster of paris or (more modern) plastic cast gives solid support, but it is rigid, cold and cannot conform perfectly to the changing contours of a live human. A cast made of human hands gives an even better level of support: it is warm and conforms more perfectly to the skin of the patient. I like the idea of a human cast. You may have noticed just now that I am suddenly talking about a practitioner sitting without moving for an hour, holding a person's foot as if he was a human cast. Consider this example to be foreshadowing of the variation on FSR technique that we use on the site of a PDer's injury. For now, talking about basic, diagnostic-type FSR, we are not sitting motionless for an hour with our hands in one position.

Skin contact: the pranam

This exercise might help you spend a little time playing with your hands and observing the forces at play.

Press your two hands softly together, palms touching, fingers touching, as in the "prayer" position, or Indian "pranam." Notice how much force you require to hold your hands together. Each hand is comfortably aware that the other hand is there, mutually supporting. Close your eyes and imagine that one of your hands is the skin surface of your patient, and the other hand is your hand, practicing FSR. Can you tell which is which? Did the pressure change as one hand took over the role of "practitioner"? Hopefully, the answer is "no." Notice how little energy is required for your hands to stay still, touching each other, making contact, but not exerting any force on each other?

Now, let one hand rotate a little bit so that your hands are in the "keep your hands folded in your lap" position. In this position, the thumbs cross each other and the distal ends of the fingers of each hand drape gently towards the back of the opposite hands. In this position, the palms of the hands can make even closer contact than they did in the pranam.

Hold your hands like this for a moment and notice that, even though your hands are now pressing on each other with a little more firmness and contact, they still don't really seem to you to be putting force on each other – even though they are. Again, it's hard to say which hand is pressing on which. The two hands are both doing the same to each other, making equal contact with each other. This firm equal-to-both-parties contact should be used while supporting and evaluating your patient.

The force of gravity

Let your hand and arm drop onto the table top or desk top. Do not hold your arm and hand up with your shoulder muscles. Let the full weight of your hand and arm collapse onto the supportive surface. Let your arm go limp from the shoulder. Feel how much dead weight your body is applying to the desk top or table top. This dead weight force is how much force you can have pushing down on your patient with your hand. You were not pushing on the desk top, were you? No. You were being passive. Passive can have a lot of force to it.

Combine contact and gravity. Rest your elbows on the table about a foot and a half apart. Let your hands press against each other again, other making firm contact, contact in which neither hand is pushing harder than the other. Now, let the two hands apply the same weight, the same force, onto each other that they put on to the desk top. With your elbows propping up your hands, your hands will hold themselves up against the weight of gravity. This should create a very nice, somewhat firm but very comfortable position. Your hands are not being gentle, they are being strong and firm. They are applying force, but it is just the natural force of gravity. Neither hand is pushing on the other. They are both resting on the other. This is the way your hands should settle on your patient.

More practicing with a partner

One hand

Remember to be comfortable. Both you and your practice partner should be seated on chairs. The partner rests a forearm on a nearby table. Set your hand down on his forearm. Do not press down. Just let gravity settle your hand snugly down onto his forearm. Use your full hand. Do not try to be "gentle."

Notice how it feels to be making relaxed, even contact with absolutely no pressure other than the weight of gravity. Continue to rest. You will notice that the amount of pressure you need to apply to keep your hand in place is exactly equal to the amount of upward pressure of your partner's arm resisting you.

Notice that when you set your hand on your partner's arm, your hand does not sink into his – it doesn't float through his arm and come out the other side! There is resistance coming from his arm that supports your hand. So you can relax your hand completely.

If you press down at all, using willful force, your partner's muscles will begin to exert a matching force back outward onto your hand. You don't want that to happen. Your partner's arm should be able to stay just as relaxed as if you weren't there. It won't be able to, of course. For your partner to actually stay relaxed, you are going to have to use your other hand to provide the support, support, support that was described in the last chapter. But we aren't quite there yet.

Ordinarily, air is pressing at all times on all sides of our bodies. Also, the skin of the arm is holding the insides of the arm together, exerting a slight force on the insides of the arm. In this exercise, your hand is taking the place of the air that ordinarily would be pressing down on your partner's forearm right at that spot, and becoming like a second skin. By becoming a second skin, your hand is also taking the place of some of the pressure that the partner's skin is exerting to hold all the arm contents inside the skin. Your hand should make nice, firm contact with their arm, just as air does. Air pushes firmly and equally against all surfaces of our bodies at all times, but we cannot notice it. We don't notice air pressure because we are used to it. We also don't notice the air pressing in on us because it is intention-neutral.

Another baby example

Have you ever tried to move a sleeping baby? Most often, if your movements are self-confident and direct, the baby can and will sleep right through almost anything. However, if you try to be oh-so-gentle, and whisper, and act as if the baby is the most fragile construct in the world, the baby is sure to wake up.

Your patient's injured body parts will respond favorably to confident and direct interaction because, if you are confident enough, he can't actually tell what your hands are doing. However, the patient's injured body part will know that it is able to relax more than it has in a long time. Oppositely, uncertainty or insufficient support will be detected at once, and spurned.

Two hands

Next, place both hands on opposite sides of your partner's forearm. (Review fig. 13.1.) Use the same amount of contact that you used with one hand, and apply it with both hands. Just rest there for a moment.

It does not matter exactly in which direction you are holding, whether side-to-side or top and bottom. The important thing is that your two hands are more or less opposite each other so that the partner can feel completely supported.

The second hand, just like the first hand, doesn't need to do anything. If the first hand is on the top of the partner's forearm and the other hand is under the forearm, holding the forearm so that it can't get away, that is just right. The second hand can be thought of as a passive receptacle for the weight of the first arm. By the same token, the first hand might feel like a passive receptacle for the force that the second hand is using. That is just right, too.

The patient's arm should be able to feel completely held up, supported by the lower arm and the upper arm. This doesn't mean that either hand is doing anything aggressive or overt.

They are not. The hands are simply preventing the patient's arm from giving in to gravity. The hands should provide as complete a support as possible, giving a nice base of support, while conveying the idea that they are not doing anything at all and they don't intend to do anything at all. Your hands are just there, and they are solidly there.

Letting Go

Now that you know how to hold on to your partner, you need to learn when to let go. The rule is: let go when the patient's skin tells you to let go.

Your patient's skin in the area where you are holding will do a microelectric shift when your support is no longer wanted. If you keep holding, the patient's entire body and soon his mind will also start sending you a message that it wants you to let go. However, if you are not used to observing these small but definite signals, you may want to practice the steps below.

Practice this exercise in knowing when to let go. Start by doing the holding exercise above one more time: place your hands on either side of your partner's arm.

This time, after following with your hands the movement of the partner's skin as he relaxes, notice that there is a tiny, momentary sensation of connectedness between your hands and the skin of the partner. It might feel to you as if your skin is being magnetically bonded to your partner's skin. Oppositely, your skin may, a moment or two later, sense that your partner's skin is pushing you away.

Holding skin is different from holding a book. There is a feeling, a slight feeling of something alive. (Do not practice this on a PDer. Much of a PDer's body might be lacking this feeling. You must learn to recognize these feelings on a healthy person.)

This tiny tingling has been compared to static electricity. One student hypothesized: "It's like when I made contact and the arm relaxed in response, the relaxation released not just tension, it also released the Qi that was holding onto the tension, and that Qi scattered all through the area and made static between everything."

This sensation of a static connection has been compared to the feeling that exists between two socks that have been tumble dried together and have become charged with static. They can be pulled apart, but the pulling will require a small amount of force: there is a perceptible attraction between the two socks. A similar, or smaller, level of attraction may be palpable between your hands and the skin of the partner if there has been a recent relaxation movement in response to your supportive touch.

Do not let go of your partner as long as you can feel that static, or tingling.

If you try to remove your hand before that Qi has dispersed, it will feel as if you are using a bit of force, as if you are wrenching your hand up off of the partner. It will feel somehow wrong. If you wait until the static has dispersed, your hand will come up easily off of your partner. If the static disperses and the skin actually reverses its charge, your hand may almost feel as if it is being subtly repelled away from the partner. If you feel as if your hand is being pushed away, then do not impose your hand a moment longer.

When should you let go? Do not let go as long as the feeling of electric attraction is ongoing. Do not let go if you feel as if your hands are being pulled in to the partner's skin. Do not let go if, when you try to remove your hands, you feel as if you are having to use force to extricate your hands. Do let go if the static or feeling of attraction has dispersed and you feel that you no longer need to leave your hands on your partner's skin. Do let go if you feel an electric sense like that of two positive ends of a magnet being pushed at each other, repulsing each other. Do let go if you feel uneasy in any way. Such a feeling of uneasiness may be coming from some

energetic turmoil that has been stirred up in your patient, and, if you don't want to be a party to it, that's perfectly reasonable. Of course, do let go if your partner verbally asks you to do so.

Electric resistance to being touched

Sometimes, a PDer will have such strong resistance to being touched, particularly in the vicinity of an injured area, that when you first begin working with him, you cannot actually rest your hands on him for the first few minutes of the treatment. Sometimes this palpable resistance to being touched can last for an hour, or even for weeks.

(When I have a patient with this level of fear around being touched, I just rest my hands in the air space several inches immediately above his injured body part. I support my hands with the muscles of my arms and shoulders, as if my hands were resting, nonchalantly, up against the electric field of his injured area that is emitting the "go away!" signal. Usually within a few minutes or a few weeks, the area is less afraid and allows me to set my hands down on the skin.)

When you are practicing holding, try to feel the sensation between your hands and the partner's skin. You will soon learn when it feels "right" and when it feels "wrong" to rest your hands on his skin and when it feels right to let go.

Sometimes it will seem a bit awkward at first if you are working on a patient and a full minute goes by before you get the signal to let go. More often, the static feeling will disperse quickly.

More advanced students notice that they are sometimes aware of a feeling of relaxation in their own hands, or even in their arms or torso, which occurs at the same instant that the static cling feeling goes away. As you become adept at this work, you may even begin to notice that your own body perceptibly relaxes at the same time as the partner's. Sometimes it is as if you were unintentionally holding your breath, and at the moment when the partner relaxes, you find yourself exhaling, or relaxing your abdomen.

So there are actually many cues: the static sensation, the attractive (holding) force, the repellent (letting go) force, the partner's relaxation, and even a feeling of relaxation somewhere deep within your own body. These are all signals telling you to either hang on or let go. You may notice one or several of them. When you feel anything that is telling you to let go, let go.

To be redundant, when the skin of the partner's arm is no longer clinging to your skin, it is time to let go. If you feel as if your hand is being sucked into the partner's skin, this means that the partner's skin wants you to keep holding on.

Working through clothes

By the way, this technique can be practiced on a partner's clothed limbs. At the very beginning, it may be easier for you to imagine that you feel the static release when working with bare skin, but, in fact, electromagnetic fields are not diminished with clothing. The force of these fields does decrease over distance. But thin clothing does not make a huge difference in the distance between your hands and the skin of your patient.

Within a day or two of practicing, a person should be able to feel these things right through thin clothing. If you doubt this, answer this question: can you feel when someone has hugged you for a little too long, even if you are both clothed? Of course you can. The same "go away" signal that you unconsciously (or consciously!) send to a person who hugs for too long is the same kind of signal that a partner's skin will send you when your FSR work is done.

The hug that lasts too long: an example

Hopefully, most of us who are planning to do this type of work already know, via our intuition, exactly how long to keep hugging someone and when to let go. Is there anyone among us who has not experienced an uncomfortable feeling when he is hugged for a bit too long, to the point where he suddenly feels discomfort in being hugged? Oppositely, haven't we all wanted, at some point in a harried or stressful day, to just be held tightly until we feel that we've had a chance to collect ourselves?

When holding small children, it is always obvious when they want to be held more tightly. They snuggle in and almost burrow into your chest. And yet, the moment that they've decided that they don't need a hug anymore, they are impossible to restrain; they squirm and fidget, making it obvious that the time for holding is over.

No one should need to be taught how to recognize when someone needs a hug, or when someone wants the hug to end. However, in our untouching culture, when it comes to therapeutic touch, we actually have to study and practice in order to be able to perform these basic, human functions correctly.¹

So start practicing holding and supporting a partner's arm, leg, foot, neck, or whatever wants holding. Note carefully if there is a quick, fleeting relaxation response to the touch, and also note when the static in the skin stops pulling you in like a magnet and starts pushing you away.

Children are very quick at learning this technique. Adults sometimes take more time.

YOU ARE READY TO PRACTICE FSR

You now know the basic technique of FSR. It consists of hold, notice if anything happens or not, and let go.

Hold with the correct amount of pressure, follow the person's skin with your hands if the skin or underlying tissues move in such a way that you need to move, and then, when the partner wants you to let go, let go. That's it.

I could probably get away with writing several chapters describing over and over the technique of FSR. Instead, I have put it all down in about fourteen pages, and I think I have said what I started out to say.

The reason for the brevity of text describing the techniques is this: there is not very much about these techniques that can be taught in words. The techniques are very simple. The trick to mastering these techniques does not lie in intellectual understanding. The best way to become proficient is to jump right in and practice these seemingly simple techniques. It is the practice, not the intellectual understanding, that will make you skilled.

Response to FSR: the diagnostic portion of your work

As you have seen by practicing the above sections on position and pressure, your partner's forearm will usually perform some quick and tiny movement, a relaxation response, when you hold his arm with just the right amount of support. You used just enough support to take the place of the energy that he was using to resist gravity. He experienced a release of tension in some part of his forearm. Diagnostically speaking, if your patient had a relaxation

¹ Some people do have trouble recognizing these signals. Autistic people and those with Asperger's syndrome may not be able to recognize when their touch and/or their presence are not wanted or needed. Also, I have noticed that people taking certain drugs, notably the antidepressant, the anti-anxiety, and most of the Parkinson's drugs, are not able to ascertain when they are receiving a "go away" signal.

response, then the area being held is healthy enough, for our purposes: you don't need to work any longer in this particular area. If your partner had a response, you can make a mental note of the fact that this particular body location is able to respond, and you can move on to the next body location, a few inches away.

Watching for movement

Practice supportive holding on your partner's forearm and notice whether or not any response occurs. This may seem redundant at this point, but though the material is similar, notice that the focus has now changed. We have moved away from "how much pressure" and "when to let go" to "how much of a response was there?"

So even though you have practiced touch, try it again, but this time stay focused on whether or not your patient responds. Whether your practice partner does respond or not, when you get a "go away" signal, lift your hands off the skin and move them to a new location a few inches away. See if this new location responds. After you are done at the new location, lift your hands off the skin and move your hands yet again, to yet another location a few inches farther down the arm.

You may have already noticed this movement that we are looking for when you did the "two hands" exercise a few pages ago. A tiny movement of the skin is all that you will notice, but it is very significant. This is the *immediate* relaxation that often occurs from supportive human touch. This is the movement that we look for when doing FSR. This movement, or the lack of it, allows us to evaluate where the patient is having normal responses or not.

When you are working with a healthy patient (defined here as a patient who does *not* have an unhealed injury in the area), you will notice that the moment you place your two hands on the patient in an opposites (supportive) position, there is usually a tiny, automatic, reflexive movement coming from the patient, as if in response to your support.

One of the PD Team members refers to this immediate, unthought response to being touched as "The Dance." As she sometimes says, "It looks like my partner's skin dances with my hands."

Responsive movements can be small and quick, large, or slow

The movement may be very quick, a small, instantaneous movement, over in a blink, or it may be a languorous, undulating move. The movement, if small, may not be visible to the eye, but if you watch your hands, you might see that your hands moved a bit: your hands were carried by the partner's response.

Notice carefully the exact angle of your hands, their exact position relative to the floor and ceiling, as you go to put your hands on your partner. Within a split moment of putting your two hands on your partner, you may see that your hands are no longer in the exact angle, relative to the ceiling and floor, as they were when you started to place your hands on the partner's skin. Sometimes the partner's skin and muscles will relax just a tiny bit, so that you end up with your hands a few degrees off from where you intended them to be. Other times, a partner might relax so deeply that your hands will end up 180 degrees from where you started.

Even if your hands are not carried away to a new position, you may feel something, a sense of life or a brief acknowledgement of your hands, in the body part that you are touching.

Even though the partner might not consciously feel the movement that his skin and underlying tissue is making, this movement or alive feeling is certainly palpable to the experienced FSR practitioner's hands. The skin of the partner is not particularly moving away or

toward the FSR hands; it feels more as if the skin and its underlying tissues are relaxing just a tiny bit into a different, more comfortable position.

Sometimes, when people see me demonstrate this technique, they want to protest that the partner was not relaxing. They accuse me: “You were shoving their arm around!” I have to insist that I was doing nothing of the kind. Other students take the opposite stand: “Nothing happened in response to your hands, the partner just relaxed a little.” Well, of course. That is the whole point: the partner will relax when supportive hands are placed on his skin. This relaxation is extremely fast and it usually seems like nothing significant has actually happened.

Because the response is so unexpected, so hard to feel on the part of the partner and so surprising to the new practitioner, it is possible that both the practitioner and the partner may want to insist that the other person must have been intentionally “moving the forearm around.”

However, whether the movement is large, small, quick or slow, all that matters to us, for our purposes, is whether or not this particular body part was capable of being held and capable of responding in any way, shape, or form. If it was, then the diagnostic answer is “yes, healthy enough for our purposes right now,” and we can move on to another area on the body.

Holding on: maintaining the support during movement

Holding on, keeping the hands in supportive contact even while the patient responds or moves around, is a critical part of the support. When you hold a person with supportive touch, you are rather implying that you are there for him, holding him for as long as need be. This means that, if your patient’s arm (or whatever body part) does move in response to being held, you have an obligation to continue to follow the movement wherever it goes, providing support until you receive a “let go” signal. Sometimes this means that a practitioner’s hands may end up in a very different position than where he started. But wherever the patient goes, there you, the practitioner, must follow.

Letting go temporarily, moving to a more comfortable position

If, in response to your support, the patient’s arm (or body part) moves in such a direction that you can no longer hold on comfortably or keep your balance, then, of course, you should let go and quickly reposition your hands in a way that will allow you to be comfortable while continuing to provide support. The patient will not go to pieces if you let go for a quick moment. Sometimes, if you sense that the patient’s body truly does not want you to let go, but you simply must move to a more comfortable position, then rotate your arms around or move your torso in such a way as to accommodate to the new holding position without actually lifting your hands off the skin, if possible.

Use your common sense with this; there is no value in having the practitioner get a crick in his neck. Picture a worried child wanting to be held tightly by a parent: the parent can move as much as he needs to get himself in a comfortable position and the child will not fall apart while the parent does so. However, once the parent gets to a position of maximum comfort and stops fidgeting, the child also settles down more deeply.

Sudden jerks

The practitioner must be prepared for those rare response movements that are large or jerky. If your hands are committed to supporting the patient and the patient’s arm (or whatever) suddenly twists or bounces, you need to hang on even though you may feel, for a split second, as if you are being carried somewhere unexpected.

As you become more experienced with this technique, you may, once in a while, notice that a faint electrical discharge that feels sort of like static energy moving through your own hands often precedes a major jerk or twist. If you are in tune with this sort of thing, you can use these static discharges as a warning to brace your feet on the floor or loosen up your elbows in preparation for a sudden lurch or lunge.

Problems that might arise when starting to practice this technique

This section is based on questions or problems that often arise in class.

Problem #1: The practice partner's forearm simply refuses to respond.

It is possible that the partner you are working on actually does have an injury in his forearm. If this is the case, his arm may not respond whatsoever to your touch. When students in my class find, while working on one particular fellow student, that no movement ever occurs no matter who is holding this student's forearm, it often comes out, upon questioning, that this particular student-partner has had a memorable injury in the forearm. The injury is usually a broken arm bone, at the very site being practiced on.

While students tend to feel like failures when their partners don't make a response, the students should always be aware that this technique can reveal areas of non-responsiveness. If your practice partner has an injury in the vicinity of the area you are practicing on, he may not respond. His injury is the reason that there is no response.

So if you do not get the results you expect, do not immediately blame yourself or the technique. Consider that the partner may have areas that need a spot of work. And merely choose another part of the arm, or use the opposite arm. Or practice on someone else.

Work with the above technique of holding until you find that you can place your hands on someone's arm, and then, in most cases, observe an immediate, visible, very slight accommodating movement of the skin or underlying muscle.

Problem #2: Not using enough support. Most students, after having worked on their own with this text, are amazed when they get into a FSR class or workshop situation and receive an actual demonstration of the touch from the class teacher. They may say, "You (the teacher) are using much stronger pressure than I had expected." or "You're using way more pressure than I imagined from the reading."

After awhile, as they keep working with the teacher, they realize that the teacher isn't actually using a lot of pressure. The teacher is being practically passive, but his hands are merely matching the inherent outward force of the partner's body with a matching, inward, supportive force. However, the person on whom the teacher is working may perceive both that the teacher is being firm, or solid, and that the teacher's touch rapidly becomes undetectable.

Again, what students usually were doing wrong was trying to be gentle. What they needed to be was "unnoticeable." To be unnoticed, the touch must be confident, firm, and use no apparent force upon the patient other than the force from the dead weight of the practitioner's hand – a weight supported by the practitioner's other hand – a force that turns out to be strong enough to convey support, support, support to the patient.

A so-called "gentle" touch is very, very noticeable. A complete, full hand contact can be almost invisible. Think again about how the mother was holding her neighbor's baby. Her contact was utterly firm and confident, but no pressure, no intention was being exerted on the baby.

One student described it as "the way you hug a friend who's had a hard day. You hold on with enough strength to show that you're there, but you're not trying to be pushy."

Once, while I was demonstrating the technique in class, a student asked the demonstration patient to describe for the class just how much pressure I was exerting with my hands at that moment. My hands were *firmly* holding both sides of the patient's foot as I lectured. The demonstration patient opened her eyes and looked around, slightly surprised at the question. "Is she touching me? I can't feel her hands at all."

Practice, practice, practice

Practice will teach you more than any more words can ever teach you on this subject. Do not practice this on a PDer. A PDer will not make a normal response. You may be able to force a response, or he may be able to force himself to create a response, but, in general, many parts of a PDer's body, not just the center of the foot, will fail to have a normal response to being touched.

Have you ever rested your arm or hand against the arm or hand of a friend and leaned it there for a long time without moving, such as can occur while watching a movie together? You may recall that, when you returned your sense awareness to your hand or arm, you realized that you could not tell (without looking) where your own arm ended and the arm of your friend began. That is the level of support you are learning to attain, only you want to learn how to attain it quickly, instantaneously, without first having to watch a whole movie.

ADVANCED FSR TECHNIQUES

Holding combined with a bit of a nudge

If the person's body does not respond immediately to correct, supportive contact, it is possible that some tension is lurking therein, and the area under contact will not budge until something disrupts the tension pattern. It is very possible that the area under consideration will be able to move and respond if it is given a little bit of a nudge. Sometimes, a slight nudge of movement from the practitioner is all that is required for the body part in question to wake up to the fact that it is being supported. Once it is awake, the recalcitrant body part may respond nicely. It will usually move in the opposite direction of the nudge, as if it is resisting the intrusion of the therapist. However, once it does move, it may be loosened up enough that, a moment or two later, it will respond to the simple holding technique of FSR in the normal, reflexive relaxation manner of healthy tissue.

Even without the presence of an unhealed injury, a partner/patient may have some little bit of hesitancy or tension, some snag that prevents relaxation. If a patient's body does not seem to respond in any way to being supported, the following nudge and/or imagined movement techniques may be enough to suggest to the patient that he let go of the snag. Once the hesitancy is gone, he may respond normally to simple touch. If he does respond, then the area will be treated just like the other others that do respond to FSR. It will be held, the fact that there was a response will be noted, and the practitioner will move along to an area of the body a few inches away. The practitioner is hunting for areas that do not respond. As soon as an area responds, the hunter can move along.

A tiny nudge

What is meant by a tiny nudge? Let's say that you, the practitioner, find yourself supporting your partner's forearm with your hands in what we shall call "position A." From this

position, with your hands opposite each other, you may bring your hands together ever-so-slightly and then immediately let the hands bounce back to position A. The tiny movement is not really a push, it is more like a tiny bounce, or pulsing motion in which the practitioner's hands move momentarily closer together and then move back apart again. Note that I never use the words "push or shove on the patient with your hands." Instead, my language is that the hands of the practitioner come closer to each other and then bounce back apart to their starting position. The practitioner is focusing on his hands, not on what he is doing to the patient. If this move is done correctly, the patient will not perceive the force of the nudge. The nudge will not be felt because both of your hands are opposite each other, taking up the nudge pressure from each other. Since the patient is supported, he doesn't need to do any work to resist the change in pressure. Therefore, he won't really notice what you are doing.

Very often, if a slight tension in the patient/practice partner is preventing the normal type of response that most people have to supportive holding, this tiny, invisible- to-the-naked-eye nudging movement will dislodge the tension. Once the tension is dislodged, the area being held may well move a bit in one direction or another. The area may take advantage of the support being provided to relax to a more comfortable position. When this occurs, when the area starts to move, the practitioner's hands must follow the movement, continuing to provide support, as described in the section on holding on, until such time as it is appropriate to let go.

If nothing happens: change hand positions

If there is no response to the little pulsing movement, move the hands a little bit. Maybe move them a little more anteriorly/posteriorly, or maybe a little bit laterally/medially. If the practice partner immediately relaxes with the hands in this new position, then try going back to the previous position and see if now you can also get a response in the previously unresponsive position. If there is no immediate response in the new location, try moving your hands closer together and apart again in the quick, pulsing, nudging manner described above, to see if the patient will respond. Remember, as soon as you get a reaction – any reaction – in response to your touch, you are finished in this area, and can move on.

Looking for problems

Don't lose sight of the idea that you are doing diagnostics. You are not actually trying to release tension in your patient: the relaxation just happens as a side effect of the testing. What you are doing is looking for areas that truly are not responsive. You will be using this technique to try to locate those strangely rigid areas on a PDer's body that do not, no matter what you do, respond in a normal, healthy manner.

At this point in your study, you are practicing FSR to learn what a "normal, healthy manner" is. There is a pretty wide range of normal, and, after working with just two patients, you may think that there is a huge difference between the two. But if you work with half a dozen or more healthy people and then hold the foot of a PDer, you will be able to detect immediately that there is something deeply, unnaturally wrong going on in the injured areas of your PD patients: their bodies will present an obvious, possibly alarming lack of normal response. If you have used the simple holding technique of FSR with enough people, when you go to work on the feet of a PDer, you may almost feel, as one student described it, "as if you are holding a corpse that's been covered up with living skin."

If nothing happens: do more

If there is no response even after you try doing a gentle, two-handed nudge, and there is no response after you have tried shifting your hand position a little bit, try using a slightly more forceful nudge.

If nothing happens: do less

If no response occurs after you've tried giving a pulsing movement and then an even firmer nudge, try using a smaller nudge. How small? This small: do not actually move your hands. Instead, remain right where you are with your hands not moving, and mentally, with your imagination, picture that you are moving your hands towards each other and then mentally imagine that your hands spring back apart. Do not actually try to move your hands.

Despite your best intentions not to move, and even though your physical hands do not move, the mere thought of moving on your part will stimulate an electromagnetic manifestation of the thought of movement. This electromagnetic suggestion of movement coming from you may resonate with your partner and suggest movement. This extremely subtle type of "movement" (imaginary) is very often the most effective type of stimulation for stubborn tensions. Very often, imagining that you are moving your hands is the best way to wake up an area in your partner that is alive and healthy, but stubbornly stuck.

Notice that you are *not* imagining that the patient is going to move. This would be an imposition on the patient. As always, this technique allows the patient to do whatever he wants. If you are going to impose your thoughts on anyone, impose them on yourself. Your patient will respond best if he is in control. Like the contented baby who is being ignored, your patient will be most comfortable if he doesn't have to deal with your expectations. Again, "forceless" refers to the mental exchange between patient and practitioner, as well as the physical perception.

Do not try to stare down your patient's unblinking foot

Your attention is on your own hands, by the way, and not particularly focused on the patient. The fact that you can notice whether or not the patient makes a response is to be attributed to the obviousness of the patient's response and the disinterested quality of your observation. Don't be scrutinizing the patient too severely. A watched pot never boils and a sensitive person or silverback gorilla does not like to be stared at. You can learn to be aware of whether or not a patient has responded without conveying to the patient that his every move is being assessed. Be somewhat detached, like the sailor that shifts and sways ever so slightly in response to the movement of the ocean, even though he is not paying conscious attention to the ocean. There will be more on this subject later.

If nothing happens after this: move on

If a particular area, a particular body part, does not show a reflexive response to simple supportive touch, nor does it respond to slight nudges or thoughts of nudges, make a mental or written note of the location of the stuck area, and move on to the next area. Very possibly, after the surrounding areas have relaxed, the stubborn area will be able to respond.

If the stubborn bit simply does not respond no matter what, but instead sits impassively, as if it isn't really quite alive, it is very likely that an injury, subconscious tension, or tissue displacement has happened in this area. If this is the case, as noted above, you will want to make a written or mental note of the area, and then move on.

How can this recalcitrant area be helped? The variant of FSR that we use on Parkinson's injuries can be used on areas that do not respond. This will be discussed in an upcoming chapter.

The next chapter will have some specific training drills that will move your focus towards holding the leg. Although PDers often have neck, shoulder, hip, arm, cranial and spinal injuries, in addition to their foot injuries, they *all* have foot injuries. Not only that, their foot injuries are responsible for setting in motion those physical alterations that are most closely associated with classic Parkinson's. Sometimes the other injuries help to mentally and emotionally cement the foot injury in place and to create the unique variation of PD that each PDer brings to the syndrome (no two PDers have the exact same presentation of symptoms), but, for the most part, the foot injury is the one that needs to be addressed first. Therefore, the next chapter will focus on performing on the leg and foot the holding and diagnostic techniques explained in this chapter.



“If I have said, ‘My foot hath slipped, Thy kindness, O Jehovah, supporteth me.’”

- Psalm 94:18

CHAPTER THIRTY-SEVEN

APPLYING FSR TO LEGS AND FEET

Looking for the root problem

This chapter suggests some leg, ankle and foot handhold positions for applying the FSR techniques that were taught in the last chapter.

Although people with Parkinson’s may have injuries all over the body, the energetic blockages that all PDer’s have in common are centered in the foot and ankle. Therefore, we usually start holding and assessing these patients somewhere on the leg, usually at the knee, and work slowly down to the foot and toes.

The question in our minds when our hands settle supportively onto the patient’s legs, ankles, and feet is this: is there any sort of detectable response? If there is a response, we wait until our hands are asked to let go and then continue down to the next place that we have selected lower down the leg; we usually start at the knee and make our way down towards the feet. If, as frequently happens, there is a faint response but the leg fails, even after several minutes, to give a “go away” signal, sometimes we just have to let go anyway, so that we can assess the rest of the leg.

Getting waylaid on the way to the feet

Sometimes, the legs of PDer’s are so desperate to be held that the skin of the PDer, as high up the leg as the knee, will seem to be magnetically grabbing your hands deep into itself with powerful attraction, as if it never wants you to let go. If this occurs, you must respectfully remove your hands, at some point, despite the desperate clinging. After all, your job, at least in the first session, and at frequent intervals thereafter, is to assess the entire area from the knee to the foot, to find the areas that need the most work, the very spots that seem to be the root causes of the surrounding deficiency and/or chaos.

This can be a bit of a challenge. I have heard from several practitioners who can never get past the knee, because, when they start working with a client’s knee, the knee never ceases “wanting to be held.” These practitioners are usually wondering how long it might take before the knee stops wanting to be held. Some reasons that the knee might be wanting to be held are because it is injured, it has taken up excess load because the ankle or foot isn’t doing its share of the work, or it is receiving inadequate energy. Typically, the energy supply to the knee is insufficient because the normal energy patterns in the entire leg are disrupted; but the source of the disruption is usually in the foot. Therefore, holding the knee, even for a long, long period of time, may never resolve the underlying problem.

Although, in the preceding chapter, I made the point that you don’t let go until the patient’s body gives you permission to let go, you must also keep in mind that, sometimes, in order to get to the root of things, you must let go of an area even though it is still screaming for attention. Consider, therefore, that, at least the first few times you work your hands from the knees to the feet, you are doing assessment and not therapy. Actually, of course, the two are one and the same. But to keep from spending too long at the knee and never getting to the foot, try to

keep in mind that you are looking, at least during the first few sessions, for that place on the leg, ankle, or foot that is the very worst, the core problem, the spot that seems the most dead to the world.

I do not need to remind you, at this point, that you should practice the work below first on a healthy person before starting to assess the legs and feet of a person with Parkinson's disease.

Practicing on legs

Place your supportive hands either on the knee or just below the knee. Your two hands should be on opposite sides of the leg or on opposite sides of the knee.

If you get a response to your FSR holding, move the hands a few inches down the leg and place your supportive hands in the new position. Again, note whether or not you get a response. If you do get a response, again move your hands a little farther, maybe one, three, or four inches farther down the leg.

If, at some location, you don't get a response to the simple touch, try the very small nudge, a sort of almost imperceptible compression-and-release move. If you still don't get a response, try a slightly more overt nudge. If that fails to get any sort of response, try giving a mental nudge two or three times.

If you get no immediate response, you may want to wait at least half a minute between each of these nudge attempts. As you get more adept at doing this work, you may find that you can tell within a few seconds whether or not there is even a capability for significant response in an area. But in the beginning, you may wish to wait half a minute if you think you are detecting an absence of response, just to make sure that there isn't going to be some sort of delayed action.

If you do get a response, move on. If you still don't get a response after you have spent several minutes in the area and have tried physical nudging and mental movement of your hands, go on to the third part of the technique: make a mental note of it, don't worry about it, and move on. Possibly you can try this area again later if you get releases in the surrounding areas.

Moving on

What, exactly, do I mean by "moving on?" There are several possibilities. One possibility is that you might place the hands a few inches lower down the leg from where you were before.

Or you might choose to place the hands on the opposite orientation from your first position: if your hands were on the front and back parts of the leg, you might wish to move them to the left and right sides of the leg.

If your hands were up at the knee and that felt fine, you might wish to place your hands halfway down the lower leg, or maybe even put your hands on the ankles. In general, the more dead and empty of response the leg feels, the smaller might be the increments you want to move in. If everything feels just fine up at the knee and halfway down the lower leg, you might want to go straight to the ankle and from there right to the middle of the foot.

Part of the reason for going slowly with a deeply unresponsive leg is respect. A leg that cannot even generate a response is also a leg that has lost a lot of sensory and proprioceptive capability. A limb that is not able to respond is not able to tell you if it is frightened or that it wants you to go away. Since it can't tell you to go away, it is best to err on the side of patience and respect, and assume that you are treading on territory where you are not trusted, or maybe even not wanted.

Of course, your PD patient will tell you that he wants you to hold his leg. However, from an emotional and mental perspective, the leg that you are trying to hold is the leg of the much,

much younger adult or child who was injured and never healed. That younger person may not want you to hold his leg. That person may not trust you.

Therefore, always go slowly and respectfully if you are getting no response. It is likely that when you are working on the legs, you are working on a part of your patient's body that is mentally disassociated from the rest of the body. Rather, the legs may have a mental association with being young, helpless, hurt and possibly even betrayed by those in authority. So assume that the leg in question does not trust you – why should it? It doesn't even trust the healing ability of the person to which it is attached. So move slowly and respectfully.

Asking permission

In our PD treatment group, we make it a habit – call it silly if you wish – to always, at the beginning of every session, ask the patient's permission to hold his leg, arm, foot, or whatever. Very often, the patient is taken aback the first few times that we ask permission: “Well of course I want you to hold my leg! That's why I'm here! Sheesh.”

However, after about five or six sessions, the patient usually responds in a very different tone; he understands that he has the right to say yes or no, that he is taking charge of what happens to his body, that he is the loving steward of his body. Then, when he says, “Yes, you have my permission,” he feels as if he is stepping into a position of authority.

Even more importantly, his subconscious self and the parts of his body and mind that are still locked up in their childhood fear, self-pity, or shock, get to hear “the boss” giving permission for the practitioner to approach the injured area. It does seem to us that it is very important for the injured area to know that we are only approaching it with the permission of the head office.

So, even though you are only going to practice these steps on healthy people to start, you may wish to practice saying to them, prior to actually setting your hands down, “May I hold your leg?” It may seem extremely formal, even stilted and unnatural at first to say such a thing. However, after a few hundred times, you will start to notice that, when you forget to ask permission, it seems almost inexcusably rushed, pushy, and even somehow violent to just go and impose your hands on someone without making sure that it's OK.

Continuing down the leg

Where exactly should you put your hands? Place them wherever your intuition inspires you to do. The exact details of where you place your hands are not important. The main thing you are doing is being a respectful detective, looking for parts of the body that have a profound inability to respond.

Continue moving down the leg, moving the hands a few inches (more or less) each time, and/or move the hands from the fore-and-aft to the side-to-side positions, if you like. Do this all the way from the knees to the ankles.

Again, your purpose here, at least the first few times you do this, is not to do on-site healing work per se. Your purpose is to determine whether or not the leg feels as if it can respond in a somewhat healthy manner.

Practicing on ankles and feet

I am almost hesitant to describe where to place the hands when you've moved all the way down the leg to the ankle; many students cling too rigidly to whatever I write, particularly when I describe some ankle and foot holds, even though I state over and over that the following are just

suggestions. But some people are so unaccustomed to holding feet and ankles that they truly do not know where to start. So I will provide the following suggestions for places that a practitioner can hold his hands.

Looking for articulations as well as a general ability to respond

Working on the ankles and feet will involve a slightly different set of instructions than you have used so far. When holding the leg, the point was to see whether or not the leg made any sort of response. With the ankles and feet, we want to see two things: if there is any response in the tissues that overlay the ankle and foot bones, *and* whether or not the bones of the ankles and feet can move in relation to the adjacent bones.

The many small bones of the ankles and feet are supposed to meet each other in exactly the right manner so that every bone junction can move in a particular direction and cannot move in any unintended direction. The various shapes and the intersections of the foot bones allow the feet to flex, extend, flatten and rebound. The feet flatten slightly with every step, supporting a tremendous amount of weight, and they rebound back to the less flattened position after every footfall. They can do this only if all the bones of the foot articulate (move at the joint) in exactly the correct manner.

Therefore, you need to know whether or not each of the foot bones is able to move independently (correctly) or if a pair or a group of bones is so jammed together that they can only move as a group (incorrectly).

In practicing Yin Tui Na on the ankles and feet, or any part of the body that has underlying joint articulations, you will first notice whether or not the skin or muscle parts of the foot seem to respond to supportive holding, and then you will gently nudge the various meeting areas (articulations, joints) of the bones to see if the bones can glide in the correct direction.

You must practice this on healthy feet. Each of the foot bones, if healthy, moves in a slightly different way. While you don't need to know the names of the bones or memorize what they do at their articulations, you do need to develop a sense of the types of movements that healthy feet seem to do in response to being gently nudged. If you familiarize yourself with the sensations generated by healthy foot bone movement, you will immediately recognize a joint that is jammed shut, even if you don't know the names of the bones in question or the direction that the general area should have moved.

The following list of possible places that you might want to put your hands also includes, with each holding place, an idea or two for the vector (direction) in which you will perform your extremely gentle nudge.

Again, the only reason for resting or nudging one's hands in these suggested positions is to determine whether or not the touch evokes a healthy response. You are *not* trying to move any bones, loosen up anything that feels tight, or return a displaced bone to its correct position. The following suggestions will help you assess which areas might need more holding and if they need a different type of holding. If the areas do not respond to holding or nudging, they may be needing the resting variant of FSR that we use on PDers. That variant will be described in a coming chapter.

If you want to place your hands in different places than the ones suggested, do so. The following suggestions are merely starting points.

A teaching video: an aside

Although many people ask for a video or DVD of someone practicing the following technique, it would be pointless: there is nothing to see. A video of me holding someone's ankle to see if there was a subtle responsiveness would just show, to the observer, footage of me sitting motionless, with my hands motionless, on a person's motionless ankle. I might sit there for several minutes, not doing anything, waiting to see if the ankle was going to respond. This would be supremely boring. There would be nothing to see.¹

Still, students are often keen for detailed instructions that they can use as a jumping off point. So I will make some suggestions for where to place the hands when working in the ankle/foot area.

Working with less than a full hand

Feet parts are often so small that it is impossible to place the entire hand over some foot part. For example, when supportively holding the big toe, sometimes you can only fit a little bit of your hand around the toe, or maybe you can only fit one finger up against one side of the toe and another finger on the other side of the toe.

Likewise, because of the curve of the foot's arch, it may be impossible to place the palm of your hand firmly up against the bottom of the foot. In this case, you can nestle the gently curved backside of your hand up against the sole of the arch. In other words, the important thing here is the support, not which part of your hand or how much of your hand you are using to provide the support. For that matter, sometimes when I am working on a patient's foot and I sense that the patient would feel more supported if I had a third hand, I press my shoulder gently up against some part of the sole of the patient's foot while I am using both hands to support the ankle. (I am so short that, sitting on a stool at the foot of the treatment table, the table comes up to the middle of my chest. My shoulder is only a few inches higher. If the patient's foot is close

¹ I once bowed to popular demand and made a video of myself holding a person's leg, ankle, and foot in various holding positions. I spoke into the microphone very clearly, stating that I was *not* actually doing FSR. FSR is very slow and boring to watch. Instead, I was merely placing my hands on the subject's leg, ankle and foot in order to demonstrate some of the holds. I moved fairly quickly through the various hand poses, stating over and over that I was just demonstrating hand positions; I was not doing FSR.

After I released the video, I got many complaints from patients: previous to seeing the video, their therapist, working only from my book, had been going nice and slow, feeling his way along the leg and feet. After seeing the video, the therapists had copied the tempo of my videoed hand movements. Just as I had quickly moved from one position to another, the therapists were now moving their hands quickly from one spot to another. In other words, the visual cues from the video were too compelling; the spoken instructions on the video were completely ignored.

If I ever release another video, it will be the most boring thing on earth. In it, I will demonstrate the tempo at which I go when a person's legs do not respond at all: I shall set my hands down in one place and hold them there for a solid minute or two, maybe longer. Any nudging or movement on my part will be so small as to be invisible.

Then I will go to the next holding position and hold my hands there for about five minutes. It will take an hour before the viewer has seen a fraction of all the possible ways that a practitioner might want to set his hands down on his patient. It will be so boring, no one will watch more than a few minutes of it before he is saying, "Enough already! Just show me the various hand positions, I understand that I am supposed to go slowly."

But I will not be fooled this time into thinking that this time will be different: too many people will *not* understand. Also, every patient is different. Each patient might need to have his foot bones held from a slightly different position and for a different amount of time.

People usually follow visual images more exactly than they follow words. The whole point of FSR is that the practitioner has to learn to follow his hunches and respond to the patient, not to a video. So I really do not think that there will be, or should be, a video.

enough to the edge of the treatment table, I can lean forward, causing the patient's foot to press up against my shoulder.)

I am not saying that you need to do this. What I am trying to get across is that the patient must feel supported by human touch, and it is your job as practitioner to provide the support. A supportive pillow is not the same as a human hand. But sometimes, that "human hand" doesn't need to be a full hand. A mere finger or a chunky human shoulder can sometimes serve the function of a hand.

PDer's will not get the therapy they need from supportive pillows or inanimate objects. They don't need foot braces. For that matter, orthotic devices in the shoes usually do more harm than good. They do need human support. If your hand doesn't fit comfortably onto the area that you are working on, use whatever part of your hand does fit, so that you can provide support, support, support.¹

Ankle and foot holding/nudging positions: some suggestions

Place a hand on either side of the ankle, with the medial and lateral malleoli (ankle bones) each held snugly in a palm of your hand. If the ankle area feels responsive to your touch, good: you can move on. If not, make a note of it and move on. You may wish to return to this area later.

Next, to find out about the ankle articulations, you may want to try nudging – or thinking about nudging (usually more effective) – the ankle bones in a few different planes of movement.

If you know a lot about the ankle bones, you might be thinking about the way that they are merely extensions of the long bones of the ankle. You might be thinking about the way that these long bones come to rest on the talus bone, in between the two ankle protuberances. Or you might not want to think about any of this. You might, instead, just think about how the ankles move when you try moving them in various directions.

Fascia

Bones cannot actually move in all possible directions. They are designed so that any given joint can only move in a very specific manner or direction. There are bumps and ridges all over the ends of bones that limit movement in all but the correct direction. However, even though bones, tendons, and ligaments may be limited in their ranges of movement, the fascial membrane that overlies these tissues should be able to move a tiny bit in any direction. The delicate, transparent fascial membrane that overlays every bone, organ, blood vessel, skin layer, and all other body parts is a smooth, almost slippery sort of membrane. The fascial tissue can

¹ I treated a patient who had impaled his foot on a pitchfork. The fork entered his foot from the front, slicing in between the first and second toe and drove in deeply to the center of the foot. The injury had occurred many years earlier but the white scar was still quite visible between the toes. There was no way I could place my entire hand between his toes. Instead, I wiggled my index finger into the space between the first and second toe. My other three fingers looped around the bottom of the ball of the foot and came to rest on the medial (inside) side of the big toe. My thumb pointed towards my index finger and was somewhat wedged into the groove that runs between the toes and the sole of the foot, under the three lateral toes. My other hand was holding his ankle. I just sat like that for about fifteen minutes, giving very firm support to the area with the pitchfork scar. After about fifteen minutes, the entire foot relaxed, all the toes, especially the first and second, separated wide apart, and the patient reported feeling warmth and life spreading throughout his foot.

This example is provided to show that it is not always necessary to get the whole hand onto an injured spot. I was using only my index finger on the spot indicated. But the whole rest of my right hand was also providing support, and my left hand was bringing up the rear by supporting the ankle against the pressure being applied from the front end of the foot.

move a tiny bit in almost every direction. So when you are mentally trying to see whether or not movement can occur in every direction at the site of an articulation, what you are really trying to see is whether or not the fascial membranes at those junctions are able to move in every direction. If the fascial membranes can move in every direction, it is a good bet that the bones will be able to move in the way that they are supposed to as well.

What is fascial tissue or fascial membranes? Answering a question with a question, I will ask if you have ever noticed a thin, transparent membrane that lines the outside of the bone when stripping the meat off a chicken bone? Thin transparent membranes run between the meat and the bone, between the bones and the ligaments, between every tissue group in the body. This thin stuff that seems sort of like clear food wrap or Saran wrap is fascial tissue. When you are looking for subtle movement in various planes, you are actually noticing whether or not the fascial membranes can respond to your suggestions and thoughts.

Three planes of movement

Generally speaking, since we live in a three dimensional world, there are always three planes of potential movement when any two bones come together. Next, considering that in joints each of the three planes of movement can go in two directions, namely one direction or its opposite direction, we must multiply three times two to calculate the number of directions that a joint might be able to move in. All told, we can test each joint for movement capability in a least six directions.

The six directions are these: one of your hands moves up while the other moves down. The first hand moves down while the second moves up. (Up and down can be with regard to the room you are in: up is towards the ceiling, down is towards the floor.) Next, one hand might move to the left while the other hand moves to the right. Then, the first hand moves to the right while the other hand moves to the left. Then, your hands might be willing to move closer together, or they might be willing to move farther apart.

I cannot emphasize too strongly that you are not actually moving the bones in any of these directions. What you are doing is holding a person's foot with your motionless, firmly supportive hands, and thinking about what might happen if, *in your mind*, you moved your hands in various directions. In response to your thinking, or possibly, once in a while, your making the smallest, most imperceptible of nudges, you may notice that the joint under your consideration at the moment jostles a bit or makes some sort of response to your thought.

Without even knowing what the bone looks like or which direction it normally moves, you can test the willingness of the joint's fascia to consider movement in all these directions. Again, you are not trying to see how much a joint can be forced to move. You are trying to assess how much a joint can be responsive to what is going on around it.

One interesting thing that comes to light while doing this type of work is that sometimes a joint wants to move a lot. In response to simple holding, or sometimes in response to your thinking about moving your hands a bit, a joint may spring into a completely different position from the one that it has been locked into. Other times, you will feel in your hands an imperceptible shift in the joint that is covered by your hands. Sometimes, if a joint responds a little to this type of mental planar movement, you can repeat the mental motions and the joint will respond yet again, and then again. Every time you go through these motions it is possible that the joint will loosen up further. Eventually, a joint that felt rigid and unresponsive may, after a series of almost imperceptible shifts, respond to simple FSR holding with a generous relaxation response.

Applying planes of movement to the ankle: an example

You may wish to push your hands that are holding the ankle bones towards each other and note if the ankles respond by moving in the opposite (outward) direction. You may wish to see if the ankle bones will nestle closer to each other as a rebound move when you imagine that your hands, closely connected to the skin of the ankle bones, move slightly apart for a moment.

You may also want to try mentally moving your hand in such a way as if one of the ankle bones, for a fleeting moment, would be nudged upwards, towards the long bone of the leg, while the other one moves downward, towards the heel. See if there is any sign of a response. If not, make a note of it. If there was no response, you may want to return to this area later. If there was a response, you still might want to keep holding the ankle so see if there is a response when you think about moving your hands forwards and backwards relative to each other, and then the reverse.

The main thing you will want to do is practice doing these directional suggestions on many healthy feet so that you can ascertain just how a normal set of ankle bones moves in relation to the leg bones, the heel bone, the talus bone, and each other. Even if you don't know how all these bones should move in theory, if you hold enough feet and try mentally moving your hands over most of the areas of the feet, you will soon come to have a sense of what a foot should feel like, and how it should respond.

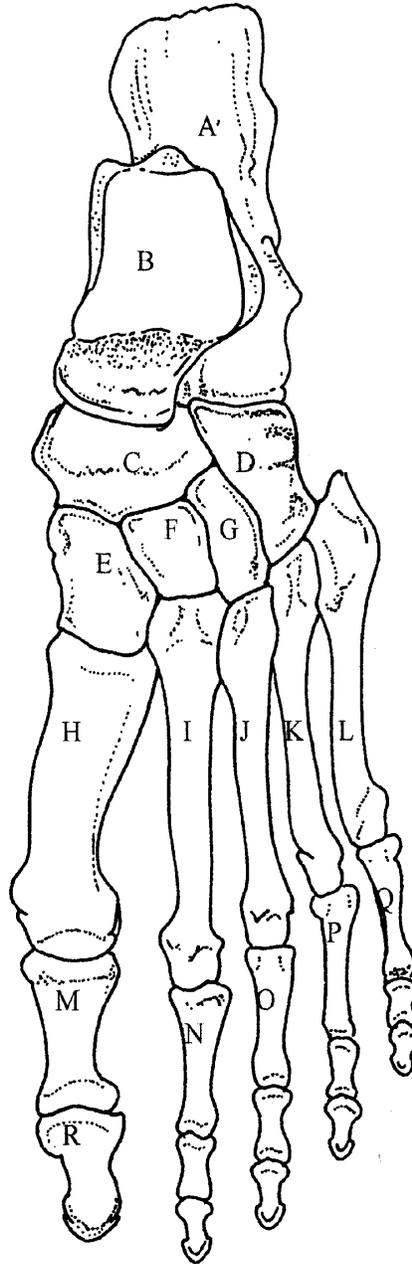
Again, you are not trying to see exactly what the foot does, but rather how it feels when it responds, or not, to your support, your thoughts, or your very small movements.

Diagrams of the foot

Detailed diagrams of the foot bones, viewed from seven different angles, are included in the appendix of this book. The following, oversimplified diagram of the foot bones is given here to familiarize you with the names of the bones and their approximate locations. The detailed diagrams give a better sense of the articulation directions. Note in particular the size of the middle (intermediate, 2nd) cuneiform bone in the detailed drawings. Observe that this bone is quite substantial when looking down on it from the top view of the foot (Plate I, in the Appendix). The view of the same bone from the underside of the foot (Plate VII) will show you that this substantial, square bone is so severely wedge-shaped that it tapers nearly to the point of disappearance by the time it gets to the underside of the foot; all that can be seen of it is a tiny sliver.

The ankle bones are not pictured. To be perfectly accurate, the ankle bones do not exist as separate bones. The ankle bones are actually knobs at the distal (moving away from the head) ends of the two long bones of the lower leg. These knobby ends of the leg bones nestle into either side of the talus bone.

- A. Calcaneus
- B. Talus
- C. Navicular
- D. Cuboid
- E. Medial (1st) Cuneiform
- F. Intermediate (2nd) Cuneiform
- G. Lateral (3rd) Cuneiform
- H. 1st Metatarsal
- I. 2nd Metatarsal
- J. 3rd Metatarsal
- K. 4th Metatarsal
- L. 5th Metatarsal
- M. 1st Phalange 1st toe
- N. 1st Phalange 2nd toe
- O. 1st Phalange 3rd toe
- P. 1st Phalange 4th toe
- Q. 1st Phalange 5th toe
- R. 2nd Phalanges



The Bones of the Foot
Fig. 37.1

BACK TO PRACTICING

Another possible place to position your hands is with one hand under the calcaneus (heel bone) and the other hand supporting the Achilles tendon. Notice if you have a sense that

you are holding a healthy ankle/tendon? If yes, then good. But if nothing seems to be moving, or this intersection of the Achilles tendon and the heel bone seems uncannily rigid, you may want to gently and quickly nudge or imagine a nudge as you bring your hands together and then let your hands rebound back into position.

As before, if a gentle nudge gets no response, wait half a minute or so and try a slightly stronger nudge. If still nothing happens, wait half a minute and try just mentally nudging your hands. Also try thinking about having one hand move towards the posterior while the other moves anterior. And then try the reverse. And what happens if you think about moving one to the left and the other to the right, and then the reverse? Learn how healthy feet respond to this sort of play, and then, when you meet your Parkinson's patient's foot, you will have a sense as to whether or not all is well in this area.

If all is well in the ankle, a movement or thought that compresses the ankle should evoke a separation response in the ankle and a movement that pulls the tendon/calcaneus apart should evoke a coming together of the two parts. In either case, the area should respond. If it doesn't, of course, try doing these movements mentally again, or try them using an invisible amount of actual movement, and notice whether or not anything happens. Repeat this once or twice if you like, trying to assess whether or not this area may be wanting a deeper type of holding (the resting FSR variant). Eventually, even if it didn't before, the heel bone/Achilles relationship may begin to feel responsive. If it does, then good. If not, make a note to yourself that this area might want some deeper work and then move on.

On the other hand, you may feel that slightly nudging (or thinking about nudging) the heel sideways to the left while thinking about moving the tendon to the right (or vice versa) would be a good thing to do. Fine. Do it. Possibly by getting the heel/tendon relationship to loosen up by moving from side to side, the relationship will also loosen up in other directions as well. Do what you like, do what your intuition tells you to do, do what the patient's ankle tells you to do. Let go of the ankle if the patient's ankle tells you to let go.

Try some or all of these suggestions on healthy feet so that you can learn to understand how this tendon/heel relationship works when everything is moving nicely.

The Talus-Calcaneus relationship

Next, if you are satisfied that the heel/tendon was moving properly, or you decided that it wasn't and you made a mental note to return there later on, you might wish to place one hand on the talus bone and the other behind the corner of the calcaneus. Or you may wish to choose some other area to hold. These are just suggestions.

Do the usual routine on these bones to assess whether or not they can move. The "usual routine" means that you will notice if the foot feels responsive when you support these bones. Then you will gently nudge these bones towards each other. Or possibly, you will gently imagine them moving apart. Or you might nudge them or imagine them moving one to the left, the other to the right. Or you might think that one is moving towards the head and the other is moving towards the toes. You can try to test these bones on any plane that you can imagine. By gently provoking a reflexive response in every possible direction, you will be able to create a mental picture of the way that these bones can move, relative to each other.

Foot bones make very small movements

Do bear in mind that even if these bones can move correctly, they will not move very far. Instead, what they will do is move a tiny bit, maybe invisibly. Of course, there is always the possibility that they will make a generous sweeping relaxation movement. But you should have no expectation, one way or the other.

Your hands, only through experience, can eventually know what it feels like to work with a responsive foot. The movements, tiny or languorous, that do occur will feel “right” to you if they are right. If you do the nudging and the thinking and nothing happens, if you do the tiny pushes and pulsing and you get a sense that the bones involved are putting their backs up and saying, “No!” then you will know that this is an area that wants more work. Don’t try to change its mind; just make a note that this area wants more work and go on to the next place.

Navicular-Calcaneus relationship

Next, you may wish to place one hand on the navicular bone and the other behind the corner of the calcaneus. Again, determine whether or not this area can respond.

In terms of finger/hand placement while doing this, you may wish to put one hand on the navicular bone and the other on the talus. Or you may want to drape the middle finger of one hand over the navicular bone with the thumb of the same hand supporting the sole of the foot. Or possibly, you will find that placing your thumb over the talus and the rest of the hand around the back of the ankle may feel the best for you. If the foot doesn’t relax in response to supportive holding, you may try gently pulsing your hands together in such a way that the navicular and talus bones are pressed towards each other. Or you might try thinking about your hands pulling apart from each other. Or move one of your hands that is over a bone to the left and the other to the right, or move one of them towards the front of the foot and the other towards the back.

Cuboid bone

Place the palms of one hand over the top (dorsum) and the palm of the other hand over the bottom (sole) of the cuboid bone. Look for a response.

Place one hand on the lateral side of the cuboid bone. With the other hand, grip the navicular bone between the thumb and middle finger. If there is no response, quickly and gently compress the bones towards each other and release.

Place the palm of each hand on either side (sole and top of the foot) of the medial cuneiform. Compress and release.

Place the palms of the hands on either side of the intermediate cuneiform bone. Compress and release.

Somewhere between your first and your hundredth treatment, this bone may shudder and jerk and possibly even whip around. Until then, just do all the above supportive holdings and note whether or not the area is capable of responding. In a PD patient, it probably won’t be. As always, make a mental note of this and plan on using the Parkinson’s variation on this area for a long time.

Place the palms of the hands on either side of the lateral cuneiform bone. If it feels stodgy or stubborn, try gently pushing your hands towards each other to see if the foot tissues between your hands will push back outward on your hands.

More ideas on where to hold

Place the thumbs of both hands across the top of the cuneiform bones, and the middle fingers of both hands under the sole of the foot, under the cuneiform bones, giving you a way to hold onto all three cuneiforms as a group. Maybe move your hands towards each other and then relax (compress and release) and see what happens. Does it feel like a healthy response?

Place your palm or the middle fingers of both hands over either side (top and sole) of the cuneiform bones as a group. Compress and release, either physically or in your imagination.

Grip the cuneiform bones with one hand (thumb and middle finger over the top and sole) and grip the navicular bone with the other hand (top and sole). Compress the cuneiform bones, as a group, towards the navicular bone and release.

Now we come to the metatarsals. Place a hand on either side (top and sole) of the proximal end of the 1st metatarsal. Compress and release. Move the hand so that the center of your palm is centered over the 2nd metatarsal and repeat. Repeat in this manner over the proximal ends of all 5 metatarsals.

Place the palm of either hand over either side (top and sole) of the distal end of the 1st metatarsal. Compress and release. Repeat for all 5 metatarsals.

Grip the cuneiforms with the thumb and middle finger over the top and sole. With the thumb on one side and index and middle finger on the other, grip the proximal end of the 1st metatarsal. Compress the metatarsal towards the nearest cuneiform and release. Repeat this with the other 4 metatarsals.

Place the thumbs across the tops of all five metatarsals, the proximal ends, and the middle fingers across the sole sides of all five metatarsal and compress them all towards the 3rd metatarsal and release.

Grip the distal ends of the metatarsal using a handhold similar to the above. Compress toward the 3rd metatarsal and release.

Place the thumb and middle finger on either side (top and sole) of the cuneiforms. Place the other thumb, and all four fingers as a group, on either side (top and sole) of all five metatarsals, as a group. Compress all the metatarsals towards the cuneiforms and release.

Try the toes. Place thumb and index (or middle, or fourth) finger on either side (top and sole) of the first phalange of the big toe. Compress and release. Move to the first phalange of the second toe. Compress and release. Repeat across all five toes.

Move to the second phalange of the big toe. Compress and release. Repeat across all toes until all the phalanges have been relaxed.

Note: The toe joints may move very quickly. I usually only spend a few seconds assessing each toe unless there is something clearly wrong in a toe joint. If there is a problem with a specific toe, then spend extra time and attention on that one spot. In general, the toes will be able to move quickly and will not even require much holding. Hammertoes and other toe contortions are very often caused by tensions a good distance away from the toes. Sometimes hammertoes relax in response to work done on the center of the foot: sometimes hammertoes don't relax until the ankles relax.

Repeat the above toe sequence with the thumb and finger-of-choice on either side (medial and lateral) of each toe, going over every phalange.

You can go over the foot many times. You can hold the bones in the ankle-to-toes sequential order suggested above or in whatever order you feel like, several times. If some stubbornly held places did release in response to your holding, it is very likely that some other previously stuck joint articulation may now be able to move. The bones are assembled somewhat like those old wooden ball puzzles in which the pieces are so curiously interconnected that you cannot really move any puzzle piece until you figure out which one to move first. Sometimes it may seem as if no bones will move until they are all ready to move. On each pass over the foot, each bone may make scarcely perceptible adjustments even when you are doing nothing but assessing. At some point, all of the bones may have corrected their own position enough so that suddenly they will all move smoothly and easily.

On the other hand, while working your way across the foot and ankle, it is very likely that you will come across one or several locations that feature such supreme rigidity that you can safely assume that this area wants something deeper. This area wants the type of FSR that we do on PD-related injuries. Fine. Finish going over the foot, make your assessments, and then, returning to the foot, apply the resting type of FSR in the location that needs it. Or, if you prefer, you may stop the assessment process right where you are and switch over to the even more Yin type of holding that we do on PDers. You may wish to do the latter technique for the rest of the session and forget about assessing the rest of the foot and ankle. Again, you will need to follow your intuition.

Neither sequence nor timing for “correct” FSR is carved in stone

To make the point that you truly can approach the sequence and timing of the leg-to-foot FSR in whatever manner seems best to you, according to the silent instructions that you are receiving from your patient's leg, I will share this frequent remark from practitioners who come to observe members of the PD Team: “I think I'm starting to get it; I've seen five of you doing FSR, and you're all doing exactly the same thing, but you're all doing it so differently! And when I watched you in particular working on the same patient that you'd worked on a week earlier, you approached his leg completely differently the second time.”

Do not use force

The movements that you are making must *NEVER* involve any actual force. *NEVER* use enough force so that you are actually pushing or shoving a bone in any direction. For one thing,

you may displace the bone. For another, if you use your hands on another person with the intention of moving a bone, you are practicing medicine without a license. Even acupuncturists, in some states, are not allowed to move bones.¹

APPLYING FSR TO THE PARKINSON'S PATIENT.

Though I have already mentioned this, I am going to state it again. When you start working with a person with Parkinson's disease, trying to assess via FSR where the injuries lie and where you will need to hold your Parkinson's patients for a long time using the PD variation on FSR, you will *not* want to lunge right in and grab your patient by the feet.

Those feet – like all parts of all people's bodies – should be handled with great respect. Instead of plunging right in at the arch of the foot, we usually start by holding the patient's leg in the vicinity of the knee. We slowly work our way down past the ankles to the feet. In this way, we are able to get close to the foot without frightening the injured person hiding inside the brave patient.

Sometimes, when working on a patient for the first time, the entire one-hour session might be spent slowly holding one area after another, starting at the knee and working slowly down the leg. Finally, in the last few minutes of the session, we might get to the point that we are resting our hands on the injured arch of the foot.

Even though this tempo may seem boring or unnecessary, the first session is more oriented towards assessment than treatment. Also, the first – and every – session is an exercise in trust building. Don't forget, the injured area is the one that needs to learn trust. The patient may be perfectly willing and trusting right from the start. However, the foot from which the patient's consciousness is completely disassociated is the body part that you must develop a relationship with. This foot, despite the assurances of its owner, does not trust you. Therefore, you will start at the knee, and if possible, make your way down to the foot.

No response in the knee or lower leg

Sometimes, a patient's knee and lower leg is so unresponsive or resistant that we need to start above the knee just to ascertain how much responsiveness the patient is capable of in his healthier areas. However, it cannot be assumed that there is damage or injury in the area of the knee and the area above the knee even if these areas are unresponsive. Very often, the leg and knee are rigid and unresponsive because of the injury in the foot.

Because of the creeping immobility that, over decades, moves up the leg from the injured foot, you cannot be certain that *rigid* legs and knees are necessarily *injured* legs and knees. Instead, if you try to start working with FSR just below a PD patient's knee and you get no response, you still might wish to continue down the leg. In this case, since there is an absence of response everywhere, you are trying to discern just where the absence of response is the most

¹ Because of two events of spinal damage from incorrectly applied Yang (forceful) Tui Na, the state of Texas has determined that acupuncturists cannot perform manipulative Tui Na. Considering that most acupuncturists are not adequately trained in this field and that they can do damage through their ignorance, this is probably a good decision. Yin Tui Na, it must be emphasized, never imposes enough force to move a bone, joint, tendon or ligament into an incorrect position. Yin Tui Na allows the tissues to relax enough so that, if a bone or tissue wants to move, it can. This passive approach has this advantage over forceful work: a tissue that moves into a more correct position by its own volition will not stray back out or return to its previous place. A tissue that is forced into a "correct" position may soon return to its accustomed "wrong" place.

acute. You will be trying to differentiate between mere rigidity of degeneration and the more intense, deathlike rigidity of serious injury.

On the other hand, if the knee and leg are sending “go away” signals to your hands, you may need to start above the knee. There is no point in forcing yourself on any body that is sending you distinct “go away” signals.

The maltreated dog

When working with a PDer, it may help to think of his injured foot as a dog that has been maltreated. Such a dog may be wary of anyone who comes near. Even if you have the best of intentions, you cannot befriend such a dog by forcing yourself on him. However, if you respect the guttural snarls of the dog and keep your distance, you may find that, after a few days or weeks of keeping a respectful distance, the dog may become inured to your presence. If you casually throw a few bits of doggie treat towards the dog now and then and still keep your distance, the dog may start to accept your presence, as long as you do not make any sudden moves.

Over a long period of time, the dog may decide to approach you. As you know, it will be up to the dog to make the first move. After the dog does learn to trust you, the relationship may develop along some sort of mutual lines. But in the beginning, it is all about the dog.

The foot of a PDer has been maltreated. It has been injured. When it cried for help, it was scorned and rejected. The foot of the PDer is still wounded and it no longer trusts the master, or anyone else, to take care of it. The foot, though physically attached to the body, is emotionally alone and feeling betrayed.

When you are performing FSR on the wounded foot, keep in mind the image of the maltreated dog. At heart, both the dog and the foot want what everyone wants and deserves: unconditional love. However, if you try to impose love too soon or physically move too fast with either a mistreated dog or a rejected foot, you will be snarled at. Keep your emotional distance, and approach slowly, respectfully. Let the foot make the first move.

Using force

Never force a move. The interconnectedness of the foot bones, tendons, ligaments and memories is such that if a bone doesn't move, it's because it cannot. Whether or not the immobility is due to a twisted and trapped piece of fascial tissue, a displaced ligament, or an unhealed (and should-have-been very painful) bone bruise, be patient. Let the muscles and bones and tendons take their own sweet time. Remember, the less you force it, the faster the work will go. The tissues will know if you are giving them the time they need and if you are respecting the overprotectiveness of decades. Your work will be rewarded by the quickest results if you slow yourself down and let the foot set the pace. If you are not getting results, you may be either holding too hard, too lightly, not waiting long enough after the compression and release, or else the area just doesn't feel like moving yet. Lighten up your touch. Or tighten it up. Wait until you can feel the muscle group under your hand "breathe a sigh of relief." Listen with your hands. But if there is no movement forthcoming, do not worry; you can return to the area with an even gentler, more deeply Yin technique: resting FSR.

The above completes the written training for FSR leg, ankle, and foot protocol. Of course, there are infinite ways of holding a person's ankle and/or foot that were not described in the above. You will discover some of these as you repeat the above technique in your practice.

As you practice, either repeat the entire sequence and see if anything has changed or else select the area that most seems to want to be held, and apply the techniques of resting FSR.

The first time that you work on the foot of a person with Parkinson's disease, it may take you a full hour to go over the knee-to-toe sequence even once. You may be able, in one hour, to go from knee to toe two times. The second go around might be done to see if there are changes occurring in response to the first administration of FSR.

If you are going any faster than this at first, you are going TOO FAST. Slow down. Only work on one side of the body for the first session. This will force you to really get a sense of what that leg/foot feels like. Work on the leg, ankle and foot on the same side of the body on which symptoms first appeared.

Then again, if you have the luxury of a two-hour appointment, you may be able to assess both legs, ankles, and feet. Be sure to spend at least the first full hour on the side where symptoms first appeared.

After several sessions, the articulations in the feet may be noticeably smoother. With a healthy foot, you will be able to do the entire leg/foot assessment protocol in 5 to 10 minutes, and then you can select the spot that wants to be held very still for the rest of the hour. Very possibly, bones and tissues will move smoothly and easily into a more and more comfortable place at each session. Then again, maybe not.

Surprise! Practice on a healthy person first!

I shall be redundant: practice this first on a person with healthy feet. Then when you begin working on a person with Parkinson's disease, you will appreciate the extreme difference between them. Not only the articulations on your PDer, but the entire foot may feel strange. A person with advanced Parkinson's disease may have feet muscles with the texture of soft cheese, or bread dough. The feet may be swollen and puffy. It may be very hard to feel the bones through the water-logged skin. Or oppositely, the foot and leg may feel hard as steel, and the toes may be tightly torqued in one strange position or another.

The patient may not be able to feel your hands. You may not be able to feel the bones in the patient's foot. Just do your best, and visualize where the bones must be if you cannot feel them. Within a month or less, they may begin to show changes. Severely damaged feet, those that have become shapeless and numb, may take years to recover, but most feet usually show faint signs of improvement within a few months.¹

Again, the above sequence for working on the foot bones is merely a suggestion. If you feel that you want to work from lateral to medial on one pass over the cuneiforms, instead of medial to lateral, fine. If you want to consider the cuboid as being in line with the navicular instead of in line with the cuneiforms, fine. As long as you work over the entire foot, you will find the areas that are stuck or injured, and you will be able to work with them until they come free.

¹ The most stubborn feet we ever worked on had no joints in the feet or ankles that could move in any direction. Working with those feet was like working with feet cast in cement. The patient was in his late 40s. It took three years of FSR before any of the joints started loosening up. Once the bones started loosening up, the progress was steady; all of the articulations moved freely and easily in just another three months.

How to know when the physical blockage is gone from the feet

Apply some tests! While a foot's ability to move in the directions described below is not a 100% guarantee that blockages are fading, it can be a fairly good indication that things are loosening up.

A healthy reflex

The foot has a wonderful reflex that it can do only when all of the bones in the foot are gliding across their articulations freely and easily. The reflex can be triggered with the following stimulation:

The patient should be lying down on a treatment table with straight legs. Place one hand over the patient's foot, with the center of the palm placed over the medial cuneiform bone of your patient's foot and the rest of the hand resting on the top of the foot wherever it's comfortable. Then, place your other hand, in a fist position, under the same bone.

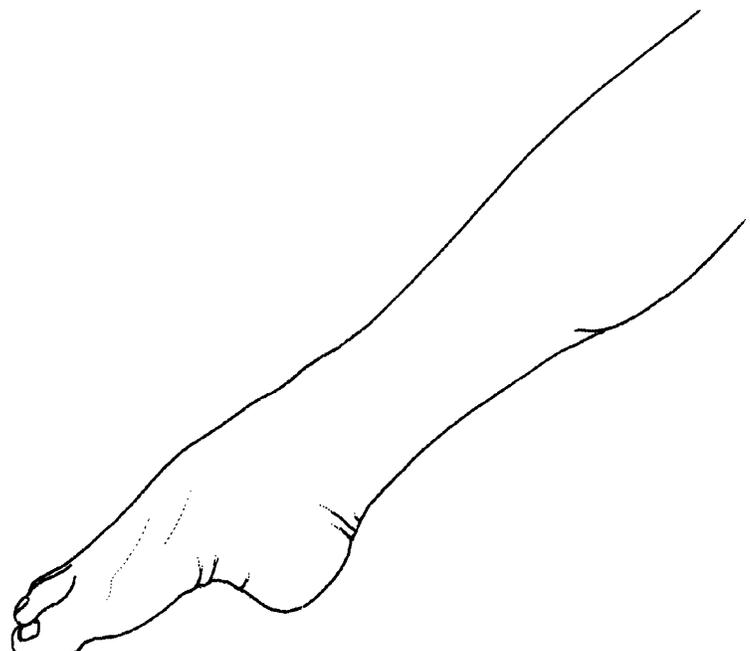
Press the hands together slightly and then release. This use of the word "press" refers to an actual, physical compression, followed by relaxation, as opposed to a mental, infinitesimally small pulse. Your hands should remain on the foot during the subsequent reflexive movement, if any. The foot, if its bones are all in the correct position and unhampered by tensions, may reflexively relax in two specific directions. The two foot movements are these:

1. The foot may stretch out as if the toes are being pointed like a ballerina. The center line of the top of the foot will straighten out, forming a straight line which is a continuation of the tibial crest. (See Fig. 37.2)
2. The foot may rotate, causing the toes to form a line that is perpendicular to the floor. The big toe will be the toe which is farthest from the surface of the table. The line from the big toe through the little toe forms the perpendicular line. (See Fig. 37.3.)

A straight line from leg to toes Fig. 37.2

A completely relaxed foot, when pushed quickly and gently in the arch area, might easily go into a pointed-toe position. If the ankle is also aligned correctly, the ridge of the tibial crest to the center of the foot will form a nice, almost straight line. If there is a problem in the ankle area, the ankle joint may form a concave dip instead of making a nice smooth line.

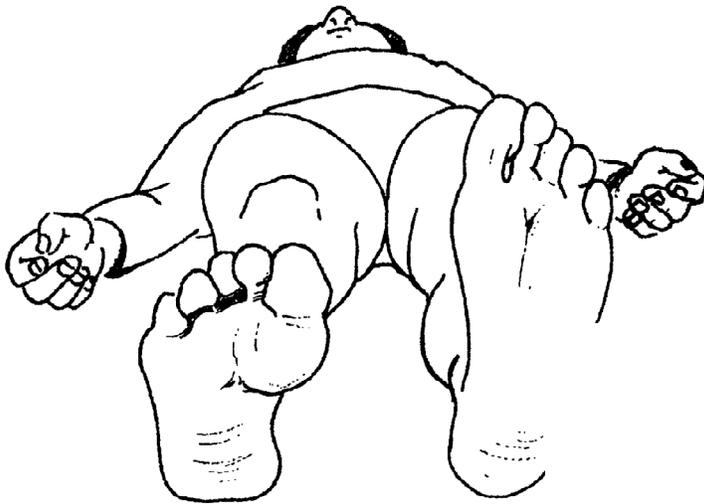
If a PD patient is doing very well, he will at some point be able to form this pointed toe posture on his own, without needing to be pushed in the arch.



If the PDer gets cramps in the bottom of his foot when he first regains the ability to point his toes, remind him that he needs to be sending energy into the muscles of the top of the foot as well as the muscles of the sole of the foot. If the muscles on the top side (dorsum) of the foot are also being energized while the toes are pointed, the muscles on the bottom (sole) will not be able to go into their seizing up routine. (Then again, any time the person dissociates from his heart, his foot may cramp up regardless of whether or not any injury is present. See chapter xxx.)

Outward rotation of the foot

In the starting position, the foot is pointing towards the ceiling, and midline of the foot is more or less in a straight line with the knee.



After being bumped gently but firmly in the arch, the foot may respond, if it is completely relaxed and flexible, by rotating laterally. The knee will not have rotated a considerable distance; the rotation will have come from mostly from the relaxed ankle.

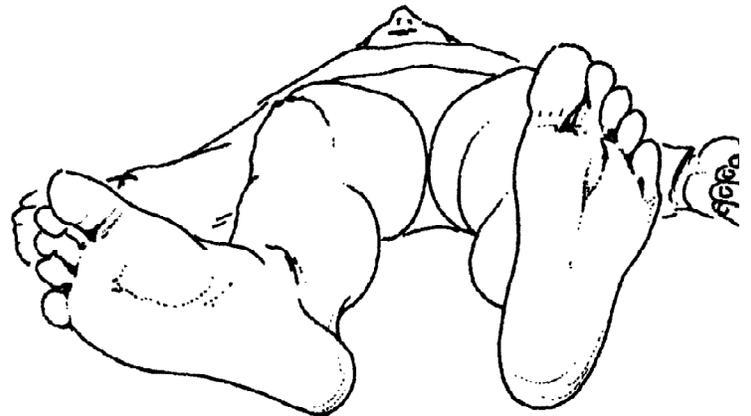


Fig. 37.3

If the foot is not relaxed, it might:

1. Not straighten out (Fig. 37.2), but will remain at more or less of a right angle to the *tibia*.
2. Instead of rotating laterally (Fig. 37.3), a foot that is still injured may reflexively rotate medially, *towards* the arch, as if protecting the arch of the foot instead of exposing it.
3. Also, if the foot is not relaxed, it may pull back on the toes, creating hammer-toes (see Figs 37.4 and 37.5). Hammer-toes are not uncommon in Parkinson's disease, and in many people who do not – and never will have – PD. Hammer toes are a sure sign that there is still tension somewhere in the foot, ankle, lower leg, knee, or even somewhere upstream from the knee.

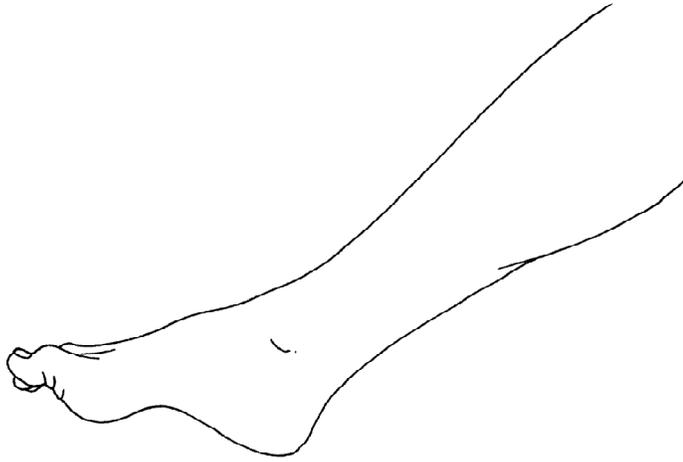


Fig. 37.4
Moderate hammer-toe of the big (first) toe

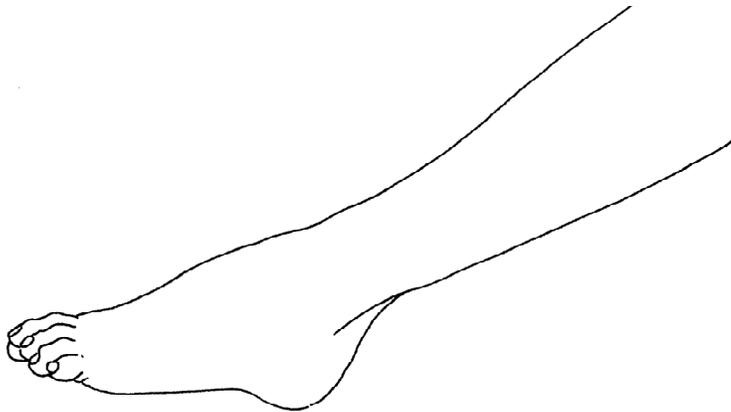


Fig 37.5
Mild hammer-toe of the second toe

Be very careful when you do this to insure that *you are not trying to influence the direction of the foot reflex*. Do your reflex pulse, and then be a passive observer of which way the foot wants to go. Sometimes it is hard to be impartial; after working for hours on a foot, it is

only natural that you will be secretly rooting for the foot to relax straight and long and rotate outward. But try not to impose your wishes on that foot. Do a realistic assessment of the reflex. When the foot responds correctly to this test, and the joints all seem to glide smoothly and easily, and there are no areas of the foot that feel somehow less than “correct,” you *may* be finished with working on the feet. If so, congratulations.

Flexible feet that still want FSR

But you may not yet be finished working on the feet. As you will read in the chapter on treatment plans, the real indication that you are finished is when Qi flows deeply and at full volume through the feet.

Sometimes, because of residual emotion resistance in the injured area, scar tissue upstream from the foot, scar tissue on the foot, or injuries in the toes that do not impede foot flexion and extension, all of which can impede the flow of Qi, you may not be finished with FSR on the foot in question. Because resting FSR can be an effective way to assist in emotional healing, sometimes FSR will still be beneficial even after the foot begins to resume flexibility.

We have received many questions that relate to FSR, including the very important question of how you can know when you are finished holding the feet. However, since so many of the questions and answers can also pertain to other issues involved in treating PD, these questions and answers will come later, in the section of the book that addresses treatment plans.

An observation from the classroom

When I teach this technique as a weekend class, the students spend the first day working on each other. By the end of the day, the students invariably assure me that they know what they are doing, they know what they are feeling for and they know just how the feet should feel. They are confident and ready to go.

The next morning, when we reconvene the class and the new PD patients are introduced, the students begin doing the FSR. Within five minutes, the classroom is a sea of puzzled faces. Finally, a student speaks up. “Could you remind me,” he might ask, “what is it we’re supposed to be doing?” or else, “My hands aren’t working today, I can’t feel anything at all. What should I do?”

The fact is, we are absolutely unused to holding with our hands a bit of living human limb or flesh that is so profoundly unresponsive. As noted before, some practitioners say that movement in the center of the feet of PDers feels corpselike. After ten minutes of holding their PDers’ legs and getting no familiar response, first-time students are usually convinced that the problem is their sudden lack of sensitivity and not the patients’ lack of response.

It is bizarre to hold onto another person and feel no response, no energy, no Qi. Or the Qi may be so chaotic or disordered as to make you subconsciously feel uneasy, or even queasy. If you have first practiced on healthy people, then when you get your hands on your first PD patient, you will realize the enormity of what you are going to try to do. You are going to try to restore living, throbbing Qi to an area which has been exiled from its own body for decades.

A person reading the above may be discomfited. “How can I tell the difference between a moderate lack of response from a healthy, but injured, person and the *acute* lack of response that you are talking about?” he might ask. To answer this, I will say, “Practice on healthy people.”

Practice, practice, practice: support, support, support

When you practice on healthy people, you are going to perceive some objective sensations of your hands moving in sync with the patient's responses. However, you will also be learning some deeper sensations that cannot be described in words. You will be learning to recognize with your intuition when some body part of your patient responds correctly or incorrectly. This can only come with practice. By spending hours and hours practicing what it feels like to support a person in such a way that he can relax, you will also be making yourself more comfortable with the process. As you become more comfortable, you will stop thinking quite so much about what you are doing. At some point, hopefully, you will stop interfering with the process via your thinking, and your intuition will step in. At this point, you will suddenly know where to put your hands, whether the area under your hands feels "right" or not, and you will know when to let go. You cannot know these things as long as you are trying to cognize what it is you are doing and feeling. Only by practicing doing and feeling can you become easy enough with this practice that you can start ignoring your thoughts.

Once you can do this technique without paying any attention to what your hands are actually doing, once your hands move quickly and easily to the places that need to be held without your mind getting in the way, you will find that your hands know exactly what to do.

Yet another baby example

As an example of this unlikely idea that thought-free, intuitive touching can be more effective than carefully considered, thought-intensive touching, consider the new parent trying to figure out where to put his hands to hold the new baby. That baby is likely to be screaming by the time the parent has found a position that should, logically, be most comfortable. Fast-forward three years and consider how the parent is holding his second infant. He grabs the newborn quickly and assuredly while explaining to the older sibling why baby can't be fed buttons. His grip on the newborn is easy and smooth, and the new baby feels safe in the gentle but firm, supportive grip of this experienced parent. As you can see, the premeditated, thoughtful grip is not always as effective as the supportive, intuitive holding that becomes second nature through hours and hours of practice.

The PD lack of response

In the same, intuitive way, you will come to be able to sense a profoundly disquieting, almost macabre stillness in the areas of injury in your PD patient. This stillness will be different somehow, in a way impossible to describe, from the mere lack of response that you perceive in other areas of his body. Even if his legs are unresponsive, they may still feel as if they have life inside. The stillness in the feet, however, may feel as if it is due to lifelessness, or even active repulsion of your hands.

When you are confronted with the stillness of the PDer's foot, you will stop assessing, at that point, and perform resting FSR. Resting FSR is sometimes called, jokingly, "doing nothing." The next chapter will tell you what "doing nothing" entails.



“How beautiful to do nothing, and then rest afterwards.”

- Spanish proverb¹

CHAPTER THIRTY-EIGHT

RESTING FSR

In this chapter I will use quite a few words to explain how to “do nothing.”

Doing nothing, also known as resting FSR, is what you will do in those key stubborn areas where your patient doesn’t respond or seems to want to be held indefinitely. When you find what seems to be the most stuck place on your patient, a spot where no movement occurs in response to any of your overtures, you will set your hands on the patient, support the area by using the correct amount of pressure in both hands, and then leave your hands there indefinitely.

More details

To be more specific, you will settle yourself comfortably in your chair, get your hands settled as nicely as possible into the contours of the patient’s skin so that you can best support this crucial stuck place, and, with your hands giving just the right amount of support (as discussed earlier in chapter thirteen: someone else’s baby), you will just stay right where you are.

When the area under consideration pushes you away or seems to no longer want to be held, then you can remove your hands and go somewhere else.

I suppose there might be something more I need to say about how to perform this technique, but anything I might say would just be more ways of stating the above.

How long should I hold

You will need to hold for as long as feels right: until the area starts to loosen up or until it pushes you away. Of course, if your arrangements call for a one-hour session and the area hasn’t started to respond yet, then, at the end of fifty-five minutes you will need to let go your hands and start wrapping things up. The next week, when you resume treatment, you may wish to do a little bit of assessment work, or not, and then go back to the area on which you were doing resting FSR and settle in for another session. On the other hand, if your intuition tells you that you need to work somewhere else, or if this week the area that you had been working on feels as if it is actively pushing you away before you even get settled in, then of course you will work in a different area.

As for how long it usually takes for the area to start to respond, that question is impossible to answer. Some feet respond in about fifteen minutes. One of my patients had an utterly frozen-rigid right foot, as mentioned earlier in this book, that required three years of patient holding before any of its articulations were able to flex in the slightest.

What can I do to accelerate the process?

Stay out of the way.

We have found that trying to impose love, light, or healing energy onto the patient definitely slows things down. The following case studies may help explain why.

¹ The saying and the attribution “Spanish proverb” was taken from a postcard printed by the Tushita postcard company, <http://tushita.com/tibetimage>.

Some PD case studies

Father Rickman

Father Rickman was driven to his first FSR appointment at my office by a member of his congregation. The driver, Ida, was a sweet, round-cheeked woman who was keen to tell me all of the wonderful work that the congregation was doing to the good Father.

“A group of us from the church get together with Father Rickman to do a healing-light ceremony once a week. And once a week we get together and do JoShinDo on him. We do group chanting and affirmations. I do Reiki on him once a week,” she bubbled.

“My gosh,” I replied, looking at Father. “You must be exhausted!”

“Why should he be exhausted?” asked Ida. “We’re simply filling him with energy.”

“No, you’re not.” I replied. “Father Rickman is having to work like a beaver to prevent all of your good intentions and vibrations and healing light from getting into his body. He probably dreads these sessions, but doesn’t have any polite way to tell you so. He would rather suffer silently through these sessions, working as hard as he can to repel everything you are doing, than offend you by telling you he doesn’t want your efforts.”

“That’s ridiculous,” exclaimed Ida. “He loves it!”

I turned to Father Rickman. “Well, Father,” I said, “how do you feel about those sessions?”

Father Rickman looked away from Ida and then turned his gaze on me. Then he stared at the opposite wall for about half a minute. Finally, avoiding Ida’s gaze, he said, “You’re right: I do work to resist it. But I really do appreciate that they want to help me. I don’t mind putting up with their treatments; it makes me feel good to have them feel good. They want to feel they’re doing something helpful, and who knows, maybe it’s working.”

An awkward silence squatted in the air. I tried to break the tension by explaining, “He’s got Parkinson’s, you know. Most people with Parkinson’s typically don’t like being messed with. They are defensive about the injuries that are lurking in their bodies. They don’t want them exposed to all the world. Also, if there is something wrong, they usually don’t trust anyone to fix it; they’d rather do it themselves. But at the same time, they don’t like to make waves, as a rule. They will go along with whatever makes other people happy, even if they suffer inside.

A PDers strong dislike of “being messed with” or aided

While not every PDer is completely antagonistic to being touched, most of them are highly antagonistic to being molded, altered, influenced by factors beyond their control. This chapter is not the place to go into depth about the Parkinson’s personality, but the Father Rickman example is very, very typical. Even those PDers who have taught themselves to enjoy a bit of massage or who have learned to tolerate well-meaning, generalized attempts at “energy work” are still extremely guarded in their injured places with a ferocious protectiveness, a protectiveness that, unbeknownst to the PDer, prevents even himself from being able to mentally visualize the injured area or even imagine *himself* sending healing light into it, let alone anyone else.

Imelda

I had one patient who was very affectionate. I had known her for many years before she became my patient. She loved to hug and kiss all her friends, she snuggled constantly with her

children, she was a big proponent of loving, physical contact as a way of healing the little hurts of everyday life.

I was surprised then, when I started to work on her feet, that she drew her feet away from me and pleaded, "Please, be careful; I am so scared. I've never even let Seamus (her husband of fifteen years) touch my feet, not ever. I don't even like him to *look* at my feet." This fear or concern of having the feet touched or looked at is not uncommon in PDer's.

"I can't do this"

I had one patient who drove all the way up from Los Angeles, a trip of over three hundred miles, to be a demonstration patient at a weekend class that I was teaching. She sat through the first half of the day, but when it came time for her to lie down on the table and let me demonstrate FSR technique by holding her right foot, she started crying.

"I can't do this," she whimpered. "No one has ever touched my feet. No one has ever seen my feet. I have to go. I'm so sorry." She picked up her purse and walked out the door. I never saw her again.

TJ

I had one patient, a horse trainer, who could smoothly and easily lift you off the floor with her right uppercut if you made a threatening move at her. She was radiantly happy when she was mixing things up with the rowdier sort of cow or cowhand. But when came the time for me to start holding her foot, she found herself reduced, for the first time in her life, to a sobbing marshmallow.

At our first session, as my hands moved in slow motion closer and closer to her left foot, she stopped crying and started screaming hysterically. She alternated between apologizing in a perfectly calm, almost laughing voice or screaming at the top of her lungs, writhing, and pulling on her hair for the duration of the session. (Many is the time I've thanked the powers that be that my office is fairly soundproof.)

I never did touch TJ's feet that first day. The closest I could get to her feet was about nine inches away. It was an unforgettable session: I sat on my stool, my one hand up in the air about nine inches away from the top of her foot, my other hand suspended in the air as well, nine inches away from the bottom of her foot, while she either screamed or apologized for screaming. It took almost eight weeks before I was able to actually place my hands on her feet. Every week I got a little closer, and every week her screaming got slightly more under control. When I finally was able to rest my hands on her feet, she didn't scream; she sobbed as if her heart would break.

Lynne

Lynne was a body worker herself. When she took a craniosacral class she was terrified to let the other students in the class practice on her. She finally teamed up with one classmate, an old friend whom she trusted deeply, but even so, she was edgy throughout the class. Considering that most light-touch practitioners consider Upledger's craniosacral techniques to be the gentlest of the gentle, Lynne's level of fear and resistance might have been surprising. But since Lynne had PD, I was not surprised at all when she told me about her fear of having someone "do things" to her.

By the end of the weekend class, Lynne was exhausted from resisting, with all her mental strength, the techniques that her friend gently perpetrated on her.

Craniosacral therapists often tell me that I am wrong: “Craniosacral work is so gentle, the patient can’t even feel what’s being done.”

I’m sorry, but I must beg to differ. Many of the PDers I have known have *hated* craniosacral work. The fact that it is so well-meaning and gentle makes it seem somehow even sneakier, ever more insidious and, thus, something to be even more staunchly guarded against. These PDers can physically resist strong, Yang-style therapies such as Rolfing, but they have to engage their mind as well in order to resist the gentler techniques such as craniosacral. This means that craniosacral or other gentle and light-touch work can be even more trying, even more exhausting than the brute force techniques.

These case studies are merely the tip of the iceberg, but I think they make the point that most PDers don’t like other people imposing themselves on their (the PDers’) feet. Most people with PD are tough, indomitable and stoic, or were at the time of their injury – and the injured, terrified person is the one you are treating, not the logical person who is in your office. PDers, for the most part, and certainly in the part of them that is still scared and injured and hiding, really don’t like to be toyed with, helped, loved, supported, healed, or messed with in any way. Their battle cry might as well be: “I’d rather do it myself.”¹

Irony

The ironic nub of the situation is that, even though they’ve done so much with their lives, they can’t do their own healing by themselves. It almost seems as if *admitting* that they need, want, and dread the healing presence of another person is a part of the emotional healing process.

Again, keep in mind that the admission that help is needed has to come from the child/young adult who is still lurking in the background with an injury and an injured state of mind, not the thoughtful adult who is telling you that he really does enjoy massage. This dread of the very necessary physical hands-on part of the treatment can contribute to the challenge of recovery.

The mental part of the recovery from Parkinson’s, in which the PDers must retrain their minds to acknowledge their injured body part, is addressed in a later section of this book, but I can say here that their mental resistance to opening up and allowing healing to occur in the area of the long-suppressed injury can be ferocious.

The self-control and self-reliance of Parkinson’s

Most PDers will be quick to tell you that self-reliance and/or self-control has been a crucial factor in their “success” in the world. When they learn that their extreme level of self-reliance or self-control is a normal part of the Parkinson’s pathology and that their treatment is

¹ Sometimes there is also anger being held inside, accompanied by subconscious fear that the anger might come out. One patient, telling her sister about our program, was stunned when her sister said, “Nothing is going to help you until you get rid of your anger.” The recently diagnosed PDer was flabbergasted. Never in her life had she spoken an angry word or responded with anger in any situation. She was the most compliant, easy-going person she knew. She was so shocked by her sister’s words that, later that day, she locked herself in her bedroom and said out loud, “If there is any anger in my body, I want to behold it right now.”

A month later, when she was telling this to me, she said, “A moment later I could see black clouds of rage billowing out of my body. I don’t know if would have been visible to anyone else, but to me, the black clouds filled the room with horrible, choking smoke. That smoke was full of my rage at my parents, my teachers, my children, my husband, my siblings, my pastor, my relationship with God. I was seething with fury at a thousand suddenly-remembered insults, unfairnesses and hurts. And I swear, until that moment, I didn’t even know that I had any anger at all in my body.”

going to involve learning to climb down from their self-reliance throne and accept physical ministrations to their foot from another human, they usually have one of two responses: they see the irony and perfection of the situation or they become obsessed with a self-pitying litany of all the situations in their lifetime in which life was harsh or they were treated unfairly. In either case, they are still usually resistant to opening up their hearts and minds to their own injured area.

Which brings us back to why this extremely Yin form of Tui Na, resting FSR, is used for treating PDer's. With resting FSR, the injured person and the injured area are being held. That's all. The injured person, in response to this quickly undetectable support that is accompanied by complete emotional detachment on the part of the practitioner, finds himself in the position he has long been looking for: a little time to be by himself in a safe setting so that he can get around to mentally, emotionally or somatically (with cellular feeling) looking into that old injury that he put on the back burner so long ago.

When a person is receiving resting FSR treatment, the part of his body that is being supported is essentially "all alone, but being protected." While receiving this type of supportive holding, there are no demands being placed on the patient's physical body, there are no demands being placed on the mind or the emotions of the patients. The practitioner is performing in the role of Human Poulitice. The long-awaited conditions necessary for healing are being met: respectful treatment is being given, the injured area is not being threatened, the emotions that were used to block the injury are not being threatened; in fact, nothing is being threatened.

As noted in the previous chapter, *a person cannot relax and cannot let go if he is busy defending himself, however silently and invisibly.*

Emotional relaxation

When a person no longer needs to interact or defend himself, he can relax. If this PDer was at home, ostensibly able to relax, he would not do so. Instead, he would probably feel the need to be doing something, possibly sleeping or making himself helpful around the house. Most PDer's like to stay very busy and productive. It is not unusual in my experience for a PDer to hold down three jobs, at least two of them being full-time jobs. It is not necessarily for the money, but "Because I can" or because "Someone needs to do it and I'm the best person for the job."

It may be that they are trying to stay busy to avoid having to ever look too closely into their past. For many of them, they have been supremely productive as a way of fighting a life-long impossible battle to prove their worth to some humiliating childhood memory. The average PDer does not, cannot relax very deeply for very long. Even if he did relax, the part of his body that is injured would be on guard and wary.

When the PDer is lying on his back in the therapist's office having his foot held, he realizes that there's nothing practical and distracting that he can be doing at that moment. If the therapist is not challenging the injured part of him in a physical, emotional, or mental manner, the PDer may, if he is supported just right, begin to relax the emotional barrier that has long been protecting his injured area.

Physical relaxation

Many PDer's have asked me if they can't get the same benefit by doing mental Tui Na on themselves. Others want to know if their spouse or friends can't just do the Tui Na mentally, thus avoiding the tedious, hands-on work.

The hands-on aspect of resting FSR is crucial. The physical support being received by the tissues causes them to relax over and over again, getting slightly more relaxed each time, as they try to maintain what they think is their normal level of tension. What the tissues don't realize, if the FSR is done correctly, is that the hands of the practitioner are, over time, subtly supplanting the tensions that are usually in force in the injured area. The net result, after an hour or so of continuous relaxing, is that the tension that's historically been holding the injured area rigid is now being performed by the therapist. Meanwhile, even though the net amount of support and tension in the injured area hasn't changed, the tissues in the foot are completely relaxed: the health practitioner is holding it all together.

When the part of the mind that normally protects the body is relaxed, and the tissues of the injured area are relaxed, the injured person has finally gotten himself into the situation he has long been waiting for: a little quiet time, during which he can mentally/emotionally attend to whatever it was that happened so long ago, that event that was put on the back burner to be "dealt with later." Finally, in the office of the health practitioner, "later" has arrived.

Once this situation is set up, the injured part of the body can, if it wants to, spit on its hands, haul up its slacks, and get to work on healing the problem area in the foot. It may take some time, since the mind can barely even remember that it has a foot. But the warmth of the practitioner's hands, the tiny movements that the practitioner may be unconsciously making (more like gentle sighings than actual movements), these little indications of supportive, non-judgmental human contact draw the patient's attention to the area in a non-threatening manner.

Eventually, over hours or years, the mind will start noticing there is a foot, that there is something wrong with the foot, and from there, the foot healing can commence.

Exceptions to the rule

Of course, there are a few patients who are not as terrified as the patients described above. Also, some patients learn to lose their fear after a few sessions or a few years of being worked on. Very often, a patient will deny that he is afraid of being held, and yet, after the feet begin to relax, he may start crying and even start reliving some of the emotional events that prevented him from healing. He may suddenly, or over a few weeks, realize that he has, in fact, been living his life with a part of himself stuck on "extreme alert" even if his conscious mind was trying to create an image of his being self-possessed, calm, or complacent.

Now that we've been doing this work for many years, we have learned to not accept at face value a patient's assessment of his own fear levels. No matter whether or not a patient tells us that he is open and unafraid, or terrified and distrustful, we treat all patients with the same caution and respect. We always ask permission before we start. We always assume that the patient should be treated as if he is injured and afraid. Even if we are laughing and joking around, the underlying principle is that the patient's body is sacred and we are mere servants in the temple.

How much mental attention to give the patient

As noted previously, we have found that sending mental images of love, support, and healing has a detrimental effect on the healing process.

Just as the best physical support is the kind of support you give someone else's baby, the best mental attitude for treating PDers is the one in which you mind your own business.

A member of the PD Team said once, “Sometimes I start daydreaming while I’m holding someone’s foot. When I stop daydreaming, I realize that I’m holding his foot more gently than usual, and it feels just right. If I try to change and use more pressure right then, it feels wrong.

He continued, “Sometimes, still working on the same person, when I stop daydreaming the second time, I realize that I’m holding his foot with really powerful pressure, and it feels just right. If I try to relax my hands at all, it feels wrong. The most important thing is to give the patient exactly what he needs at that spot at that moment. I find that I do the best job, the patient seems to get the best releases, when I’m not directing my thoughts directly at the patient.”

The poetry example

The best way I can explain the degree of emotional detachment that you want to use on your PDer is the poetry example.

When we were running the free PD clinic at the college where I teach, each student would work on the same PD patient for two semesters. The students understood that, while doing resting FSR, they were supposed to be emotionally detached from their patients. The students were not supposed to be giving mental suggestions to the feet as to where, when, or how they should wake up and start moving. The students also understood that they were not supposed to be sending “healing energy” into their patients.

The students usually felt that they were doing a good job of keeping themselves distant from their patients’ emotions during the resting FSR work.

About three months into the semester, I would introduce an experiment, without explaining my purpose to the students. At the beginning of this particular clinic session, I would wait until each student was sitting down quietly, resting his hands on his PDer’s foot. Then I would pass out to each student a sheet of paper with an uplifting but fairly long and somewhat obscure poem – usually something from Shakespeare’s sonnets or the Rubiyat – and tell the students that, while holding their patient’s foot, they needed to memorize the poem. I would tell them that they had twenty minutes to memorize as much of the poem as they could, all the while holding onto their patients’ foot. Then I told them that, at the end of twenty minutes, they, the students, were going to have to stand up and recite their poem in front of their fellow students and all the patients.

After the usual exclamations of resentment or protest, which I would ignore completely, answering only that, “The clock is ticking,” the energy in the room would change. Usually, a peaceful silence prevailed in the large clinic room with seven patient tables placed around the perimeter. When the poetry assignment was given, the energy of the room was more charged, yet even more deeply still than before.

When the twenty minutes were up, I told the students to stop holding the PDers, and stop looking at the poem. Then, instead of having the student recite poetry, I went around the room, asking each patient in turn how this particular treatment felt, compared to the treatments they had been receiving for the last few months.

The students were often insulted by the patients’ responses. For the most part, the patients said things like: “This session was by far the best I’ve ever had,” “I felt a warmth surging through my legs that I’ve never felt before,” or “Something seemed to change inside of me. I almost felt like laughing or crying.”¹

¹ The reason that I qualified the reactions in the above sentence, saying “for the most part,” is that, once in a while, a nervous student would have lost all sense of what he was doing to the point where his patient afterwards

The most interesting thing was the student responses. They were usually defensive, and somewhat bitter. After all, the patients were saying that this treatment, during which the students were paying no attention whatsoever to the patients, had been the best treatment yet. It usually took me five or ten minutes to explain to the students why it was that they felt so insulted by the whole thing. They were insulted because, even though they had wanted to imagine that they weren't mentally invested in their patients' responses, in fact, they were invested, and deeply. Even though they were trying to be detached, they were making a point of their detachment. They were still focused, despite their determined lack of intent, on helping the patient.

When the patients stated that they felt and experienced more, had almost miraculous changes, or simply enjoyed the treatment more when the student got out of the way, the students felt miffed. The student response might be summed up as, "You prefer it when I am nothing more to you than a pair of hands? What about how much I care about you? Don't you realize how hard I've been working at not imposing my own ideas on you? How dare you say that you prefer my touch when I act as if you're just a piece of meat!"

And yet, after the students calmed down, they did absorb the point of the lesson. Their previous detachment had not actually been very detached. The students were, despite their desire to be providing unconditional support with no expectations, expecting *something*, however subtle. Even that very subtle degree of emotional involvement had been detrimental to the treatment. This only became obvious when, in panic and fear, the students had been intensely absorbed in something even more important, at that moment, than the patient: the dread of having to recite a poem from memory in front of the class.

Focusing on the blue sky

The best attitude on the part of the practitioner's hands is one of utter alertness. The hands should be utterly responsive to any change on the part of the patient so that if the patient's foot, ankle or leg moves, the hands can follow the patient perfectly.

The mind should also be utterly alert. However, the mind of the practitioner should be alert and working on *something that is personally important to the practitioner*. Hopefully, anyone who is doing much healing work has some background in meditation, silent chanting, visually focusing on some inspirational image, or some other regular experience with uplifting subject matter such that he always has something that he can be focusing on with all his power of concentration. You may recall the story of Shinzo Fujimaki, the shiatsu teacher in chapter xxx, who focused on the blue sky.

The expression "mind your own business" applies. The business of the practitioner is not always to be mentally focused on the patient. The business of the practitioner, the business of all people, is to always work at becoming a better person. For a practitioner, doing resting FSR is a wonderful opportunity to focus hard on self-improvement. This is much, much harder than it sounds. I remind students of this when they tell me that they feel uneasy charging money for "doing nothing."

Getting paid for "doing nothing"

Many practitioners, especially after taking a weekend class, ask me how they can possibly justify charging money for doing nothing. I have to point out to them that they have just

would say, "My student had such a painful death grip on my foot I thought it would turn blue and fall off!" These patients did *not* prefer the poetry experience.

spent two full days realizing that their preconceived idea of doing nothing was not correct, and that when they were, to the very best of their abilities, “doing nothing” to their patient, it required enormous mental focus and restraint.

Most beginning practitioners have a very difficult time “doing nothing” for an hour at a time. If the mind is not accustomed to this level of discipline, the practitioner finds that his mind refuses to stay away from the patient. Also, a person who has not disciplined his body somewhat may not be able to hold still for more than a few minutes at a time. While the essence of this work is not difficult, the application requires discipline. As with any discipline, the student improves with practice.

Singing

Continuing on with the poetry/mental detachment aspect, I have tried other experiments; once, as an experiment, I had the students mentally (silently) sing songs while holding a PDer’s foot, to see if that would be a positive experience for the patients. After that one experiment, the patients reported nothing especially good, and several reported that they felt agitated by the unconscious rocking that their students were doing. I have not repeated that experiment.

Talking during treatments

For my part, I often engage the patient in conversation, especially during the first few treatments. I find that, by diverting the patient’s conscious mind away from the feet by chattering, while simultaneously keeping, to the greatest extent possible, the silent part of my own mind focused on my morning’s inspirational reading, I am able to provide a maximum level of security for the injured area.

The injured area, assuming that I am busy conversing with the head office, is less likely to perceive me as a threat. Meanwhile, as long as the patient’s conscious mind is busy answering my questions, it is not contributing its usual internal dialogue of negativity about how it doesn’t like the foot to be held, or whatever the case may be.

As I sit there nattering away, and the patient is explaining whatever he needs to explain, the injured foot is experiencing the sensation of being held in just the very way that it always thought it should have been held, so long ago, when it was hurt. The injured area is not having any attention paid to it. It is just being held. My hands are at peace, they are resting on the injured area, and they are giving it so much support that the foot may feel safe enough to relax, just a tiny bit, for possibly the first time in decades.

And yet, though my mind is minding its own business, my hands are able to notice if anything changes. Just as a really good fisherman, sleeping on the banks of the lake, knows instantly when a fish has bitten his bait, my hands know exactly how to respond if the patient’s foot makes a move. Just as a busy and preoccupied parent does not miss a stride while he expertly moves his hands to accommodate a shifting child that is sleeping on his shoulder, my hands, resting on the patient, move on their own, without my mind getting in the way. It just takes practice.

Many a patient has been surprised when, in mid-sentence, I will suddenly say, “Aha!” Or “There now, did you feel that?” Very often, the patient, and sometimes my conscious mind, was completely unaware that things were shaking it up down in the foot. But my awareness in my hands conveys to me, when it needs to, that something has shifted. At this point, I might stop my chatter and reassess the situation using the more dynamic type of FSR described in chapter xxx.

Then, within a moment or two, I might go back to “doing nothing” again – with or without some level of small talk.

It takes a tremendous amount of concentration to keep the hands thoughtlessly, but responsively, doing their job, while also keeping one’s thoughts on a strong internal focus, all the while answering questions from a patient.

Returning to the issue of the practitioner who asks how he can possibly charge money for doing nothing, I might reply, “You are attempting to do work that is extremely difficult, work that few people can do, and work that few people want to learn to do. It can take time and mental focus to even begin to master this work. You are providing a singular service. It is reasonable to charge money for this work.”

Finding a practitioner

This brings me to the subject of finding a practitioner. While this subject might not seem appropriate for a chapter on technique, you shall see that it is not unconnected. As I explain what I have seen of the patient-practitioner relationship, it may become apparent that the subjects of “how to do this work” and “who should do this work” are actually closely related.

PDers, when first seeking a practitioner, usually want to find someone who is highly experienced in FSR. However, this type of bodywork is not yet common in the western countries, and only beginning to come out of hiding in the east. Therefore, a lot of information is posted on the PD Recovery website on the subject of finding a practitioner. But I would like to add a little something to the practical suggestions that are posted.

Most of the PDers that have visited my program from afar have brought with them the person whom they recruited to learn and perform FSR. These practitioners are very often new to the entire field of light touch therapy. These practitioners have been, almost without exception, very capable and deeply inspired people. They do understand what FSR is all about. Most of them tell me that they have read between the lines of my text and that what they have read resonates with something they had already known.

It also seems that the patient-practitioner relationships that I have seen have, very quickly, developed into a significant, beneficial relationship for both parties. Even when the practitioner is the spouse, a new dimension in their relationship develops.¹ Without wanting to sound too mystical, it almost seems as if the practitioner-patient relationships we’ve seen were relationships that were, for some reason, meant to be.

They have come about in this way: the patient usually had to give up on the idea of finding someone who already knew FSR. He had to fall back on making inquiries of local physical therapists or friends who knew someone who knew someone who was interested in massage or acupuncture. After some amount of work on the part of the patient, he connected with someone who was interested.

¹ I am not advocating one way or the other for the spouse to be the practitioner. It has occurred that some spouses are keen to do the FSR. Others sense that they can or should do a little bit, but that they should probably not be the primary FSR provider.

In general, it is very, very difficult for a spouse to attain the necessary degree of emotional detachment. Even some of my colleagues, very experienced FSR providers, find that they cannot work as effectively as they would like on close friends or family members; they realize, while trying to do FSR on these loved ones, that they cannot let go of their emotional involvement. They can sense that their treatments on family and friends do not have the same detached ease that they attain with their other clients.

So that I do not paint the patient-practitioner relationship as being more one-on-one, exclusive, than it is, I will also point out that, very often, the practitioners are fascinated enough by the work that they go on to seek out more PDerS.

For example, I know one FSR practitioner with a supreme sense of detached touch and a sixth sense of what area needs to be worked on. He is rapidly becoming an effective FSR practitioner. He only started learning this work because it was a source of extra income. Within six months of practicing FSR on several patients with good success, he is considering becoming an acupuncturist and full-time health practitioner. He told me, “The almost miraculous changes that I see in these patients make me think that maybe I have a talent for this. And while I’m pretty sure that the last thing I really want to do with my life is spend my years holding people’s feet, I’m also starting to realize that, if I can do so much good in the world by sitting still and holding, then maybe I have some sort of higher obligation to make this my life’s work.”

The above is actually nothing more than a long aside, but it may serve to explain, better than my other attempts, that performing resting FSR sometimes seems almost like a calling. Certainly, doing this work changes both the patient *and* the practitioner. In light of that, it may be not so important that a patient find an experienced practitioner. It may be more important that the patient find the *right* practitioner. And so far, from what we have seen, patients have, almost invariably, found the right one.¹

HOW NOT TO WORK ON A PDER

Some rules for FSR practitioners

These next two pages include some important admonitions for budding health practitioners. These pages address the very common problem of people starting to imagine that they have healing powers in their hands. The techniques that you are going to be mastering are so seemingly mysterious that they were banned in China in the 20th century for being too charismatic. Don’t forget: in centuries past, people were burned alive if they were able to evoke powerful responses in sick people simply by using their hands: they were in the employ of the devil.

The problem is, when one sits still, trying to be detached from the patient, the temptation is always there to mentally try to look around inside the patient, to move things around using mental energy, perform psychic surgery or any number of other highly invasive, energetic techniques.

Just as these techniques can be very impressive to some observers or patients and terrifying to most PDerS, they can be dangerously ego-boosting to the practitioner.

I find myself in a difficult position. By teaching these techniques of very Yin Tui Na in a book for the general public, I am keenly aware that I cannot be certain that the usual teacher-student admonitions are being conveyed. These admonitions would be warnings to never participate in any of the types of therapy in which the health practitioner uses his own energy to try to fix the problem of the patient. Familiarity with very Yin Tui Na techniques can sometimes

¹ We do hear from people who say that they suspect that their FSR practitioner is not the right one: the therapist is not following the protocol, he is not going slowly, or carefully, or it just seems like something isn’t right. We need to point out to the patient, in cases like these, that evidently this particular therapist is not the right one. We like to point out at the same time that the patient knew, via his own intuition, that the practitioner was not the right one. Reminding the patient that his knowing is an indication that he does know, on some level, the right practitioner from the wrong one, we then suggest that he continue his search for the right one.

give a person the realization that he can, especially with a weak-willed patient, use his hands to perform healing. From realizing that he *can* do this, sometimes it is only the merest of steps before the practitioner thinks that he *should* do this.

Patients who are manipulated via the energy of others do not thrive in the long run. Practitioners who imagine that they are receiving instructions from the heavens to invade the privacy of a patient's skin are usually greatly mistaken.

Historically, techniques such as the ones I am writing about were only taught to health practitioners who had studied alongside a teacher for many years. That teacher usually gave students injunctions as to when they should and when they shouldn't use certain techniques. Since I am sharing this material with the general public, the warnings and admonitions must be included here.

While it may seem as if these warnings are on behalf of the patient, that is only superficially the case. The real danger of these simple yet powerful techniques is to the ego of the health practitioner.

Looking under the skin

Sometimes, if we are holding a patient just right, we can feel what is going on under the skin, as if our hands were X-ray machines. I used to teach that this was a reasonable thing to observe. I no longer think that it is, especially in a course for the general public.

Now, I teach something else: when I teach a workshop, I have the students do the following while partnered with a fellow student. I ask them to put their hands on the sides of their partner's arm. Then I ask them to imagine that the atoms of their hands are so far apart that their hands can slip down inside the partner, in between the molecules of the patient's arm. Once inside, I ask them to imagine that they can discern the various muscles, bone articulation, and anything else that they fancy. I ask the partners to notice whether or not they can feel these invisible hands.

If I guide the students through this exercise slowly, and explain to them that all they are doing is adjusting their mental attunement to the vibratory rates of different tissues, all the students are able to do it.

I put them through their paces. I ask them to mentally imagine that they are placing their hands on their partner's radius (arm bone), and then gripping the ulna (another arm bone) with their invisible hands. The partners can feel the sensations inside the arm as the practitioners do these exercises.

Then comes the important part of the class.

I ask the students if they were able to do it. They are usually all looking very pleased with themselves, because they have done something pretty unexpected. I ask for a show of hands of the people who were able to do this. All the hands go up.

At that point, I look around and nod my head a few times, for effect. I really want them to hear what I am going to say.

"You were all able to do this exercise. That proves that the ability to do this is not a special gift. You are not special because you can do this. Just the opposite; you are normal. However, as you know, in normal society, in our culture, we do not do this. To do this is considered rude and invasive.

"Many people, when they discover that they can do this, start to imagine that they must be very special indeed, that they have a mission in life as a psychic healer. Nothing could be

further from the truth. Everyone can do this, as you have just demonstrated. But as a culture we agree that we do not do this.

“The skin is the organ that is designed as a barrier. When you mentally probe inside the skin, the person being probed has no defenses. You are invading his body, and his body has no defense against your mental probe. This type of work is invasive and dehumanizing.

“It is not your job to get under someone’s skin and fix things. That person’s own body can fix things. Our job as health practitioners is to inspire our patients’ own innate healing force to rise to the job. We can do that with acupuncture needles, herbs, western medicine, surgery, whatever the patient thinks it will take for him to be rid of whatever is blocking him from calling in his own healing ability.

“Never intrude into a patient’s private spaces under the skin. Thank you.”

The yoga sutras of Patanjali

The great sage Patanjali of India, at about the same era as Socrates in Greece, wrote a brief book outlining the various stages of spiritual progress. He wrote about the various abilities and matter-conquering attributes that mark these stages. He wrote that in one advanced stage of spiritual development, a person will be capable of healing others. *But* that wise person will choose not to do so. Unless commanded by God, usually for some obscure reason known only to the heavens, the saint will not perform such a feat.

The saints and sages know that when you perform a healing on someone, you have not done that person a favor. Instead, you have weakened his will power. The patient will still have the wrong thinking that allowed his illness to thrive in the first place.

When these patients are cured via a miracle, they have not learned to do the work of battling their own weakness; they have instead started or reinforced a habit of relying on others to do their work for them.

Most saints and sages of every faith are familiar with this principle. Once in a great while, a very humble saint will be instructed by God to perform miraculous healings. This is not necessarily for the health benefit of the person healed, but for the benefit of certain people who are lacking in faith. After, all the bodies healed by great masters still must someday feed the flames of cremations. The lasting miracles are in the spiritual growth inspired by the great ones.

Sorry to be going on about this, but as a teacher in an acupuncture school, and as a lecturer for practitioners of alternative medicine, I run into a steady stream of would-be miracle workers.

I like to remind them that, when they are truly saints, and God is telling them whom to heal and whom not to heal, they will no longer be attached to the fruits of their actions. As long as they *want* to be a healer, they are ego-bound and their actions will be prone to error. As long as they think that they are the doer of a healing miracle, they are doing it wrongly.

Again, going back to the beginning of this section, everyone in the classroom is able to do the various “psychic” tricks. Therefore, these abilities are *not* special. What is truly special, what is very rare, is learning to mind your own business.

And what is your business? Your business is not healing patients. That is the patients’ business. Your business is knowing your own soul, being a master of your own consciousness. If you become such a master, then by proximity your patients will feel a sense of deep peace and love such that they may be able to summon up their own healing powers. Heal yourself, that’s your business.

As for the rest, if you are training to be a doctor, do what you learned in school, and do it to the best of your abilities. Don't go showboating. By just doing what you were taught in school, without trying to add your own variations, you will learn humility. Humility is the first step in becoming a master of your own soul. Don't worry about whether or not the patient gets well. You do your work to the best of your ability, and in this way you may inspire your patient to do the same for himself. When the patient begins to physically, mentally, and spiritually take charge of his own health, then lasting healing will begin.

A summary of the don't's

Never impose your own healing "vibe" on a patient.

Never imagine that you put your Qi into a patient.

Never imagine that you are a healer.

Never imagine that you have been divinely ordained to do healing work. Don't worry about letting God down; if God is determined that a patient shall get better, that patient shall recover no matter what you do.

A summary of the do's

When working with PDers, your job is holding their feet. You need to keep your hands alert to the various sensations that are received by your hands, and letting go at the right time. Your other job is minding your own business. Any moving, healing, or changing that occurs in the patient is the responsibility and choice of the patient.

This is a lecture that I give to all my students, not just my FSR students. This lecture is even more important for people who are going to work with PDers. PDers, more than most anyone, are wary. They really would rather be healing themselves. They don't really like the idea that some friend or stranger is going to be working on them in a manner that will unlock doors that they can't consciously unlock. PDers are so extremely guarded.¹

¹ A patient from across the country came to visit me again after having been to an acupuncturist who lived in his area. The local acupuncturist had assured him that he was going to perform Tui Na and nothing more. However, when the therapist started working on the PDer's shoulder, he exclaimed that there was a psychic tear in the shoulder that needed to be "sewn up." He proceeded to take it upon himself to do the sewing.

When I saw the PDer, I asked him why his arm was so tense; the last time I had seen him he had been doing so well; at that time, his legs and feet were already healed and all that remained was some tremor in one arm. Now his right arm was strangely rigid, he wasn't using it at all, and he even told me that it was OK if he never used the arm, that he could get by with one arm from now on. When I asked him what the heck was going on, he sheepishly told me about the psychic sewing job.

I asked him if he had wanted this work done. He replied that he hadn't, but that he didn't know how to say no. Now he was ashamed of himself, and he felt so bad about his shoulder that he didn't care if he never used that arm again. I was both furious and deeply pained; this was the same person who had needed three years of work before his foot had finally loosened up. It had taken three years of confidence building before he had allowed his foot to respond to me, and now, here he was, admitting to being scared and ashamed after having been shamefully invaded.

We had a talk about whether or not I should go into the shoulder and remove the psychic stitches. While that might help, it might also be perceived as yet another invasion. It was a difficult situation. I greatly resented the unprofessional, disrespectful behavior on the part of the distant Tui Na practitioner. This sort of abusive practice is the very reason that most PDers do not like to be messed with.

We ended up deciding that I would support his shoulder using Yin Tui Na and he could, if he wanted, remove the stitches himself. He felt very unsure as to whether or not he was successfully doing this. After this, he never again showed any interest in recovering the use of that arm.

When you work with PDers, you will only get results if the PDer can be certain that you are not going to do anything to him. Only when you have shown yourself to be a complete respecter of his person will he be able to relax enough to let you do your supportive work.

Talking to the feet

Now that I have drummed into you the idea that you should not get involved in any way with your patient's wounded self, I am going to contradict myself.

Sometimes, we find that the body part will respond in a favorable manner if it is addressed respectfully, honestly, and briefly.

For example, I sometimes will talk directly to the injured foot when I first start working. After having asked the patient out loud for permission to work on the feet, I then introduce myself, either silently or out loud, to the foot or body part in question. Sometimes I also add that I am going to be holding the foot (or wherever) for as long as it wants me to. I sometimes add that I have received permission from (the name of the person) to be doing this. I may add that the person (I refer to the person by name) wants me to be doing this work, and wants the body part in question to be able to relax and/or heal itself. Sometimes, I then tell the body part that I am going to leave it alone while I hold it, and I tell it to "do whatever you want to do while I'm holding you. If you get scared, I'll be right here. If you don't want to do anything, you don't have to, but if you do want to try moving around or taking advantage of my holding, you only have so much time (however many minutes are left in the session)."

After that, I stop talking to the feet and leave them alone.

You will notice that this conversation with the feet is not coercive in any way. There is no statement as to what should happen. Just the opposite. I am saying that my reason for being there is that the person in charge of the body wants me to be there; it's not *my* desire that I be there. I am obeying the person in charge of the body (the patient's conscious choice). The most important thing is that I keep it very short. It is not a dialogue. No response is expected. I am stating who I am, why I am there, and for how long. After that, I mind my own business.

Many Tui Na practitioners find that everyone feels more comfortable if this type of verbal communication, whether silent or out loud, is performed in the very beginning of the session. I often do this at the beginning of every session. Sometimes I vary it a bit, adding remarks such as, "I worked with you last week. I'm going to do it again this week," but I try not to get too fancy, and I never suggest that I am wanting a response. After I have laid my cards on the table, so to speak, I mentally detach myself from any result, and go about my own business.

CHAPTER SUMMARY

Resting FSR using the same amount of support as the assessment type of FSR. The difference is that resting FSR goes on for an extended period of time.

While sitting for an extended period of time doing nothing in particular, the temptation to start looking for signs of progress can be compelling. However, the best results will be obtained if the practitioner minds his own business. When the injured area does decide to move, it will be

At this point in his recovery, he had resumed playing tennis, golf, and going for long walks. However, his rejection of that right arm was so extreme that when I asked him to use his right arm in any fashion, such as carrying a key in that hand or using that arm to carry a sack, he was barely able to walk, could not figure out how to negotiate stairs, nor could he figure out how to get into his car. It was as if the motor function of his brain slammed shut if he was forced to use the arm that had now become hateful to him.

apparent. Until then, the practitioner should remain fairly still for as long as seems appropriate. If it seems as if the foot no longer wants support, or wants it somewhere else, even if no relaxation has occurred, then the practitioner should move to the new position.

The best way to learn this work is by doing it. After a few dozen hours, the hands of the practitioner, if his thoughts are minding their own business, will become attuned to the needs and wants of the injury site. After that, it is just a question of letting the injury heal itself at its own pace.



"Tell me, tell me why?"

- The Four Aces - 1951 hit song

CHAPTER THIRTY-NINE

FSR: HOW DOES IT WORK? A HYPOTHESIS

This chapter will offer some thoughts on the how and why of Forceless, Spontaneous Release and all other forms of light-touch therapy. We do not actually know exactly why light touch therapy is so effective. The following explanation is only hypothetical. Even so, the explanation below seems to make sense, and it is supported by the results. By understanding the thinking behind it, some of the theory, a practitioner might be better able to master the techniques. Therefore, I am including this refreshingly short chapter.

The basic premise is this: when tissues are held in such a way that the holder can't really detect the holder, it may be that the tissues, unable to tell that the support is coming from outside the body, assume that the support in the area of holding is coming from inside. The muscles and such then reduce their tension-holding levels to accommodate for the extra support that is coming from the hands of the practitioner.

Conversations between brain and muscle

The muscles of a healthy person, even when relaxing, are never perfectly rigid nor perfectly limp. Muscles work in opposing pairs, and the two paired muscles are always performing a balancing act in order to create the effect of "being relaxed." They each are always tightening a little and then relaxing a little and then tightening again. The brain is always sending signals to the various muscles saying, "You seem a little tight; loosen up." This is followed by, "Now you're a little loose; tighten up." This type of back and forth goes on constantly between the brain and the body parts.

If a health practitioner is able to hold onto the skin of a limb or body part with the right amount of pressure and support, the limb or body part won't suspect that extra pressure is being added to the system. When the brain sends its usual inquiry, "How are you, too tight or too loose?" the body part answers back, "It's just fine, but maybe just a tad too tight." The brain tells the muscles in the area to loosen up. Then the brain sends another query, "How are you now?"

If the health practitioner is continuing to hold with that steady level of imperceptible support, the response from the muscles to the brain will be, "Just fine, but maybe just a little tight." The brain instructs the muscles to loosen further. Meanwhile, the practitioner continues to lay low. "How are you now?" says the brain. "We're just fine, but maybe we're a little tight." The brain instructs the muscles to loosen up just a skooch, and then it asks, "How are you now?"

Over a period of an hour, even a body part that has been fairly tight will usually have loosened up enough that the tension in the area has relaxed somewhat. If this tension was holding some joint, tendon, ligament or muscle in an incorrect position, that body part can start sliding back to a more comfortable place: the place where it's supposed to be.

When some body part returns to its correct place because the system was relaxed, that body part will not return to the incorrect place even when the tension resumes. Why? Because the tension will not resume. The tension is usually working to maintain the incorrect position into which the body part was forced during some injury or application of force. The tension is there to

prevent the body part from being shoved any farther out of alignment than it already is. If the body part moves back, closer to its original position, the tension in the area has no reason to resume.

During many types of forceful therapies, a body part is jammed back into the vicinity where it belongs, and then, over the course of a few days or a week, the underlying tensions – which have never let go – assure that the body part goes right back to the incorrect place that it occupied prior to the therapy.

Light touch therapies are becoming popular with practitioners and patients who have noticed that the relaxations achieved by light touch therapy last longer. Also, some of the tensions that cause pain or illness are very subtle and due more to fear than to actual physical impediment, such as impingement on a nerve. Shoving a bone or muscle may not relax a body part that was in the grip of terror. Support, support, support may allow the dread, panic, shock, or alarm to dissipate. After that, the movement of the associated tissues will occur naturally, without the use of force.

More theory: how some people tighten up in response to a blow

Injurious impacts don't necessarily cause an injury that won't heal. Most of the usual blows that we receive heal by themselves with nary a second thought. But in cases where the body doesn't snap right back, due to fear or a tension holding pattern, the injury may take more time to heal.

In the case of displaced tissues that do not go back into place following an injury, very often the problem is retention of the force of the incoming blow. The body stops the incoming blow through muscle tension and then continues to hold indefinitely.

Very often, a person who braces himself for an incoming injury tightens up more than he needs to and never lets go. The force of the incoming blow is stopped – and retained – by the defensive mechanisms in the body. This tightening is not necessarily a good thing, as the story below will demonstrate.

Different styles of response to injury

Some people respond to injuries with tension. Others never get tight. Here's an example of someone who didn't tighten up.

A friend of mine who used to drive an ambulance often regaled us with his stories of the poignant and the bizarre. He told us once, "The alcoholics never get hurt as badly as the sober ones. They just sort of flop around. It's not fair." He had the following example to back him up.

One memorable night, he got a call to rescue a man who had driven his car off a bridge near the levee. The car had broken through the bridge's guardrail and was upside down in the river. Fortunately, the river was only several feet deep at that time of year. The car was in the water, upside down, the wheels in the air.

The ambulance team expected to find a person strapped in his seat, upside down. If he was alive, he might have a broken neck or back; he would certainly have a whiplash injury. He might be unconscious; his head might be under water. As they scrambled down the riverbank in the dark, heading towards the car, they were prepared for the worst.

Instead, when they got down to the water's edge, they saw the driver of the car staggering around aimlessly in the water, chuckling to himself. When the driver saw the ambulance drivers, he peered at them questioningly through the darkness. Then, giving up hope of identifying them, he giggled sheepishly, "Oh wow. I must be really messed up!"

Upon investigation, they found that this carefree person's blood alcohol level was at high tide. He was apparently uninjured except for a few inevitable bruises. He certainly wasn't tensed up or holding on to anything. He incurred no lasting injuries.

Ski lessons

Skiing teachers usually tell their students to "go limp" when they lose control or are about to crash. A limp, relaxed body will allow the power of a forceful blow to pass through the body and out the other side. There may not even be any whiplash type of movement if the person is limp enough to allow for perfect follow-through of the forces of impact.

After the force of impact has passed through, even if injuries are sustained, the injuries can set to work healing themselves; there is no residual tension preventing relaxation and healing.

On the other hand, if the body tightened up during the dangerous event, the tension may depart slowly, or never. The physical displacements from the injury will probably not be able to set themselves aright until the tension is dispersed.

The innate healing force in the body can heal just about anything. But when fear rears its head, and our minds get in the way of healing, either through retained fear, which can manifest as adrenaline and/or through retained tensions, that's when our innate healing force can't do its job.

To brace or not to brace, that is the question

When a person does not brace himself for an incoming injury, he very often does not have the same level of injury as a person who stiffens up in anticipation.

This principle is demonstrated frequently by inebriated people: they often walk away somewhat unharmed by a blow or fall that might have killed a sober person. The corollary is this: a person who sees an incoming injury and braces for it may actually do himself more harm by stiffening up.

Where this principle ties in with our work is this: sometimes a person stiffens up in response to an anticipated or actual injury and then never lets go, or doesn't let go completely. Yin Tui Na allows the muscle to let go.

Also, the immediate pain of an injury can trigger a normal, protective, immobilizing response on the part of the muscles. If this immobility is not relaxed, the tension will stay in place indefinitely.

Westerners are trained to respond to pain by diverting the attention away from the pain. As children they are taught to distract themselves from the pain. A candy, a diverting toy, a verbal instruction "don't think about the pain," may be proffered as helpful amelioratives. According to eastern theory, faster cessation of injury pain can be attained by focusing on the pain.¹ Where the mind's attention is focused, there the life force and healing energy of the body is likewise focused.

¹ When I was in college and trying to distract myself from a painful sprain via aspirin, a roommate said to me, "In China, when they get hurt, they focus on the pain. If you confront your pain, it can't hurt you anymore. The injury goes away faster."

I assumed that my roommate was an ass, and took another aspirin.

Her words stuck with me, however, and over the next few years I experimented with focusing my attention on pain. After taking up the study of yoga, I added another component: when I was injured, I would immediately stop what I was doing and focus all my attention on the injury site. I would gently tense and relax the muscles in the

Energy is neither created nor destroyed

When an injurious force makes impact, the force of the incoming injury may not even be allowed to follow through the body. The area may tighten up, absorbing the force of the impact, and by holding tightly, prevent the force from dissipating throughout the body. If this happens, the energy behind the force, being neither created nor destroyed, remains in the body, held in place by muscle tension. To hold the impact in place, the force of the body's tension has to equal the force of the impact; this can be a lot of force.

Very often, all of the tissues are involved: the skin tightens up; the muscle tightens up; the fascial linings get twisted; one bone may be displaced or broken and the muscles that hold the bones in place may get torqued.¹ The blood vessels themselves may become twisted and then hold onto that twist.

area of injury. Usually, my entire mind was screaming at me to do the opposite. In particular, my mind did not want to involve the muscles closest to the injury site. However, I found that if I forced myself to gently tense and relax the very tissues that were most afraid of being tensed, the pain level would suddenly drop. If I continued for a few minutes more, all the while imagining sending light and fearlessness into the wounded area, the injury would sometimes heal instantly.

Twenty years later, I had an opportunity to put this theory to a strong test. After a very steep, four mile descent into Yosemite valley on a trail that might as well have been made of polished glass, and which was liberally scattered with tiny round stones that acted as ball bearings, I finally reached the valley floor. My knees had been in a state of terrific tension during the descent, as slippery slope, combined with the deadly drop-off that lined one side of the trail all the way down, forced me to hold each footfall in place with supreme tension until the next foot had found certain footing.

As I was striding to the end of the trail, my parked car a mere fifty yards ahead of me, my right knee suddenly buckled, the knee cap jerked over to the side of the leg, and a burning sensation shot through my knee. I let out a scream as the pain dropped me to the ground. I had torn my left knee ligament years before and knew the feeling. This time I was certain I had done it again, on the right.

Probably the extreme tension that I had held in my knees while on the trail had finally let go, and the resultant extreme relaxation allowed my knee cap, tibia, and femur to each go their own independent way. The result was a major blow-out of the knee. I could not take a step. I was shaking and gasping for air.

The car was in sight. My husband and son asked if I could make it the rest of the way, hobbling, if they supported me.

I thought about it for a moment, and then told them that I was going to be fine but that I needed to stay right where I was for a moment. Then I sat down in the scrabbly dirt and held my knee in both hands. I thought about oh-so-gently tensing the knee, counted to ten, and then thought about relaxing it. My mind was telling me to do anything but this, but I kept at it. After a few exercises in thinking about tensing the knee, I found I was able to actually get a tiny bit of tension response in the knee, a tension that corresponded to my thoughts of tension. Then I began in seriousness to gently tense the knee, hold the tension to a count of ten, and then relax. I was utterly focused on what I was doing. My entire mind was paying attention to the knee. It soon stopped hurting. I kept going. It was as if I was mesmerized. I lost awareness of my husband and son who, when I'd last looked, were asking if I was OK. I kept focusing on the knee: gently tensing it, holding the tension, and then relaxing. At some point, I could sense that light was flooding into my knee. When my knee was gently tensed, the light grew stronger. As I continued, it seemed as if the sun itself was radiating from within my knee. There was no pain whatsoever. I have no idea how long I sat there, enjoying the rare sensation of having a bright light filling my right knee. And at some point, I opened my eyes and announced to my worried ones that I was perfectly OK. I stood up, warily, and tested the knee. It was very slightly swollen. The kneecap had moved back into place, the leg bones were lined up. I could easily bear weight on the knee but the joint felt a little warm and tender inside. I walked slowly, carefully, back to the car under my own steam.

My roommate had been right. As to her claim that the entire Chinese population treats injury in this manner, I cannot know. But I do know now that this manner of working with injury is far more effective, in both the short term and the long run, than the method of distraction and denial.

¹ A reminder for those who have joined us lately: "fascial" means "related to the fascia." The fascia is the extremely thin, transparent tissue that surrounds all the various membranes and organs of the body.

Healing

Techniques that supplant the tension in the body with externally supplied tension can allow the twistings and torsions to relax and unwind. As long as the body thinks that the correct amount of support is being supplied to the injured area, it will relax to the greatest extent possible within that context of support.

The seemingly miraculous bone settings and pain relieving changes that occur in response to externally supplied support are not really miracles. FSR provides enough support so that the body can relax, let the tissues drift back into their correctly tensed position, and then heal themselves.

The healing of FSR is about as miraculous as the healing that happens when a parent holds a child. A mildly injured child, when held snugly, will very soon relax enough so that the force from his injury is able to dissipate. When the force is gone and his body tissues have drifted back into the right position, the child may want to linger for just a moment longer. Though the child may not realize it, he is waiting for the channel Qi to start running correctly. As soon as the force of the injury has dispersed itself throughout his various tissues or even into the mother, as soon as the tissues have settled back down into their comfortable and correct position, and when, finally, the channel Qi is sending a signal to the brain that says healing may commence, the child no longer wants to be held quite so tightly. At this point, he may even get up and resume his play, as if he was never hurt.¹

If the injury is worse, he may need to be held longer. Even if the holding goes on for a longer time, the principles are the same. When the child feels snug enough, when he is being held tightly enough, he is able to relax.

Holding the baby

Holding gently but snugly, until the injured area is fully relaxed, is the essence of Yin Tui Na. If this technique is miraculous, then so is the miracle of swaddling a baby. When a baby is screaming frantically despite being dry and well-fed, sometimes the only thing that can calm him is to be wrapped as firmly as possible in a tightly tucked swaddling blanket. As the baby's limbs become imprisoned and he feels the steady pressure of the blanket against his entire body, a deep relaxation comes over him. Swaddling is impersonal. A swaddled baby is content, not because "loving vibrations" are being thrust upon him, but because he feels safe at a deep, cellular level.

Ideally, Yin Tui Na is highly impersonal. The practitioner should of course have the best of intentions for his patient, but he should not be thrusting his intentions on his patient. When a baby is swaddled, he is transported back to a realm where he feels supremely safe. It is almost as if the pressure of swaddling allows the infant to cease, temporarily, to perceive himself as a separate being. Instead, he is allowed to feel once again the peace and pressure that he felt in the womb, when he was still a seamless part of the infinite mystery, before he was thrust, at birth, into the illusion of separateness and mortality. If a baby in the pressure of the womb is being held in the arms of love, it is not the love of the mother that is most dominant in that pressure,

¹ For those who are reading the chapters on Tui Na and who skipped over the chapters on theory, "channel Qi" is the sum of the electricity-like currents that flow unceasingly in a well-known and well-studied circulating pattern close to the skin. Any of the cells or cell groupings (organs) in a living person's electrically-unified body can be accessed via these currents that run just under the skin. These particular currents are referred to as "channels," "meridians," or "pathways" depending on the preferences of the Chinese-to-English translator.

but the love of the cosmos. The perfect sense of unity and cosmic love is both personal and impersonal, with no sense of obligation.¹

Mere physical injury

Energy is never created or destroyed, and for every action there is an equal and opposite reaction. Most physical injuries are the result of incoming energy which assaults the body. There is some immediate movement of the body during follow-through to the blow. The body disperses the force of the blow over as wide an area as possible. Swelling occurs which allows room for microscopic separation of displaced tissues. These injured areas then have in the swollen area a bit of extra plasma to jostle around in as they settle back down into place during recovery. Most healing from injury requires no outside intervention.

But in rare circumstances, the injury is great enough that the body cannot restore the area to its correct position without help. This is the case with a compound fracture of a bone, for example. In this case, the incoming energy exerted a blow on the body which was not sufficiently absorbed by the surrounding areas to prevent injury. The bone is broken and displaced. There needs to be an equal and opposite force applied to put the bones back where they were.

This equal and opposite force is usually supplied by the attending physician. In most cases, the laws of physics are maintained in the healing process by adding the physician to the mix; the physician applies directionally opposite forces to restore the bone parts to their original position. If there is not an excessive amount of underlying tension being retained in the tissues of

¹ Since we've compared Yin Tui Na to the swaddling of a baby, I might as well go a step further and compare Yin Tui Na to the way that we touch animals. Dr. Temple Grandin, possibly the world's most high-functioning autistic and a woman deeply empathetic to animals, is the premier designer of humane slaughterhouses. Her engineering designs incorporate features to make the animals feel as safe as possible as they go to a peaceful death.

In her writing, she makes the point that animals feel skittish when they are touched too lightly. A light touch is interpreted as the landing of an insect. A firm touch, with steady, even pressure, is relaxing to both an animal and, as she points out, to an autistic person – a person who is cut off from feelings.

In Oliver Sack's book, *An Anthropologist From Mars*, Dr. Grandin describes the machine she built for herself. She can lie down in her "hug machine" and press a button to bring the nicely padded walls of her machine snug up against her body. After a few minutes in her hug machine, she feels her body relaxing; peace steals over her body. As an autistic person, she is baffled by human emotions and does not desire physical human contact. She finds that, just as with animals, her body relaxes and feels safe in response to firm, steady pressure.

This brings me to another aside. In high school, I noticed that one of my friends had a wonderful way of putting a hand on someone's shoulder or holding the hand of a person who was feeling out of sorts. I asked him how he had developed such a kind way of touching. He explained that his family had a small, family-run dairy. If a dairy cow is unhappy or doesn't feel safe, she can't let her milk down. He said to me, in full seriousness, with no joking whatsoever and with the highest level of respect, "I always try to treat a person as if he were a cow."

Although a person with Parkinson's may think that he is able to consciously address his foot injury, you will see, in the next chapter, that, more than likely, his consciousness cannot even begin to approach the injured area. Even though a PDer may have learned, as an adult, to be aware of his emotions and his body, the injury received while still a child, the injury received in fear, may not be accessible to the conscious mind. That injured area may behave more like a frightened animal or a sentiment-absent autistic child. The most appropriate treatment for the injured area may be firm, gentle holding, such as one would give to a dairy cow who has just heard a scary noise, or to a newborn baby who can be comforted only by snug, firm swaddling that compresses his entire body into a tight package.

the injured person, a simple repositioning of the broken bones is all that is necessary for healing to commence.¹

Parkinson's disease: physical injury with emotional stonewalling

In Parkinson's disease, there is a more profound type of injury. In nearly all cases, the injury to the foot was never even mentally acknowledged. A force was applied to the body (injury), it was absorbed by the surrounding muscle, holding the injury in place so that no further damage could occur, and then the force was retained. No equal and opposite reaction ever occurred. The injury just sat there, an energetic time bomb. The injury very often never swelled, or never swelled to the normal extent. The injury may very well have never given a pain signal. The force of the PD-causing injury still is sitting right where it was at the point of impact. The injury just sits there, with all the energy of the incoming blow being retained by mental and muscle tension.

Until that energy is released, responding with equal and opposite force, the force of the injury will just sit there in the foot. The foot remains injured though the conscious brain and the rest of the body may have long forgotten the event. But the body has been using energy, for decades, to prevent the injury from going any further. This is the injury that must be cajoled out of its hiding place to begin the healing work in Parkinson's disease.

Foreshadowing the neck injury

Most PDers have a neck injury as well as a foot injury. We have begun to suspect that the neck injury is set in motion by the foot injury. While working on some patients' feet, we have sensed, in a few cases, a violent release of energy in the neck a split second after the foot injury relaxes. Other PDers have had their foot injuries successfully treated but still have tension in the neck that needs to be addressed separately.

A chiropractic researcher in Colorado has noticed that PDers usually have a displacement in the cervical vertebrae. When she restores these neck bones to the correct position, the PDer feels increased energy and improved mood for up to a week. However, the neck bones invariably creep back to the incorrect position. We suspect that the reason the neck bones will not hold their adjustment is that they are intrinsically connected to the foot injury. Until the foot injury is relieved, the neck must maintain its accompanying displacement.

The subject of compound injuries and neck injuries will be discussed in a later chapter. However, the subject also applies to this chapter: **retained tensions can prevent healing, even if displaced bones are shoved back into their correct location.**

A hammer and chisel treatment

A PD patient of ours who wanted to accelerate his recovery went to an osteopath to have his foot bones realigned. The osteopath was puzzled by the extreme tightness of the navicular

¹ This is why those people who are drunk or otherwise uninhibited in expressing response to injury are able to heal easily. However, as all of us in the medical realm know, sometimes a doctor sets a bone but the bone fails to knit. Very often, because the hurried doctor does not assuage the retained tensions, the bone parts are not able to stay in the correct place after being reset. When bones are not able to stay in their repaired position due to retained tensions from the injury being held in nearby tissues, modern doctors use screws and/or glue to hold the bone pieces together. Such joints usually heal very slowly and never function quite as well as before. By the way, such "screwed up" bones cannot respond fully to FSR treatment; the tension of the injury has been screwed permanently into place. If the screws are removed at some later date, these injured areas might then be responsive to FSR.

bone (the bone between the ankle and the cuneiforms) and his inability to make it budge. The doctor used increasing levels of force. Finally, he reached into his cupboard and pulled out what the patient described as “a hammer and chisel.” No doubt these were rubber implements, and yet their purpose was evident: brute force was about to be applied.

The patient was uncertain whether or not he wanted to have the hammer and chisel treatment. The doctor agreed to let him go for a walk and think about it. During the walk, the patient decided that he really did want to get his foot fixed as soon as possible, and so, despite uncertainty, he returned to the office and told the doctor to do his best.

The doctor gave several mighty whacks with his tools. No business resulted. Following that session, in addition to the jammed navicular bone, the patient had a new set of bone displacements. His Tui Na practitioner was eventually able to get rid of them.

Adrenaline

The body does not heal while it is locked into the sympathetic mode. The body waits until the emergency is over before it starts the healing process. People with PD are often locked into some number of sympathetic modes, including, sometimes, dissociation. Very often, their mental attitude dictates the use of adrenaline. Their never-ending, PD-causing foot injury seems to serve to enhance the release of adrenaline. With most PDers, even if they try to relax, they can never calm the anxious, relentless stream of negative thoughts. Though they may be able to relax superficially, they are always tense in their minds and in their injured body parts.

We use FSR to allow the tension to ease up around an injured area. When the patient’s injury-induced tensions/torsions are relaxed enough and the retained forces from the injury are dispersed, the injured parts of the body that had been tensed or displaced during the injury event are then able to fall back into their correct position. Once the tissues in the injury area resume their correct position, the nerve signals that have been sending panic/red alerts from the injury to the brain can cease. Pain subsides. Adrenaline drops. Rapid healing might then commence at the site of the injury.¹

Then again, *it may not*. If the PDer has created a mental stonewall around the point of injury, healing still may not begin. Although the emphasis in this chapter was on theoretical possibilities of how FSR helps address the forces that have been retained in the body, this chapter has not deeply addressed another key issue: how FSR helps address the fear that can occur during injury. I will simply say that having the foot held can sometimes ease the fear that has been holding the injury in place.

Then again, those people with Parkinson’s who have learned to intentionally dissociate from injury may not be able to respond emotionally to the supportive holding of FSR, even though the injury may respond and begin to heal. If the injury heals but the mind is still locked into fear, the patient may need to work on turning off his dissociation response. Instructions for that are given in another chapter.

FSR and non-tangible aspects of energetic blockages

The fear and shock that are often experienced during an injury can create electromagnetic waves, including thought waves, that can lodge somewhere in the body and/or mind. These waves can set in motion tangible shifts in the physical body and/or can build mental barricades against recalling or healing the injurious event(s). Yet the force, in these cases, is ideational

¹ If there is a mental attitude that also provokes adrenaline, then the injury may heal but the overall anxiety level and tremoring may remain. This will be discussed in an upcoming chapter.

rather than physical. This non-tangible force of shock or fear cannot necessarily be dislodged with a physical method such as chiropractic or acupuncture. The energetic disruptions set in motion by these non-physical wave patterns are part of the reasons that we insist on referring to “energetic blockages” throughout this work, rather than simply calling the problems “displaced or injured bones.”

The best way to dislodge the injury *and* get rid of the fear/shock waves is for the patient to fearlessly examine the long-ignored fear and/or shock, “look it in the eye,” confront it and evaluate whether or not it is still life threatening. If the fear/shock is no longer life-threatening, it is the job of the patient to let it go.

Using FSR when the foot injury is healed

Sometimes we use foot FSR on PDers whose foot injuries are healed but who still are working on turning off their dissociation response. A significant benefit of FSR, particularly when done on the feet – *even if the foot tissues have been completely restored to mobility and the foot can flex and extend in a healthy manner* – is that it seems to allow a patient a very unthreatening environment, one that is both symbolic and physical, in which to experiment with feeling, let alone feeling safe.

This section on FSR is finished. Much of the benefit of FSR will be purely mechanical: a relaxation in a previously tight body part may allow the tissues in the area to glide back to their correct position. Some of the benefit of FSR is more subtle: providing a neutral yet safe environment for the patient to mentally and/or emotionally review some thought wave patterns that may be contributing to the inability to heal.

While performing FSR correctly is the responsibility of the health practitioner, deciding whether or not to let go of the retained vibrations of fear and/or shock is, ultimately, the responsibility of the patient.

For those people who, on top of everything else, are intentionally dissociating from sensory awareness, they must take responsibility for their dissociation; they must relearn how to feel.



“In this drama of life, your love must be greater than your pain.”

Paramahansa Yogananda

CHAPTER FORTY

THE CORRECT WAY TO DISSOCIATE AND RE-ASSOCIATE

Nearly everyone has the ability to selectively dissociate. *Temporary* selective dissociation is a hallmark behavior for a mature person: a person who is able to temporarily compartmentalize certain thoughts or feelings so that he can process them at the most appropriate time.

An example: shaking at the knees

When my husband orally presented his Master’s degree thesis, he was perfectly calm and collected. Several hours later, when he was safe at home, his knees started shaking and his heart pounded a bit. When he felt these sensations, he realized that they were a delayed reaction. He sat for several moments feeling the nervousness that he had subconsciously felt but had not manifested during his oral presentation. These fear symptoms had been put on hold. He had temporarily selectively dissociated from them.

At home, when he felt safe, he re-associated with his delayed feelings and symptoms of fear. His knees were so shaky he had to sit down. Then, he processed the fear. That is to say, with full awareness his fear, he nevertheless took a deep breath, gave a bit of a shudder, and felt relaxation spreading through his body – thus neutralizing the fear. More colloquially, he “collected himself,” he “got a grip on himself,” he “realized he was OK,” he “shook it off.” We use these various expressions to refer to the act of processing trauma; they all refer to a very specific physiological process that humans and many mammals use following an episode of pain, fear, hypothermia, or any event that temporarily destabilizes the autonomic nervous system.

Some PDers have created a mindset in which they can never feel completely safe. Therefore they cannot re-associate. They cannot experience their temporarily delayed pain – pain that has been put on hold until a “safe time.” But there never is a safe time. Therefore, their physical and emotional pain must remain in the subconscious, in a state of dissociation.

Alternately, some PDers have forgotten how to relax: to access at will the feeling that accompanies expansion in the chest and which can be spread throughout the body, thus neutralizing any destabilizing event. Therefore, they cannot process, they cannot neutralize their pain.

Some PDers have both of these problems. They can no longer access (re-associate with) or process their experiences. But they can be taught. This chapter will explain, in minute detail, the physiological processes involved in re-association and processing.

New terminology: stabilization and destabilization of the autonomic nervous system

As you will recall from the chapter on heart-brain entrainment, a person is most electromagnetically stable when the heart and brain are entrained – working in sync. During

times of stress or trauma, heart rate variability patterns are chaotic, and brain waves are not in sync with heart waves.

We might say that, when brain waves are *not* entrained with heart waves, a person's core regulatory system, the autonomic nervous system, is, from an electromagnetic point of view, somewhat destabilized.

Restabilizing the autonomic nervous system after fear or stress

When the emergency, stress, or trauma is over, the heart waves settle into a steadier pattern; brain waves once again become entrained with the heart waves. When the heart waves and brain waves are once again working in sync, a person is *almost* in parasympathetic mode again. His heart and mind can begin to be calm from a *vibrational* standpoint, but the more crude connection between the brain and the heart, the *nerve* connection, may still be predominantly oriented towards the heart's spinal nerve – the sympathetic system nerve pathway.

At this point, a calm, deep breath and a simple shudder running through the body from head to chest, or even down to the toes and out the arms, can help complete the physiological part of the process of switching the heart's signals over to the vagus nerve and away from the spinal nerve. When this nerve switchover is finished, the person is firmly established in parasympathetic mode again. The entire process may require no more time than it takes to take a deep inhale and exhale.

When the heart and brain wave patterns are in sync *and* the heart primarily is using the vagus nerve, a person is in parasympathetic mode: heart rate and breathing are relaxed, good digestion can occur, and a person has maximum ability to use his heart to resonate with, to feel, his internal sensory experiences.

Most adults are able to temporarily dissociate from a stress or fear and then re-associate with it and process it later on, when it is safer or more appropriate to do so. This temporary, selective dissociation from a pain or fear symptoms is a normal function of the sympathetic mode. For example, this ability allows a person with a broken leg to run away from a tiger and process the pain of the broken leg later, after getting to a safe place.

A more detailed continuation of the example

Before my husband could *process* his Master's thesis fear and shakiness, he had to *feel* them. In order to do this, he had to re-associate with them. In order to re-associate, he had to feel safe.

When he got home and was feeling safe, his heart rate variability patterns stabilized. That is to say, his heart patterns ceased to be chaotic. His heart and brain began to work together. When heart and brain wave entrainment occurred and he had the leisure to think back over his day, his brain automatically brought forth the events from which he'd temporarily dissociated.

His brain waves, entrained with his "safe heart" waves, were in parasympathetic mode. His brain's neural connection to the heart was still in sympathetic mode: he was still suppressing his fear symptoms. As he calmly, contentedly thought about his thesis presentation, he re-associated: his temporarily dissociated fear and his fear symptoms came to the fore. He started shaking. He was no longer dissociated from that particular fear.

In this stage, his heart *wave* patterns were calm and steady. He was amused by the whole thing. But because his heart *nerves* were still in sympathetic mode, his overall body was half-lodged in sympathetic mode – shaking with fear.

For a brief moment, he was a mixed bag. His wave patterns were calm but his nerve patterns were shaky.

At this point, still mildly amused by his delayed fear response, he could process his fear. He took a deep breath and shuddered ever so slightly. These actions signaled his body to make the heart nerve switch over to the vagus nerve. When his nervous system shifted into parasympathetic mode, it aligned with his wave patterns that had already made the jump to parasympathetic. His whole body was now in parasympathetic mode. In this mode, he *could* relax. So he did relax.

When I say relax, I mean he allowed himself to *feel* a wave of expansion: first in his chest; and then throughout his body. He simultaneously *felt* the shaking and fear. The expansive feeling neutralized any negativity (fear) in the shaking. With the fear neutralized, the shaking stopped. He had processed the fear.

His thesis experience, delayed fear and all, was now a united memory in his mind. No significant part of the thesis experience was compartmentalized out from normal consciousness.

More about processing

This is so important that I'm going to repeat some of the above, with more detail. When I say that my husband "processed" the fear and the fear symptoms (shaking knees, pounding heart) of presenting his thesis, what exactly did he do? He did the normal, healthy series of biological events that an emotionally mature person automatically uses to return the autonomic nervous system to parasympathetic mode once it becomes safe to do so:

He *felt* his fear symptoms. Even as he noticed them, he was slightly amused at this manifestation of a delayed response. He *felt safe* even though he was shaking. His shaking was post-traumatic shaking. He was safe, and shaking from an experience that was finished and done with. Because he felt safe, his heart pattern remained calm. His brain stayed entrained with his heart even though he was shaking.

Almost instantaneously, he took a slightly deeper than usual breath. He let a mild shudder pass through his body from the head down to the bottom of his feet. This subtle shudder helps reconnect the heart and brain's *physical* connection (the nerve connection, as opposed to the *wave* entrainment). This reconnect reset his autonomic nervous system to parasympathetic mode.

Then he exhaled and felt the expansion sensation (the feeling that accompanies inhalation and which is the source of one's ability to feel) spread out from its center in the chest to throughout his entire body. This phenomenon is more commonly known as "relaxation." Internal sensory awareness returned to his mind and body. He had re-associated the events of the day, and having processed them, was now able to relax deeply, feeling his internal vibratory sensations resonating with the sights, sounds, and smells around him and also resonating with himself.

While he was shaking and manifesting fear, he was in partial sympathetic mode: he was only able to feel his external physical symptoms such as shaking of the knees. He was *not* able to feel his internal sensations. By shuddering and relaxing, he was able to turn off the sympathetic mode.

By performing these two processes, his nervous system re-established the connection between the brain and the head (the shudder) *and* re-established an internal sense of positive feeling, thus ending the shaking.

Re-establishing the internal sense of feeling is an important part of processing fears or fear-initiated symptoms or pains. A person cannot fully feel his internal vibratory sensations if he is in sympathetic mode. In order to restore the internal feeling of well-being, a person (or animal) must invoke sensory feeling in his chest and command the feeling to spread throughout the body. This sounds very glorious and dramatic; actually, healthy people do this regularly, they do it automatically, they do it without even thinking about it. Most PDers who get stuck in partial recovery do not do this. They also do not know how to feel safe.

Because PDers are going to have to relearn this innate process by mastering the mechanics of it, the next section is going to dissect and describe in minute detail the events involved in re-associating.

RE-ASSOCIATION AND RELAXATION

Healing from a dissociated pain, shock, or fear can be divided into three stages: 1) recall: recalling the feeling of calmness in the heart and then recalling the traumatic event, and recalling the pain from which one has temporarily dissociated. rerealization of safety 2) the deep breath and shudder 3) sending feeling capability from the heart outwards to the skin. Let's look at these three stages one at a time.

Recall

Recalling the feeling of calm in the heart: the realization that one is safe

Feeling safe is the first step. Feeling safe calms the heart. If a person feels safe, any temporarily repressed or unhealed pains can come to the surface. If a person does *not* feel safe, he cannot perform the shudder and relaxation. Even if a person is not *certain* that he is safe, he might not be able to convincingly perform the shudder and relaxation.

Semantics: the difference between feeling safe and feeling relaxed

Feeling safe is a condition in which the heart wave variability patterns are stable. Relaxation is a body-wide experience of expanded internal sensory awareness of a vibrant feeling of well-being. A person cannot relax if he does not first feel safe.

When a person feels safe, he can express his troubles and manifest his pain, chills, or fear. Then, to process these manifestations, he must relax, particularly in the area of the manifestation. He must simultaneously feel the problem *and* the vibratory relaxation that surrounds and permeates it.

Feeling safe is a heart feeling. The feeling of safety can be disrupted or prevented by negative or fear-based thoughts. The feeling of safety can also be re-installed by remembering a happy or peaceful event and instituting in one's heart the heart feeling that occurred during that happy or peaceful event.¹

¹ In our limited experience, most partially-recovered PDers cannot perform basic, general public-oriented, feel-safe heart stabilization techniques. Actually, they usually can't even understand the instructions, let alone do the techniques. To calm their hearts, PDers usually require very literal and sequential, PDer-oriented instruction. As you can imagine, a person who cannot even imagine that he has a heart will have a difficult time performing techniques that are designed to modify his heart's wave patterns. A person who has chosen to dissociate from his ability to feel may not be able to reinstitute a past feeling of heart stability. This is why dissociation from the ability to feel is so much more devastating, eventually, than mere temporary dissociation from an event. In order to re-associate, a person needs to feel safe: feel the heart and re-establish in the heart a previously felt pattern of calm. If a person has decided to not feel his heart, he will not be able to perform this basic process.

Feeling safe and dopamine release

Feeling safe is the trigger that causes dopamine release in the brain. Many people think that feeling good causes the release of dopamine. In fact, feeling safe causes the release of dopamine. The release of dopamine then allows a person to imagine himself doing various activities such as truthful speech or singing, locomotion, manual dexterity, hearing, seeing, and feeling good. When a person can imagine the sensory *feelings* of doing these things, he can then execute these imagined events.¹

Extremely important new research with mice

New brain research has discovered what happens when an animal feels safe: he has a surge of activity in the substantia part of the brain and is then able to move freely and relaxedly.

This research was conducted on mice. Laboratory mice can be trained to either feel fear or safety in response to specific sounds or tones: pavlovian responses.

While the mouse is neither afraid nor particularly safe, if placed in an enclosure, he will generally amble near the perimeter. When the researcher subjects the mouse to the fear-tone, the mouse severely limits his movements: he stays fairly still. When the mouse is subjected to a safe tone, the mouse experiences a surge of activity in his brain's substantia. If a mouse is let loose in an open field and is then subjected to the safe-tone, it will "stop acting defensively. The mouse walks into the center of an open field as if it owned the place, showing no signs of fear."²

When a mouse "feels safe," he moves freely and shows unrestrained curiosity. Brain scans of the mice show that, in "safe mode," the mouse has a surge of activity (abrupt release of dopamine) in the substantia area of the brain.

In the case of my husband and his Master's thesis, he had only dissociated from the fear of the event. He had not dissociated from the event – he could remember the event in great detail. He had not dissociated from his ability to feel, in general. He had not shut down his heart. He had not slid into automatic dissociation. He had simply dissociated, temporarily, from the fear portion of the event by sliding into sympathetic mode during his oral presentation. In healthy sympathetic mode, a person temporarily dissociates from any pain or fear so that he can deal with the emergency at hand. This is a normal part of the sympathetic process. What PDers do, dissociation from the *heart* in a manner that induces automatic (pre-death) dissociation, is a far more dire type of dissociation. What PDers do requires a conscious decision that one might be better off dead, or at least pretending to be dead to certain circumstances. Over decades the number of qualifying circumstances tends to increase.

I read somewhere that the Heartmath Institute, which successfully uses the "recall a peaceful event" technique while working with disparate groups ranging from fourth graders to CEO conferences, has approximately a 96% success rate with their heart calming techniques. In my experience, unmedicated PDers cannot do these exercises. I can't help but notice that 4% of the US population develops symptoms of idiopathic Parkinson's by age 70. I have to wonder if the idiopathic Parkinson's population might also be the 4% of the population that is unable to do the Heartmath Institute's very simple heart calming techniques. If so, these techniques could be used to predict if a person is on the road to developing Parkinson's disease. A person who is diagnosed with difficulty in understanding and performing basic heart stabilization techniques could be retrained in how to use his heart without waiting for the symptoms of Parkinson's to arise. He might never go on to develop Parkinson's. Or if he did, recovery would be a simple matter of addressing the unhealed foot injury.

¹ In sympathetic mode, a person performs necessary locomotion, ego-based (and not always truthful) speech, necessary manual processes, looking instead of seeing, and listening instead of hearing.

² *In Search of Memory: The Emergence of a New Science*, by Erik R. Kandel, Nobel prize winner; p. 350.) The text for the mouse research has an accompanying diagram that shows the trajectory of the "safe" mouse; the diagram shows that the mouse ambles all over the field, crisscrossing from one side to the other.

This finding is consistent with our work with partially-recovered PDer. They can move easily when they feel safe – they behave as if they are experiencing a surge of dopamine release. However, they almost never feel safe.

Reciprocity: inducing safe feeling by moving easily

We know that many body processes are reciprocal: cause and effect may be reversible. When a person feels elated, he may burst into spontaneous song or dance. In this case, the elation-based surge of activity in the substantia nigra results in uninhibited movement. However, the reciprocal is also true. A person who is in a fearful or negative mood may generate a surge of activity in his brain by allowing himself to spontaneously burst into song or uninhibited dance. The joyful movement can serve as a “reminder” of a time when the heart wave pattern was stable, and can thus stabilize the heart. The heart stability, in turn, causes a person to feel safe. The feeling of safety causes a surge of activity in the substantia, and results in an uplifted mood. In the first case, feeling safe (elated, in this case) resulted in a physical expression of joy. In the second case, a physical expression of joy resulted in feeling safe. Thus, reciprocity.

Via negative or positive bioelectrical and biochemical feedback loops, many body processes are reciprocal. What this means to the PDer is that, by refusing to feel safe, he is inhibiting activity in his substantia nigra. By indulging in behaviors that are wary or which are designed to protect him from what he imagines to be omnipresent risk, he is telling himself that he is not safe. He is actually inhibiting the very neurotransmitters that would allow him to feel good. By *logically* trying to make himself safe through practicing caution and inability to feel his heart, he inhibits the *feeling*, he inhibits the physical sensation, of genuine safety that would permit his heart to stabilize.

As long as his heart pattern is chaotic, he afraid to feel his heart. As long as he cannot feel his heart, his heart wave patterns will be chaotic. As a side effect, as long as his heart wave variability pattern is chaotic, his brain will not release the surge of dopamine that leads to full sensory function or imagination-based, normal movement.

Reciprocity of heart chaos and dissociation

If, due to extreme loss of blood or perforation of the flesh, a person slides into dissociation, his heart wave patterns become very small and chaotic and dis-entrained from the brain. His heart waves cease to resonate with sensory experiences. He does not feel safe: his brain cannot release dopamine.

If a person decides to pretend that his heart is very small and unable to feel (to resonate with sensory experiences) and that he is not safe, a biological reciprocity kicks in: he behaves as if he has received a extreme loss of blood; symptoms of automatic dissociation manifest; dopamine cannot be released.

In the first case, dopamine is inhibited because a person has received an injury that has altered his heart wave function. In the second case, inhibition of dopamine release in a person who is otherwise physically recovered from the immobility-producing injury is a *side effect* of his selective dissociation from his heart.

I'm rather beating this into the ground in order to make the case that, in order to fully recovery, a PDer must feel safe regardless of circumstances. Feeling safe maintains the healthy, normal heart mode for humans. PDers who get stuck in partial recovery do not feel safe. Those

PDers who have recovered quickly have felt safe and grateful regardless of their circumstances – even *before* they recovered.

Recalling the event and the pain

After a person has made his heart resume its calm, steady rate, usually by recalling how the heart *usually* feels – also known as feeling safe – he is ready to recall the events that he has put on hold. For a person who has recently been through a harrowing event, this recall will be somewhat automatic. For a person who is recalling something long buried, he may need to have a bit of a talk with himself to point himself in the right direction so that he can start digging up his unprocessed pains and fears.

Techniques for helping PDers learn how to most efficiently point their thoughts in the right direction and recall their dissociated pains and fear are included in the techniques chapters.

THE SHUDDER

The shudder that runs from head down to the bottom of the spine or down to the toes serves to physically re-establish the brain-heart link. The shudder is a physical event that serves to jerk the two out-of-sync systems back into kilter: a physical jostle to reorient the heart's nerve priority. The shudder can be small or large, barely discernable or quite obvious.

The shudder of relief is *not* the chill of fear, or the frisson of thrill that occurs in response to an intense reaction. The shiver or frisson of fear, cold, or shock is a cousin to the long-term, internal tremor of Parkinson's disease: a reaction that occurs when the autonomic nervous system is destabilized. The shudder is the antidote to the shiver.

The shudder of relief is used to “get a grip,” to shake off the shiver, chill, or frisson. The shudder is used to stop the physical, nerve-based heart-brain destabilization that we refer to as sympathetic mode. In sympathetic mode, heart signals are sent to the brain primarily via the spinal nerves. The shudder re-establishes the brain and heart connection and shifts the majority of the heart signals over to the vagus nerve.¹

An example

When a person sits through a scary movie, in which the sound track, lighting, and events are intentionally designed to destabilize the heart wave patterns, he may find himself shaking or gasping with fear. As he exits the theater after the movie, he may notice that he is still a bit shaky. He may say to himself, “Wow. That was a great movie. Now I think I'll go for a pizza.” He mentally reaffirms that it was only a movie, i.e. he is actually safe. He reasserts his separation from the movie via some simple action such as looking for his car keys or thinking about dinner. As he does this, he may also take a slightly deeper breath, shake off the thrill of the movie (shudder), and exhale while sending a surge of feeling to the perimeter of his body.

¹ Actually, most humans need to have some small amount of sympathetic system heart-brain communication via the spinal nerve at all times. The sympathetic system regulates breathing rate, heart rate and blood pressure. Unless a person is able to slip into a state of perfect calm and become breathless, he needs to have some small amount of activity in his heart's sympathetic system. Or you might say the reverse: only when a person is able to shut down all fears and feel truly, utterly safe, can he stop breathing for an extended period. Accomplished yogis and mystics of all faiths are able to attain this extraordinary state of fearless, pure feeling, in which they can “die daily,” as Paul refers it in his letter to the Corinthians (New Testament).

The whole thing is very, very fast. It is automatic. A healthy person does it anytime he has been slightly shifted into a heart-brain mode that is more dis-entrained than he is accustomed to – as soon as he realizes that he is actually safe and doesn't need to be stressed.

People who get stuck in partial recovery from Parkinson's disease very often have no idea what I am talking about when I describe this shudder and relaxation process.

One PDer, when I explained to him about shuddering, said, "I know what a shudder is!" And he demonstrated by shaking his head from side to side as if he was conveying the word "no." I told him that he needed to have a shake that went from his head down to his chest, or maybe even his tailbone. I demonstrated a quick little shudder that wiggled all the way out my arms and down to my knees. He looked at me in astonishment and replied, "I don't even know how even *think* about activating any muscles that would do *that*."

I assured him that, if he was sincere about wanting to recover from Parkinson's disease, he was going to learn how.

Another example

Chris's dog, Malik, was a particularly boisterous, effervescent dog. His joy and excitement incited joy in other dogs when he arrived at the local leash-free park. But one day at the park, a new, very big, very menacing dog confronted Malik. Malik stared back at him. They stood rigid, facing each other, hackles raised, both poised for a fight or flight response, until the other dog's owner saw the situation and removed his dog from the scene.

As soon as the other dog was gone, Malik started to scamper off. But his legs weren't moving quite right: they were somewhat stiff and mechanical. (One moves powerfully but mechanically when in sympathetic mode, as opposed to the flowing effortless movement of the parasympathetic mode.) Malik stopped walking and gave himself a good hard shake. His shake went from his head all the way down to his tail. Then he started to trot off again but his legs still weren't working quite right. Again he stopped walking and this time he shook himself hard, repeatedly, several times. Then, when the tension was shaken off – when he had physically jerked his heart back to its vagus nerve posture and away from its spinal nerve posture – he was able to bound back into the normal play at the park.

A yogic form of the shudder

Paramahansa Yogananda taught a yogic technique for shifting the heart nerves back to parasympathetic mode: Place a hand over the left side of the chest, draw the hand towards the sternum, and then let the hand rest momentarily on the sternum. The movement is repeated until the fear departs. A silent mental affirmation accompanies the gesture: "I am tuning out the fear from my heart's radio." The hand movement is significant: it helps physically reorient the heart towards the vagus nerve and away from the spinal nerve. The shudder that animals and healthy humans perform instinctively serves the same purpose: it physically jerks the heart out of spinal nerve mode and resets it into vagus nerve mode.

RELAXATION: THE RETURN OF FEELING

Relaxation, felt as an physical sensation of expansion in the chest of the vibratory feeling that accompanies inhalation that travels from the center of the chest all the way out to the periphery of the body (the skin), re-establishes awareness of healthy internal sensation.

The feeling capability – a sensation – that is sent from the chest to the periphery is related to the sensation that occurs in the chest in conjunction with the feeling of expansion that a person feels when experiencing something of great beauty, grace, or profound calm. It is a vibratory feeling. There is no word for this physical sensation in English. However, many languages do have a word for this feeling. Further descriptions of this feeling – a feeling that is scarcely known or remembered by most PDerers who have gotten stuck in partial recovery, will be addressed in the next chapter.

The main thing to bear in mind is that there is nothing tricky, occult, or spiritually advanced about this sensation: relaxation can be summoned up, any time, at will, by an emotionally mature adult.

Two feelings at once

When a person feels his pain, his fear, or in the case of many PDerers, his wounded heart, he must simultaneously feel relaxation, particularly in the part of the body that is feeling pain.

As he tries to feel both sensations – the pain and the relaxation feeling – he notices that it's easier and more interesting to focus on the relaxed feeling. The pain feeling becomes harder to notice. At some point, he primarily feels the relaxation in that area; the pain becomes a mere background sensation. At this point, he can forget about the pain. He will not be dissociating from the pain when he forgets about it: he will be forgetting about it because he has processed it and turned the healing work over to his brain.

Summary of the three step process

This three-step process – recalling the calm of the heart and recalling the pain, the shudder, the body-wide return to healthy feeling – is so quick, so automatic, that very few people ever consciously think about what they are doing when they perform this little stabilization trick. Nearly all adults are very familiar with the process even if they've never done it consciously. And yet, PDerers who get stuck in partial recovery often have no idea what I am talking about when I describe the process. The following vignette demonstrates:

The hypothermia example

I have sometimes used the following example of restabilizing the autonomic nervous system while talking to a PDerer and his/her spouse.

“After swimming in a cold mountain lake or the ocean, a person may start shivering hard as he makes his way back to his beach towel. At some point, he becomes very aware of the shivering. He takes a deep breath, and physically asserts that he is actually OK by giving a quick, body-wide shudder, exhaling and simultaneously letting his heart feeling reassert itself throughout his body.”

When I say the above to a partially recovered PDerer and spouse, the spouse smiles in recognition and says, “Oh yeah!” The spouse knows exactly what I'm talking about, even though he or she may never have initiated it *consciously*.

The “shaking off the cold shivers” shudder is usually done without even thinking, in the same way a dog shakes off after getting water up against his skin.

And then I look at the PDer to see if he can relate to the example. The PDer usually responds to my raised eyebrow by saying something along the lines of “I get really cold after swimming,” or even, “Huh?”

To be fair, some partially-recovered PDers do remember having shuddered in the past to stop themselves from shaking. However, it’s usually been a long time since they’ve done it, and they may not be able to recall the experience of an internal sensation of relaxation going out to the skin. If, from past experience, they do know what I am talking about, they still might not be able to perform the process on command. They can no longer invoke, at will, an expansion throughout the body of the feeling in the chest.¹

Techniques that may be helpful for teaching a PDer how to recall the feeling of safety in the heart, recall the event that led to dissociation from the heart, shudder, and feel, are included in the chapters on treatment techniques.

Looking ahead: word definitions in the next chapter

In response to my chilled-while-swimming example, the PDer may also ask what I mean by “heart feeling” or “the feeling in the chest that (etc., etc.).” Then again, the PDer may imagine that he does know what those phrases refer to, but it will nearly always turn out that he has no idea and has invented a completely wrong idea to make up for the fact that he doesn’t understand the word “internal feeling” or “heart feeling.”

PDers do not understand because they have not, in recent memory, perceived the feelings that these words refer to. This problem of non-comprehension is so pervasive and so crucial to recovery that the next chapter is devoted to trying to explain the correct meanings of phrases that many PDers do not understand. Even though the PDer reader may not know the meanings of many of the words I use in this chapter, I am going to plow ahead and move into the subject of emotional maturation: learning and practicing the method of calming the heart and processing negative experiences.

EMOTIONAL MATURATION: FROM THE CRADLE TO THE GRAVE

Infancy and early childhood

The cause of pain in an infant or young child: destabilization

When a very young child is hurt or frightened, the injury or fright causes some of his electrical currents to run amok in his body. The physical sensations set in motion by chaos in one’s internal electrical system is alarming. If the trigger is a physical injury, the injury signals

¹ Maybe our professional-musician PDers recovered so quickly from Parkinson’s because they have always been aware that they could “shake off” any unpleasant symptoms by mentally invoking or singing an appropriate song or snatch of melody. When stressed, they can mentally hear music that they know will create a feeling of joy. When they feel joy, their hearts calm down. When their hearts are calm, they feel safe. When they feel safe, they aren’t afraid of the unpleasant symptoms. They invoke a shudder and wave of feeling by continuing to invoke the music that generates a feeling of expansion in the chest. The entire process may only take a few notes of the music. All the real “work” is performed by brain, heart, and mind linkages; they execute their jobs almost instantaneously.

The music need not be fancy. I know one very musically sophisticated PDer who had always been able to pull himself out of any negative mood by bursting, full voice, into cornball Tin Pan Alley tunes from the 1920s such “Bye-Bye Blackbird” or any of Elvis’s lively rock and roll numbers. He recovered very quickly from early-stage PD, in a matter of mere weeks, as soon as his foot was fixed.

are sent to the brain via the nerves. When the injury signals and the chaotic electrical signals all get to the brain, the brain alerts the heart, and the heart wave variability pattern becomes chaotic. Brain waves are wrenched away from the stabilizing influence of the heart. Separated from the heart, the brain must handle the incoming irregular signals as if they are dangerous pain signals.

As noted in the previous chapter, the channel flow patterns are immediately altered when brain waves (thoughts) of pain or fear sweep over the body. The channel Qi alterations, together with the injury signals and the electromagnetic chaos induced by the injury are powerfully disturbing to the equilibrium of a young child's body.

Even if the trigger is only fear with no physical injury, for example, the fear induced by a loud, sudden noise or fear induced by the internal pain of colic, the same chaotic electrical processes can ensue.

In either case, fear or injury, the sum of the channel Qi shifts, the pain signals, and the electromagnetic chaos in the young child conveys an immediate sense that the body and the self are incorrectly connected, or even separating.

The very young child responds to this surge of disruptive destabilizing electromagnetic shifts by screaming. This scream of pain is very different from the whining cry of discomfort or the commanding cry of hunger.

The treatment for pain (destabilization) in an infant

When the young child screams out in pain, a loving adult is supposed to hold him, cradle him.

When a larger human holds the child, the child can still feel the pain and fear that is caused by chaotic electromagnetic forces surging through his own body, sending "error" or "danger! destabilization!" messages to his brain and heart and making his Qi run strangely.

But the calm electromagnetic field of the adult's heart waves is able to exert a stabilizing effect on the infant's heart. As the child's heart wave pattern becomes stable, his brain entrains with his heart. The child starts to feel safe. His brain releases a surge of dopamine. This takes care of the "feeling safe" phase of dealing with pain.

Next, the child shudders. Any adult who has comforted a very tense young child knows what I am talking about when I say the child inhales and gives a little shudder throughout his body a split second before he exhales and his body relaxes deeply.

The infant's fear or pain still needs to be processed. The presence of the adult performs the processing: the child's electromagnetic fields and channel Qi currents are influenced by the physically larger currents and fields of the loving adult.

The adult's electromagnetic channel Qi, moving correctly in the parasympathetic pattern, is physically larger than the child's temporarily altered electromagnetic field. The child's electromagnetic fields begin to resonate with the stable electrical patterns in the adult.

As the child's electromagnetic fields and currents begin to resonate with the much larger fields of the adult, the child's neural mode (sympathetic or parasympathetic) become resonant with the adult's neural mode. The adult, if he is mature and can place the needs of a child above his own needs, will have shifted himself automatically into full blown parasympathetic (maximum feeling and compassion) mode. The child, influenced by the adult's physically larger

parasympathetic mode, feels his own body slipping back into the calm electrical patterns characteristic of parasympathetic mode.

In this mode, a wave of feeling pours over and through him. The scary feeling that body and self were separating then ceases. The child feels that he is back in his body. After shuddering and shifting into parasympathetic mode, the child becomes peaceful and relaxed. He may snuggle up to the adult. He may look at his injury with curiosity. He may quickly lose interest in being held and may squirm to get free of the comfort-hold. The injury has not yet healed, but the fear and pain is gone, neutralized. The pain has been lovingly processed. The child is left with mere injury sensations such as gentle throbbing or heat in the injured area.

After the child's heart feels safe *and* the heart and brain re-entrain *and* body-wide feeling is resumed, injury sensations are not alarming – they are merely sensations. These sensations *must* be felt – they are sending important sending signals to the brain so that the brain can issue instructions for healing. When the body is in parasympathetic mode, able to feel pain sensations without feeling fear, healing is able to move forward.

Growing up

As a child grows up, he soon gets too big to be completely cradled in his mother's arms. When he is big and he receives an alarming, destabilizing injury, he may need to settle for a hug, since he no longer can be picked up and held. The larger child can no longer be “surrounded” by the adult's larger field. Instead, he must learn to *tune in* with the comforting feeling of being *adjacent* to the stable heart waves and the channel Qi patterns of parasympathetic mode in the other person's body and heart.

As he allows his heart to resonate with the nearby stable heart *and* recalls his memories of a calm heart, his heart rate stabilizes and he feels safe.¹ He shudders to shift the heart-brain neural connection into parasympathetic mode. Then, he resonates with the larger electromagnetic patterns of the parent's body *and* invokes remembered feelings of relaxation in the chest and the feeling of being inside his own body.

Notice that, as the child grows, he becomes able to contribute to his own stabilization process. Like training wheels on a bicycle, loving adults continue to lend stabilization to the child's heart and electrical system when needed. But over the years, the child finds increasing pleasure in learning to stabilize himself.

As the child recalls the feeling of a calm heart, shudders, and spreads Feeling throughout his body, his pain becomes neutralized and converts to the curious, even boring, sensations of mere injury. If the child feels compassion for the injured spot, he may suck on the injured finger or rub the spot where the injury sensations are located just as an injured animal will lick his injured place, thus helping improve Qi flow through the injured area. As he cares for the injury, the injury has ceased to be a source of fear: it has become bearable, healable.

As the child matures, he learns to draw increasingly on his own memory of what it feels like to have a calm heart. When the larger child gets a hug or a pat on the back, he can use proximity with a caring person's heart *in conjunction* with his own steadily growing ability to recall and invoke calmness in his own heart. When he needs to neutralize the pain and resume

¹ Notice my use of the word “allows.” As the child grows up, an element of free will is introduced. The child can actually choose to stay in a snit, if he so desires.

feeling throughout his body, he can use the proximity of a compassionate person together with his own invocation of parasympathetic feeling to shift himself back into parasympathetic mode.¹

As the growing child repeatedly does this in response to the thousand slings and arrows that the flesh and ego are heir to, he learns that he can always restore calm in his heart anytime that he becomes electromagnetically deranged.

Then, he can shake off the temporary heart-brain disconnect and feel relaxation, a return of internal sensory awareness, spreading through his body. He can then feel the *localized* electrical disarray of pain or injury while simultaneously feeling his much larger, body-wide stabilizing energetic patterns.

When the stable pattern is larger than and permeating the injury or pain pattern, the pain becomes neutralized: it becomes mere sensation.

Partially-recovered PDers who have mastered these techniques have discovered, much to their amazement, that physical or emotional injury *without* pain or panic generates a feeling that is actually rather boring. The alarming pains of even fairly severe injuries become merely

¹ In *Train Your Mind, Change Your Brain*, author Sharon Begley presents new research that proves that thoughts cause brain chemistry and brain electrical functions, and not the reverse. The “human as machine” model, so popular in the twentieth century, has been found to be incorrect. This archaic mechanical model hypothesized that all thoughts were the result of chemistry, and the chemistry was triggered by fairly inflexible nerve wiring patterns.

New research, often using as subjects monks who have spent at least 10,000 hours or more in meditation, shows the opposite: “[conscious] attention can alter the layout of the brain as powerfully as a sculptor’s knife can alter a slab of stone.” “Even when the monks were not meditating, their brains were different from the novices’ brains, marked by waves associated with perception, problem solving and consciousness... monks with the most hours of meditation showed the most dramatic brain changes.” “Monks had much greater activation in brain regions called the right insula and caudate, a network that underlies empathy and maternal love...there was a strong hint that mental training makes it easier for the brain to turn on circuits that underlie compassion and empathy.” By training themselves to consciously keep the heart calm and by keeping their attention on Feeling, monks can achieve a high level of contact with the part of the brain that registers maternal love. The process by which a child learns to comfort himself and process pain is similar: the child recalls the calm-heart feeling and relaxes, thus restoring himself to the physical posture that he learned in some loving adult’s arms.

Another example of the effectiveness of brain retraining is cognitive behavioral therapy. For the last twenty years, research on depression, an illness long thought to be the result of wrong chemistry, shows that the single most effective treatment for depression is thought retraining. People who are easily or deeply depressed tend to “calamitize” events or see things in black or white, with few shades of gray. Thought retraining to enrich these immature mental patterns often results in a lasting decrease in depression – the results are far better and longer lasting than any antidepressant medications. An abundance of books is available on the subject of cognitive behavioral therapy.

Bringing the subject back to Parkinson’s, many PDers have assured me they cannot recover because they had insufficient mothering. But sociological research has shown that a person can be emotionally balanced so long as there was one person, any person, in the child’s early years, who demonstrated compassion. The story of Remus and Romulus, among others, suggests that the “one caring person” might even be an animal! At the opposite side of the spectrum, research on orphans that are institutionalized at birth shows a high death rate from no apparent cause in infants who are not physically held once in a while. From monks to orphan hospitals, evidence abounds that we need to actively calm ourselves after a trauma. When we are young, we are assisted. When we grow up, we can learn to do it ourselves. The more often we consciously work on it, the better we get at it: our brain actually changes shape and activity level to reflect our practiced ability to calm ourselves.

Many PDers, on the other hand, have practiced a lifetime of wariness. Their brains have become oriented towards wariness, constant suppression of dissociated thoughts and the suppression of dopamine. These bad habits are not irreversible. New habits can be learned. There’s no time like the present!

sensations – throbbing, warm, or twisting sensations – as soon as the fear and electrical disarray components are gone.

A baby needs an adult to lean on in order to re-stabilize after a disruptive incident. As the child grows in maturity, he is supposed to learn how to bring *self*-calming abilities into the process. While it is always wonderful to get the additional stabilizing support of friends and loved ones, a mature adult also knows how to rectify a destabilized electromagnetic field – how to feel safe – on his own, if need be.¹

¹ The adult process of relaxation or self-rectification of a destabilized system can be thought of as a purely self-induced event, but it is known in some cultures as “turning to a higher power” or even “giving it to God,” or similar language. The internal physical sensation in the chest that spreads through the body during relaxation is known by many names, even though we have no specific word in English that is universally understood to mean this feeling. Some of the names for this feeling are Love, Something within, the Comforter, the Protector, Om, Spiritus sanctus, Divine Mother, Soul, and so on. These names tend to have a spiritual connotation, because the feeling is a vibratory sensation and is not associated with a specific physical structure in the body. It is not even necessarily associated with the body: one’s own vibratory feeling can be perceived even after it becomes separated from the body at death.

When a person experiences depersonalization, he is feeling his waves of consciousness – a subset of his vibratory self (the whole vibratory self includes Love, which is often not noticeable while depersonalized, therefore I specify “a subset”) – while simultaneously not feeling his body. This pre-death state can come to feel somewhat normal for the type of PDer that ends up getting stuck in partial recovery. I know that some western readers may not “believe” that such a vibratory self exists. However, it does, and it contains an enormous amount of energy. And even the most skeptical reader who is loath to accept the new science findings that proves that our bodies are created from vibrations, or that atoms are created from vibrations, might nevertheless admit that some people can feel the vibratory field of a loved one from thousands of miles away. Mothers in particular are notorious for being able to feel their children’s vibratory fields from a distance.

Speaking of the various names by which this feeling is known, the Chinese medical term for this feeling is Wei Qi. Wei Qi is often translated into English as Defensive Energy, but it is more accurately translated as Protective Energy. It is synonymous with the terms such as Comforter and Spiritus Sanctus listed above. Wei Qi is described in the ancient Chinese classics as being inside the body but located “not in the vessels of the body” (not a part of the chemical or structural part of the body.) The Wei Qi is the vibrational essence of the soul that remains unchanged even when the body’s electromagnetic field is disrupted. Hence it can serve as a comforter – an energy field that one uses to re-stabilize the heart and channels after the body has been physically destabilized. In this role, it serves the same function as the loving mother who cradles and re-stabilizes the screaming child.

Because Wei Qi is translated as “defensive energy,” English-speaking practitioners of Asian medicine often assume that Wei Qi refers to the cellular components of the immune system. It does not. However, the extent to which a person *feels* the protective Qi has a large effect on how well his immune system will work. To increase his Wei Qi, a person must increase his Kai Xin

(Kai Xin translation: Open (in the sense of opening like a blossoming flower) Heart, sometimes translated as happiness, but literally meaning Open Heart). In other words, the more a person opens his heart (allows himself to feel the vibratory feeling of which we are speaking), the more he can attune himself with the protective energy that surrounds and permeates him.

I recently read a proposal that the immune system components might be named *Nei Wei Qi*, or *internal* protective energy. The word internal suggests that these components are solid, tangible bits of physiology, “internal to the vessels,” as opposed to the vibratory energy of the Wei Qi that is “outside the vessels.”

Just as radio waves are always present and passing through seemingly solid objects but remaining true to themselves and “outside” of the objects through which they pass, the energetic wave patterns that define the universe and that define a given person from the moment of his soul’s inception are always present and feel-able – if one’s internal radio is consciously “tuned in” to the correct frequency. Names for these waves abound, but possibly the most common names in modern English are the Om or the Spiritus Sanctus. (A cagey reader will recognize that neither of these words is actually English.)

Getting back to the subject at hand, one might say that re-stabilization of the body’s electromagnetic fields, or “giving the pain to a higher power” if you wish, is merely re-attunement of the physically deranged patterns of

Adults can process their own pain

By the time a child becomes an adult, he is supposed to have learned how to be in control of his own consciousness. He is supposed to know how to invoke at will a feeling of calm and peace in his heart or chest. When, due to pain or fear, his core ability to feel becomes small or constricted or disappears (depersonalization), an adult is supposed to know how shake off his temporary foray into sympathetic mode and resume his body-wide self-perception as soon as his heart is calm and he feels safe.

Staying in parasympathetic mode

If an adult is truly mature and does not succumb to fear, which is to say, his heart stays always calm despite outward circumstances, he doesn't need to go through all three of the steps outlined in this chapter.

Because his heart has remained calm in spite of his physical or emotional injury, he never slides into sympathetic modes. He does not need to shudder and re-establish feeling after receiving an injury. Instead, a calm adult who becomes injured can comfort and treat the injury directly.

Caring for an injury in a mature manner

A mature adult who becomes physically or emotionally injured can focus for a moment on the pain sensation that is coming from the injured area: he feels the pain.

Note: many PDerS do not know that emotional injury can cause physical pain. It can.

The pain of emotional injury usually manifests as a lump in the throat, a tightening of the stomach, a stabbing behind the eyes, a twisting in the gut, or the pain of some other channel Qi congestion. A mature adult who feels the physical pain of an emotional injury focuses on that pain; he does not pretend that it is not there.

While feeling the pain of the injury, he simultaneously feels the vitality sensation for which there is no word in English, but he focuses on feeling that vibrant sensation in the area that has been injured instead of feeling it primarily in his chest.

The location of the Comforter

The pervasive vitality sensation is easiest to feel in the chest. However, the sensation actually pervades the entire body. A mature adult can attune himself with that sensation in any part of his body, at will.

When the mature adult simultaneously feels the pain of the injury *and* the sensation of vitality and comfort surrounding the pain and permeating the pain, the pain quickly neutralizes: it becomes bearable; it becomes mere sensation. The pain has been processed. We may say that the adult has paid attention to the pain.

In a way, the mature adult has played the same comforting role for his injured body part that, years earlier, his mother or some other adult played for his injured infant body when he was young.

channel Qi, brain waves, heart waves, and so forth, with the larger, *unchanging and unchangeable* wave pattern that defines the true Self of that person. This larger wave pattern *can* be felt. Our physical perception of these powerful, larger, body-creating waves of energy – the sixth sense - is the root source of our ability to perceive the five branch senses: sight, smell, taste, touch, and hearing.

Brother donkey

The adult treats his body with the calm and the compassion that it deserves. As Saint Francis used to say, the body is Brother Donkey. Treat it as well as you would any dumb beast.

When the great yogis say “dissociate from your pain,” they use the word in its sociological meaning: they mean stop *identifying* with pain. They mean that one should not imagine that his real self has been injured just because his physical body has a pain.

When the yogi’s body experiences a pain, the yogi quickly and lovingly cares for the injury just as he would care for any dumb beast or any human infant: he surrounds the awareness of pain with awareness of [the word that we don’t have in English: the feeling that accompanies expansion in the chest when perceiving something of great beauty or grace].

He feels his body’s pain, he feels compassion for the body that is experiencing the pain, and he feels himself cradling the pained place in the bosom of peace and joy that underlies every atom of every physical structure in his body.

As calm feeling pervades the injured feeling, it neutralizes the painful, “pay attention to me!” feeling. The pain of injury becomes the mere sensation of the injury, with no fear or drama attached to it. The injury can begin to heal.¹

Of course, a mature adult will also make use of friends and loved ones who step up to provide comfort and stabilization in times of hurt.

The point is not that an adult needs to be lonely and self-sufficient; the point is that an adult is supposed to know how to employ internal resources so that, in times of pain, he can face the pain, care for it correctly, and process it – so that it can heal.²

The commonplace nature of shuddering and relaxing

The swimmer who gets a chill and then shakes himself and relaxes or the movie-goer who shakes himself and relaxes is doing a basic, everyday resetting of his heart-brain connection. The shivering of hypothermia, the shivering of fear from the movie, the trembling of a person coming out of shock and the tremor of Parkinson’s are all related. They are all signals being sent from the brain to remind the destabilized person to take a deep breath, shudder, and reset the heart-brain relationship back to parasympathetic.³

¹ I am describing how a mature adult deals with pain. There are plenty of immature adults who do not process pain in this manner. Some so-called adults deal with pain by making a scene, lashing out in anger, demanding attention, wallowing in self-pity. These people are fascinated with every little insult or injury and think that everyone else should be, too. These over-grown children focus on their pain, tell everyone about their pain, and cultivate their pain. When I refer to “adults” learning to deal with pain correctly, I am referring to mature adults, not the ones that cultivate their problems to glorify or draw attention to themselves.

² I recall a beautiful instance of one adult helping another adult in a moment of great panic. Moments after the San Francisco earthquake of Oct. 17, 1989, magnitude 7.1, the epicenter of which was a mere ten miles from my house, a momentary lull occurred before the thousands of aftershocks started up. During this lull, my neighborhood quickly gathered together in the cul-de-sac, away from our swaying, and in one case, shattered, homes. One of the neighborhood moms, Kari, was shaking terribly. An older, always calm neighbor walked up to Kari and hugged her for several seconds. In the years that followed, Kari referred many times to the powerful, transforming, calming effect of that brief hug. “I was instantly OK. Before she hugged me, I didn’t know what was happening, I was terrified. After the hug, I knew that, whatever happened, we were all going to be OK.”

³ MDs, having no idea of the various processes involved, concluded, incorrectly, back in the nineteenth century, that shivering was a method for working the muscles vigorously, thus warming the body. In fact, the *more* a

If the body has been profoundly destabilized, the brain will initiate trembling behavior in the body. In an infant, this trembling is a social cue: the baby needs to be held. In an adult, the trembling or tremoring may serve as a gentle reminder to the destabilized person that he is not dead, and needs to get a grip on himself: he needs to take a deep breath, shudder, and process whatever he is feeling.

In my limited experience, most of our patients with Parkinson's who have become stuck in partial recovery have no visceral understanding of what I am talking about in the above section, or even this chapter.

But the PDer should know that millions of people *do* understand what I am talking about. They may use different terms, and many use spiritual terminology to explain the process, but they do understand what I mean about dealing with pain by drawing on a power *larger* than the pain: a power that is filled with calm.

Many of our patients who have become stuck in partial recovery also have no idea that it is possible to experience real pain – really *feel* it – and not be threatened by it. To many partially-recovered PDers, the words “pain” and “threat,” or even “*mortal* threat,” are synonymous. They have no idea what I am talking about when I say that one must surround the pain with love until the pain becomes mere *sensation*, after which, it can begin to heal.¹

For many PDers, a major goal of life has been the denial or avoidance of pain. As our research continued, we began to suspect that PDers have attained harm and pain avoidance by shutting down their ability to feel, instead of by learning how to make pain bearable, then neutral, and then useful – as a prod to healing.

It is perfectly reasonable for a mature adult to temporarily dissociate from pain or fear if he deems the time or place is not suitable for a display of shivers, screams, or tears. However, as soon as the adult finds himself in a safe place, he reviews the negative situation, feels the dissociated pain or fear, gives a little shudder to reconnect with himself, relaxes, and then, with compassion, neutralizes into mere sensation any pains or physical symptoms that have made themselves known.

Correct processing of pain is the exact opposite of living a life of harm avoidance, denying the existence of pain, or, worst of all, dissociating from the heart's ability to feel pain: becoming numb to oneself.

person shivers, the more anxious – and thus unable to warm himself – he may become. As he becomes more worried or confused from unstoppable shivers, he is likely to become *more* destabilized and even *less* able to regulate body temperature. The myth that shivering is a process designed for warming the body has never been put to the test and does not meet the fact that people shiver in response to various strong emotions, not just cold. However, like much of medicine, the myth that “shivers are the body's way of warming itself” will evidently endure until, against much protest and resentment, a more glaringly accurate version of physiology is grudgingly accepted. I have heard that there are three stages of scientific change: the new idea is first gently mocked or ignored, then it is vigorously reviled, and finally it is accepted as self-evident.

¹People in the southern United States have an expression, “sweet pain.” Many PDers, though very intelligent, have no idea what *feeling* this phrase is describing. “Sweet pain” refers to sensations of pain that are bearable because they are surrounded by or filled with love, harmony, or understanding. Another example of emotional pain that is not a mortal threat is contained in Shakespeare's phrase, “Parting is such sweet sorrow.”

THE ROOT CAUSE OF IDIOPATHIC PARKINSON'S DISEASE

Looking at the big picture of Parkinson's disease, which can slowly be brought on by a mere dissociation from an injured foot or which can burst rapidly onto the scene by a sweeping, body-wide dissociation from the ability to feel anything (psychogenic parkinsonism), or which can be any conceivable blend of the two, we see that the root cause of Parkinson's, the grain of sand in the oyster, is the inability to correctly deal with pain.

A history of injury and/or emotional pain is not the real trigger for Parkinson's. Everyone has injuries. Everyone has pain. But most people feel their pain. Many immature people even cultivate their pain, dwelling on it and showing it off.

But PDer's selectively dissociate from their ability to feel their feet injuries. Sometimes they selectively dissociate from their ability to feel *any* of their own pains. Then, either they never feel safe enough to re-associate with their "temporarily" dissociated pain or they *enjoy* the "impervious to pain" numbness that they have learned to induce. Thus they render themselves unable or unwilling to process their physical sensations of pains and injury. The pains and injuries lurk in the subconscious, triggering symptoms of automatic dissociation whenever the PDer senses that some mood or event is coming perilously close to touching on the dissociated, hidden portions of his heart.

A yoga student asked Sri Sri Daya Mata, a world-renowned yogini (female yogi) and a living expression of compassion, "Why do people say that God never gives you more than you can deal with? I've known people who have fallen apart from difficult circumstances; God *did* give them more than they could deal with."

Daya Ma replied gently, "No. They fell apart because they *didn't* deal with it."

Injury and pain do not cause Parkinson's disease. Failure to deal with one's own injury and pain causes Parkinson's disease. In this drama of life, your love must be greater than your pain; you must feel both so that the love can neutralize the pain.



I know you believe you understand what you think I said, but what you fail to realize is that what I said is exactly what I meant.

- slight twist of a commonly quoted phrase, original author unknown to me

CHAPTER FORTY-ONE

SEMANTICS

This subject has been touched on briefly in the main part of the text. Chapters forty-one through forty-three primarily discuss helpful meanings for words that are often misunderstood by PDer. These chapters are included in the Treatment Techniques section because the PDer may need to do some real work to learn the more helpful meanings of these words. Mastery of these words may prove extremely helpful in understanding the entire book. Several PDers have said that, after learning the literal meanings of many of the words in this chapter, they went back and reread the whole book: and got an entirely different understanding. As one PDer said, “Hey! When you say heart, you mean it the way they use it in literature!” I replied, “Yes, I mean it literally.”

Not until 2008 did we realize that many PDers have created nearly opposite meanings for recovery-crucial words such as feel, safe, and heart.

The wrong word meanings created by PDers had us going in semantic circles for years before we figured out what was going on. We were dealing with people who had dissociated from their ability to feel their *hearts*. The key to turning off the heart dissociation is the ability to feel *safe*. It turned out, most of our partially-recovered patients had no idea what we meant by either “heart” or “safe.”

For example, we’d say, “Try to feel your heart.” The PDer might say, “OK,” and start mentally affirming, “I should do good for others.” If we asked him to relax and enjoy a calm feeling of safety in his chest, he might mentally go through the checklist of all the things that he was supposed to do that day.

For the most part, we meant the words literally. PDers, unable to fathom how those words might be meant literally, had usually invented creative, but very often *opposite* meanings for our words.

PDers had also invented their own meanings for words like calm, vibrant, surrender, imagine, and neutralize, to name a few. They had also invented their own meanings for common phrases involving these and other words.

In order to recover from Parkinson’s, a PDer will need to know what these crucial words actually mean, even if he has emotional reasons for avoiding the correct, literal meanings. This chapter will only touch very briefly on the semantics problems, just enough to give the reader a hint at the nature of the beast. More specific information about semantic errors that stand in the way of recovery is included in the chapters on treatment techniques.

DEFINING HEART

The heart is an organ in the chest. When I use the word heart in a therapeutic instruction, I am not referring to thoughts of goodness, religious or spiritual thoughts, or

any sorts of thoughts. I am referring to the heart: its physical structure and its sensory abilities.

Defining “open your heart”

The general public, including young children, usually understands that “open your heart” means: allow yourself to feel the physical sensation that starts in the heart area of your chest and which expands when relaxing. It also means to allow yourself to feel the sensation that burgeons from the vicinity of your heart when you experience a mentally wordless perception of beauty or grace.

The physical sensation is sometimes referred to as calm, peace, joy, or bliss. These are *sensations*, not thoughts. You can feel these sensations more clearly if you shut down the unguided monologue that usually natters through the undisciplined mind.

The PDer who is stuck in partial recovery may have *no* idea what “open your heart” means, or that it relates to a physical sensation. Many PDers cannot feel this heart opening sensation due to the depersonalization brought on by selective dissociation. However, they *are* able to think. Therefore, they usually *decide*, incorrectly, but based on context, that the not uncommon phrases such as “open-hearted” and “open your heart” *must* mean “kindly” or “think kind thoughts; do what is logically perceived to be kind or spiritual.”

The actual meaning and the PDer meaning are almost opposites. The actual meaning refers to the physical sensation a person enjoys when his heart’s electrical circuitry re-orientes towards the vagus nerve. This re-orientation occurs when a person instructs his mind to recall and *recreate* the physical sensation of calmness that his heart has felt in the past, and to recall and recreate the *sensations* of expansion that accompany this heart-nerve shift. The correct meaning of “open heart” has to do with physical sensations of calmness and expansion in the chest, in the vicinity of the heart.

Again, the PDer definition has to do with *thinking* about behaviors or qualities such as kindness.

From a heart/brain point of view, these two meanings are opposites. Many PDers, due to heart dissociation, do not experience heart feelings. Being emotionally unable to experience the sensations of “open heart,” they assume that expressions such as “open hearted” and “open your heart” must be metaphors for “kindly” or “think kind thoughts.”

These dissociated PDers are wrong.

Defining “open your heart” in Chinese

Semantic twistings are not limited to *English*-speaking PDers who have dissociated from their hearts. The same exact error was made by a particularly rigid – both physically and emotionally – Taiwanese patient. While working on heart opening exercises, I asked him to recall the *literal* meaning of the Chinese phrase *Kai Xin*.

(*Kai* means “open” in the way that a flower enlarges as it opens up and *Xin* means the physical heart. This very common Chinese phrase is used to mean the happiness that comes from expanding the heart.)

He replied, “It means happy.”

“Yes,” I agreed, “but what does it mean literally?”

“It means happy.”

“What do the individual words mean?”

He had grudgingly come to my clinic at his wife's insistence. He now looked at me as if I were an idiot and said, "The characters means Open Heart, but the combination means happy."

I suggested, "Maybe it actually means Open Heart. Maybe happiness comes from opening the heart, letting the heart expand as if it is opening like a flower."

Now thoroughly convinced that I was an idiot, he replied, "That doesn't make any sense. A person can't open his heart."

I replied, as gently as I could, that maybe *he* couldn't, but that most people could, and that the opening and expanding sensation in the heart was the *basis* of happiness. He replied that "Kai Xin just means happy. It's just an expression."

As I tried in a dozen ways to describe the feeling of an open heart, he assured me that there was no such feeling. I asked him if he'd ever felt an expansion in the chest from feeling happy.

He thought for a long time. Then he remembered having felt good one time, many years ago, shortly after getting out of college. I asked him what the occasion had been. He replied that he felt good when he got hired to work in a bank. A bank job is a *highly* respected position in Taiwan.

I suggested that possibly what he felt when he got the job was relief, a decrease in fear, and not actually the sensation of an expanded heart and the sensation of calm that accompanies the feeling of safety. He said that he remembered how he felt when he got the bank job, and it fit the description of Open Heart, inasmuch as it had made him happy.

Yielding the point and agreeing that it may very well have been an open heart sensation, I asked if he could recall the sensation in his chest when he got the bank job and reproduce it right now.

He looked puzzled. Then he asked, "What sensation?"

He never was able to accept my statement that a person could feel a physical sense of expansion or "opening up" in the chest. So the problem is not limited to English speakers.¹

DEFINING "FEELING SAFE"

Feeling safe is a physical sensation: a sensation, not a train of thought.

When a person feels safe, he experiences a sensation of calmness in his chest. Those PDers who do not know what I mean what I talk about the sensation of expansion in the chest may also be unable to feel the calm sensation in and around the heart that is called "feeling safe."

¹ Merely having words for the sensation of joy that can expand in the chest is no guarantee that a person will not develop Parkinson's. The French, with their phrase *joie de vivre* (joy that comes from being alive), are not immune to *la Maladie Parkinson*. I have had several French patients. As a side note, it was a Frenchman, Charcot, who named the illness in honor of James Parkinson, the Englishman who first wrote up the symptoms. Back to the point, merely knowing the meaning of words and phrases such as *Spiritus Sanctus* and "open heart" do not necessarily enable a person to *feel* the sensations implied by the words. Just knowing the correct meanings of the key words does not, alas, prevent Parkinson's disease – but it's a help when trying to recover.

PDers who get stuck in partial recovery have often determined that “feeling safe” must refer to harm avoidance. By being guarded, they imagine that they have made themselves “feel safe.” They are wrong. In some cases, they have made themselves anxious or paranoid. They have *not* made themselves able to feel the heart sensation that we know as “safe.”

Being guarded is the *opposite* of feeling safe. If a person is careful and guarded, it means that he does *not* feel safe.

Some PDers tend to think that “feeling safe” is a condition that can be attained by self-monitoring their behaviors to reflect whatever they think is “good” or expected, or by pretending to be calm, or even deathlike. A PDer who thinks he can attain “feeling safe” by being ever ready to protect himself from danger or by being numb to pain is also very likely to be a PDer who is going to get stuck in partial recovery.

A healthy person understands that “safe” is how he feels when there *is* no danger. When there is no immediate danger, a person relaxes; his heart grows calm, his chest expands. Then, while feeling these sensations of *interior* calm, he can go about his vigorous or gentle, productive or contemplative, activities of the day.

When a person feels safe inside, no safety from externals need be attained through wariness. Safe is not dull or cautious; safe is sweet and leads to expansion of the heart.

Feel-safe mode is a physical condition that generates a physical sensation. Feeling safe is not a thought-based condition. However, negative thoughts can prevent a person from being able to experience a safe-feeling heart. When a person dissociates from his heart, or when a person shifts into a predominantly adrenaline-based, sympathetic mode, his attention shifts away from inner-chest sensation and towards thought. This shift towards thought is what prevents a person in these modes from being able to *feel* his heart. These modes also shift the heart’s electrical signals towards the heart’s spinal nerve, and away from the heart’s vagus nerve connection. When the heart is more oriented towards the spinal nerve, the heart does not *feel* safe. In fact, the heart hardly feels like anything at all while a person is focused on his thoughts instead of his physical sensations.

In chapter xxx, I discussed new research that showed “feeling safe” causes a surge of activity and a release of dopamine in the brain’s substantia. I mentioned the research to many PDers. I asked them for their personal take on the phrase “feeling safe” or how they go about acquiring the feeling.

Their replies were usually something along the lines of “I make sure all the doors are locked,” or “I don’t ever get caught with my guard down,” or “I try to be alert to any potential danger: I stay safe by being wary.”

I replied every time, “But those are activities that you do because you *don’t* feel safe. You take those steps to try to make yourself feel safe because, apparently, you aren’t automatically safe unless you protect yourself. Protect yourself from what? What are you afraid of? What do you do when you want to shake off that fear mindset and remind yourself that, ultimately, there is no harm; you are *already* safe even without doing anything?”

They ask me what I'm talking about.

I say, "Feeling safe is a feeling, a sensory (feeling) experience – *not* a mindset. The physical heart grows calm and a physical wave of peace comes over you – that's *feeling* safe. If a person loses that safe feeling, for a moment, he can regain it as soon as he remembers that, despite all the worries and warnings of the world telling him otherwise, he is ultimately safe. No matter what happens, he is safe."

A common PDer response to this has been, "Only an idiot could ever feel that way!"

Many PDers tend to think that wariness and being careful *makes* them safe. They have taught themselves that they are not *inherently* safe. But the problem is that no amount of caution and correctness can ever make a person actually feel safe if his underlying platform is that he isn't safe. If a person does not feel that his basic starting point is one of safety, an infinitude of Yale locks, vitamins, and socially "correct" behaviors will not make him feel safe.

I know I am being repetitive, but I have found that PDers need to hear this several times before they begin to suspect that what I am saying is *not* what they think I am saying. I repeat: being guarded is the *opposite* of feeling safe. If a person is careful and guarded, it means that he does *not* feel safe. If he imagines that by being guarded and careful, he will be able to feel safe, he truly does not understand "feeling safe."

I need to restate the above many times to most PDers who become stuck in partial recovery. It may take months before they begin to differentiate between their own definitions of "feeling safe" and the correct one. When they finally do grasp the difference, they are often stunned.

One PDer's statement that he'd had no idea what "feel safe" actually meant was particularly ironic; he had made a career for himself in the field of Risk Management. From a professional standpoint as well as a personal one, he honestly thought that feeling safe was the outcome of perpetual wariness and risk assessment. He had *consciously* thought that being *wary* and feeling *safe* are one and the same, even though they are obviously opposites.

Broadest meaning of safe

Many PDers think that "safe" only has to do with risk of grievous bodily harm. When I said to them, "You must learn to feel safe," they assure me that they feel safe all the time because they are always guarded and they know that no one is going to be able to hurt them. But they clearly do not feel safe. The following examples will demonstrate.

One brilliant PDer insisted she felt "safe" because she had a good, challenging job, a steady boyfriend, and all the trappings of a good life. However, immediately after her foot healed, she noticed a dramatic change in how she related to her co-workers. For twenty years, she and a small group of co-workers had gone for a half-hour walk during the lunch break. They chatted during the walk.

Following recovery from her foot injury and the heart dissociation that she'd used to maintain ignorance of that injury, the ex-PDer noticed a change in her conversation skills during the lunch break walk.

“In the past, I would follow the trend of conversation, and when it seemed that it was my turn, I would contribute something appropriate. It took a lot of mental effort and making choices so that I could be certain of saying the right things, and using up no more than my correct share of the time. But this last week, I just say whatever comes to my mind. I don’t keep track of whose turn it is or try to create an intelligent thread to link what everyone is saying. It’s fun! As I started being spontaneous with sharing my own thoughts instead of worrying about my “contribution,” I suddenly realized that everyone else was also just being spontaneous – and always had been. They’d been having fun; they enjoy sharing their thoughts! For twenty years, I had been making it so difficult for myself. I’d had no idea it was supposed to be pleasant!

“Feeling unsafe” can apply to the fear that makes a person rigidly self-monitor conversation and behavior. Feeling unsafe is *not* limited to life-threatening events. PDer’s behaviors may be consciously guided by a need to feel safe from social solecisms, from spiritual faux pas, or from any sort of mental, emotional, or physical behavior that the PDer might deem not quite correct. Many PDer’s also feel unsafe to the point of paralysis because of what *others* might deem incorrect.

For example, several PDer’s have been relieved to learn that they are not the only ones who are afraid to turn around and reverse direction when walking in public. If they suddenly realize that they have left something at the last shop, or forgot to bring an umbrella, they will not turn around in mid-stride. They will walk to the next corner, turn the corner, and turn the next corner, and so on, until they have made their way back to their goal without ever having abruptly turned around in such a way that a random observer might be able to say, “Look at that nut. He was walking one way, and then he suddenly turned and walked the other way.”

This bizarre fear of being observed doing something that might be deemed erratic or illogical – even to a perfect stranger, or to no one but the empty sky – is not uncommon among PDer’s. Like the woman who only spoke when she was certain her words and timing would be deemed “appropriate,” many PDer’s are terrified to move or speak in public in a manner that might be judged anything but “correct” or even “good.”

And yet, they do not imagine that they are feeling unsafe. They will argue that they always feel safe. However, the care that they take to be “correct” in front of perfect strangers *is* fear-based. They perform these carefully thought out actions because they would feel at risk if they didn’t.

Also, many PDer’s are terrified of criticism. If criticized, they might argue without end in an attempt to show that they had thought and behaved correctly. Many also have an absolute inability to laugh at themselves or tell funny stories about their mishaps and mistakes.

When I talk about PDer’s not feeling safe, I am very much including these *non-mortal* fears that reflect a lack of safe feeling in the heart.

Some selective dissociations ceases during “safe” times

Some PDer’s do feel safe sometimes. When they do, they stop dissociating from their hearts. It is fairly well known that some people with even advanced PD can move perfectly normally if they are doing what they consider to be a “safe” activity, which might be anything from the crossword puzzle to oil painting or playing the violin on

one's birthday. While doing those activities, the PDer is able to recall the feeling of calmness that is associated with safety. His heart tumbles down into "safe" mode. As soon as the "safe" activity is over, the PDer's heart is hoiked back up into the tense-and-wary positions of dissociation or emergency.

Feeling safe is supposed to be the normal mode, not a special condition

When a person or animal feels safe, he can manifest that feeling through "seeking" behaviors: curiosity, eating, and self-expressive movements and vocalizing. Seeking behaviors are dopamine-driven. They are the neurological opposite of fear-based behaviors.

This is a very important principle for PDers to understand. In order to re-associate and deal with the submerged, dissociated pain that is paralyzing him, he must first recall how the heart feels when it is in normal, *safe* mode: *before* a person can "shake off" an intense adrenaline or dissociation experience, he must feel safe.

So, on behalf of those people who think that feeling safe means being wary, I will explain further. Those of you who understand what it means to feel safe, please jump ahead to the next section.

Feeling *unsafe* is only supposed to occur when a person's life, territory, or children is immediately threatened.¹

This unsafe feeling should occur during life-threatening emergencies, but in our modern society with freeways and deadlines, it might also occur frequently throughout the day. Feeling unsafe should release a bolus of adrenaline. The adrenaline can be metabolically broken down and gotten rid of within about ten minutes. Then, if the emergency or threat is over, feeling safe automatically resumes. In healthy people, feeling safe occurs automatically when the unsafe feeling is finished. You might say that, for healthy people or animals, feeling safe is the default mode: what you might call the basic, or normal, mode. Unless there are temporary, extenuating circumstances, feeling safe resumes *automatically* when worrying stops.

Feeling safe *cannot* be attained by getting rid of danger. Danger will always exist. Trying to make oneself feel safe by getting rid of danger is like trying to get rid of the darkness in a room by removing it with a spoon – it can't be done: you can't *remove* darkness – you must turn on a light. In the same way, you can't remove danger – you must presume safety.

The guiding force of a chicken

Some PDers have protested that, by discussing safety as an ever-present condition, I appear to be talking about spiritual issues and faith. They conclude that, therefore, I am not being scientific. I disagree. Our culture has learned to associate some of these terms with spirituality, for obvious reasons. But a person need not be spiritually inclined in order to feel safe. He need not be religious in order to experience his living presence *inside* his body instead of mentally watching himself from the outside.

¹ Appendix xxx, The Adrenaline-Dopamine Relationship, lists the highly specific conditions under which a healthy mammal temporarily slides into an adrenaline-dominant phase.

After all, dogs feel safe without going to church. And what about chickens? Almost anyone (except maybe a heart-dissociated PDer) who has spent a lazy ten minutes watching chickens scratching for food or rolling in the dirt (they *love* to roll in the dust!) will have resonated with their feeling of safety, that “not a care in the world” feeling that they project, and yet, chickens don’t ponder whether or not there is a Higher Good. As for cats, cats feel so darned safe that they do whatever they want, when they want, and no sermons or pastors required.

When these animals “feel safe,” they are feeling the sensations of vibrancy, of life within. They feel that vibrancy *inside* their bodies. They use that feeling of vibrancy to activate their bodies.

Of course, during an emergency, when they are being attacked, animals lose awareness of that sense of vibrancy. They shift into sympathetic mode for the duration of the attack. But when the immediate danger is over, they go back to feeling the vibrancy that is inside their bodies. They feel safe again.

Very possibly, one of the real differences between animals and humans is that humans have the potential to consciously remain in a calm, joy-filled state in *spite* of danger or pain. This requires enormous emotional maturity. Many PDers, oppositely, constantly anticipate pain or danger and are *unable* to even know what I’m talking about when I describe the feelings of a safe heart. Many of these PDers insist that they are spiritually advanced because they are emotionally rigid. They assume that, because they are numb, they are calm. They are wrong. Calmness is a sweet sensation that results from feeling safe. And feeling safe is a sensation that occurs in the heart – a sensation that they may not even remember.

No particular religion is required

No particular religious belief is required for a person to feel the vibrancy inside that directs his actions, thoughts, and metabolism. Many people who are utterly guided by their sensory feelings have no thoughts whatsoever of a “spiritual” plane. Again, any person or animal can feel the vibrancy within – no religion required.

But when a person or animal temporarily dissociates from his heart, he cannot feel that vibrancy within. Depersonalization, the sense of perceiving one’s body from outside one’s own body, is a common symptom of dissociation. And it is the opposite of feeling vibrancy *within* the body.

So, without forcing any spiritual meaning into it, I can say that feeling safe is related to being aware of the feeling of vibrancy *within*. Of course, people with spiritual leanings will say that what I am talking about is the soul or The Comforter or the *Spiritus Sanctus* or whatever, and they will all be correct.¹

But for the person who shuns any religious reference, I can still say that the physical sensation of vibrancy that animals feel inside when they are not in a state of emergency is the same thing that partially-recovered PDers are selectively *lacking*.

What partially-recovered PDers have is a feeling of not being safe. The feeling of not being safe is associated with decreased awareness of the vibrancy inside the body.

¹ I know that many of these terms have faith-specific nuances of meaning. A discussion of these finer points is unnecessary for the subject at hand and is beyond the scope of this book.

Only an idiot

Safety is dynamic and joyful; it can be spontaneous and it can be powerful; it is mother's milk and it is delicious. Safety is the opposite of caution and double-locking the doors.

When I explain safety in this manner, some PDerS say to me, "Then only an *idiot* can feel safe," putting a lot of top spin on the word "idiot." (PDerS tend to be highly intelligent, and often are very proud of their intelligence. Coming from a PDer, "idiot" can be a severe insult.)

If I know the patient well enough, I might lovingly retort, "Only an idiot would work at feeling *unsafe* to the extent that he tremors and can't even walk or swing his arms..."

Don't have to be a saint

A person can recover from Parkinson's disease even if he has no "spiritual" leanings. But he may need to let down intellectual guard down and learn to be at least "as safe as an idiot." If he hopes to recover, a PDer must learn to feel safe. Helpless, dumb (idiot?) beasts feel safe except for those fleeting moments when they are in imminent danger. PDerS must learn to feel as safe as a mouse.

DEFINING FEELING

OK. Now I have to try to define feeling for people who can't or don't want to feel.

I have been trying to do this with PDerS for several years now. During these years I have developed greater compassion for Anne Sullivan. I've wondered how she must have felt as she tried to teach Helen Keller how to speak, spell, and read. Did she ever feel as frustrated as I? How can feeling be taught to a person who prefers to be numb? How can sensory perceptions be explained in words?

As the poet Rumi said:

"Out beyond ideas of wrongdoing and rightdoing,
there is a field.

I'll meet you there.

When the soul lies down in that grass,
the world is too full to talk about.

Ideas, language, even the phrase *each other* doesn't make any sense."¹

¹ *The Essential Rumi*, Coleman Barks translation, Castle Books, 1995, p. 36. The "field" of this poem is the locus of the sensory awareness of vibrations of love. When one lies down in that field, which is to say, "relaxes into those feelings that start in the heart," words are useless – they don't even make sense. "You" in this context refers to God, as well as to you, the reader, in whom the indwelling One God is manifested as your own unique vibrations of love.

A famous example of the inability to use words to define the feeling that I am trying to explain occurred at the trial of Jesus. Pontius Pilate asked Jesus "What is truth." The record shows that Jesus said nothing. Most people assume that Jesus was ruggedly inert during those answering moments. But Pilate's *response* to Jesus's moment of silence suggests that Jesus answered the question.

Pontius Pilate, after receiving a silent answer to his question in the form of a breath-taking wave of love vibrating through his heart, refused to have anything more to do with the interrogation of Jesus. In response to Jesus's perfect, wordless answer – a wave of heart feeling – Pilate said to the gathered crowds,

As Rumi and every other poet knows, feelings, which is to say, sensations, cannot be described in language. I will try. But I console myself in advance that, if I fail, I know that far, far better men and women than I have tried and not been able to make people understand.

Here goes. The word “feeling” is a noun; which is to say, feeling is a *thing*. For example, “I have a bad feeling in my stomach about this.” Feeling is also a verb, an action. For example, “I am feeling the wind on my face,” or “I am feeling the existence of my hand even though I’m not looking at my hand.”

Feeling: the Noun

First, I’ve decided to use the word “vibrancy” to describe the general feeling of sparkle in the chest that is crucial to feeling alive, and which many PDers do not feel.

(In the chapters on treatment techniques xxx I define the word vibrant. But for now, in this introductory chapter, if you don’t know what “vibrancy in the chest that expands when a person perceives something of great beauty or grace,” means, just keep reading. Maybe you will understand after doing the exercise in this section. If not, look it up in chapter xxx.)

Although the poets try, no one can describe *what* the vibrancy in the chest feels *like*. So I will describe *how* a person can go about feeling the vibrancy within.

Even if a person has no idea of what I am referring to when I describe this feeling using words like “vibrancy” or “vital spark” or “expansion,” he can learn to feel it himself by paying attention to his breathing.

If a person pays attention to the physical sensations that occur when he inhales and exhales, he can notice a faintly different sensation when comparing the inhalation and the exhalation. In addition to the sensations of movement of the muscles and tendons, and the air moving across the larynx, throat or sinuses, a very subtle sensation occurs in the chest during the inhalation that does *not* occur during exhalation.

This faint sensation has been described as a tickle, a warmth, a sense of expansion, a vibratory sensation. A thousand words might be used to describe it, but it is perhaps most accurately described simply as the sensation that accompanies inhalation, but not exhalation, and which is apart from the physical sensations of structural movement and air flow.

An exercise

Sit quietly for several minutes and notice how it feels as you breathe in and out. (In the case of a person with Parkinson’s, he may need to do this for five to ten minutes at a time, several times a day, for several weeks or more before he starts to notice the difference between exhalation and inhalation.)

“I will not be guilty of the death of an innocent man.” Pontius had judged Jesus based on Jesus’s unmistakable answer to the question and found him innocent of all charges. He “washed his hands” of the whole affair, and turned the situation back over to the lesser authorities, having made it clear that Jesus was innocent.

Do not try to control the breath or analyze it. Don't think about it. Just feel it the way that a pre-verbal child would feel it. No words. Notice the physical sensations. Feel the chest move, feel the movement of the air.

After feeling the in and out breath many times, try to decide whether or not you prefer the sensations of inhalation or the sensations of exhalation. This will help you pay closer attention. As you try to differentiate between the two sets of feelings, you will become aware that the two sets of feelings *are* slightly different: the inhalation is accompanied by a faintly excitatory quality. This is a tiny version of the vibrancy sensation that we are talking about.

Bigger versions of vibrancy

In a healthy person, the underlying, general feeling of vibrancy is larger than the small hint of vibrancy that accompanies inhalation. When I say larger, I mean that it occupies a bigger area in the chest. It may also be larger in the sense of more dynamic, more blatant, easier to feel. A healthy person can feel this vibrancy at any time by shutting down his internal monologue and paying attention to the vibrancy.

When most people behold something of great beauty or grace, their awareness of this vibrancy becomes larger than usual. When awareness of this vibrancy becomes larger, one experiences a physical sensation as if the chest is expanding even if the actual chest muscles are not moving any more than usual.

The illusion of expansion

Seeing a beautiful vista does not technically increase the amount of vibration that a person has: this is an illusion. What actually happens is that, stunned by the beautiful experience, a person lets down his mental guard and his silent running monologue for a moment – or makes his monologue less obtrusive than usual. In the ensuing mental silence, the vibrancy that is always present can be noticed more easily, or noticed over a larger area than usual.

As soon as a person reactivates his judgmental mind or starts to worry, the ability to perceive the vibrancy is reduced. The diameter of the area in which the vibratory feeling was noticed seems to shrink.

Actually, the feeling is there all the time.

The breath is a reminder – and a temptation

The tiny portion of vibrancy that occurs with each breath is a tiny reminder of the larger vibration that is sustaining the Self at all times – whether a person has a body or not. It is also the source of emotional preferences. As a baby enjoys the sensation that accompanies breathing, he learns to prefer breathing to non-breathing. This is the basis for all other selfish preferences that the child may come to develop.

Why bother breathing?

Babies and animals live in a world of feeling. They experience a warm, vibratory, mildly excitatory feeling from inside the context of their bodies. With every inhalation, they feel an extra tiny bit of this vital force.

The sensation is so glorious that every tiny bit is worth having. This is why we bother to breath; this is why living beings want to stay alive: every breath adds a

momentary extra bit of obvious vibrancy to the enormous amount of (often ignored) vibrancy that is actually in and around us, permeating us. And this sensation is so peaceful, so loving, so thrilling that we will do almost anything to keep breathing.

As babies grow up and become word-based, it becomes easier for them to lose awareness of the larger vibration: its warmth, tickle, and joy. But deep down, even when they are grown, they know that they *can* have this feeling. We all want increased awareness of this indescribable feeling. Subconsciously, no matter how much we have blocked out our awareness of the underlying vibrancy within, we get a tiny refresher course in this feeling with every inhalation.¹

¹ This is why the Vedic masters said, “Breath is the secret,” and “Master the breath.” They did not mean that a person should learn to hold his breath – a common misunderstanding. What they meant was, focus your attention on the feeling that accompanies breathing. When you can recognize and differentiate from the other chest sensations that soupçon of vibrancy, you can place your attention on the vibrancy. You can allow your heart to resonate with that isolated sensation. As your heart waves resonate with that particular sensation, the basis of all other sensation, the heart vibrations can tune in, like a radio, to the larger fields of the same vibration that are permeating your body’s spaces – which you have forgotten, ignored, or shut out. As you let yourself resonate with this larger vibration, you perceive that vibratory feeling even more. If you continue to allow that feeling to expand, also referred to in meditation instruction as “going deeper within,” you are actually not expanding anything or going anywhere. All you are doing is allowing yourself to *notice* a larger diameter and greater nuances of that vibratory, warm, tickling, excitatory vibrant sensation.

This chapter’s technique of noticing the difference between inhalation and exhalation is not the same as yogic breathing practices or other life-force control practices. Those practices assume feeling safe and relaxation as a starting point – as a given. After starting with awareness of these sensations, the more advanced yogic techniques teach how to neutralize the preferred feeling that accompanies inhalation. By accompanying inhalation and exhalation with specific mantras (words) or with specific thought patterns, the *feeling* of the vibrations of inhalation and exhalation can be made to match each other: the breath experience is neutralized. When inhalation has the same emotional value as exhalation, breathing becomes uninteresting. The prejudice in favor of inhalation is gone. A person can then choose whether or not to breathe. If the restless mind has been tamed, then, in the serene peace that accompanies breathlessness, the ability to feel *greatly* increases. The sensory world expands even beyond the perimeter of the body. One can feel the chirping of a bird as if the chirping is vibrating within himself. One can feel the glory of a sunrise, or a flower, as if the vibrations of the sky or the petals are a vibrating part of himself. And these are the beginner techniques. More advanced techniques can help a person apply this expanded awareness, this expanded ability to feel, towards transformation of ego-based sensation into the ego-neutral vibratory components that lie behind the cruder, sensory-attuned vibrations.

There is far more to meditation than just noticing the breath. This bit of explanation is just to give the merest hint of what is meant by “the breath is the key” and suchlike phrases. A lengthier discussion is beyond the scope of this book, but I have raised the subject for a reason. The main reason for this tangent is this: many PDers assume that they are being spiritual by keeping their heart numb and trying to kill their ability to feel. They need to know that this is the opposite of spiritual endeavors. A PDer’s bizarre set of semantic understandings allows him to justify behaviors that are often the exact opposite of what he so dearly wants to attain. So, in case the PDer has been doing Tai Qi, prayer, meditation, Qi Gong, yoga, or any other (joyless, for many PDers) practice in order to learn self-discipline, he needs to know that the actual goal of these practices is joy. Mastery over physical techniques and mental practices allow him to regulate his mind and banish prejudices – even the prejudice in favor of breath – so that he can increase his ability to *feel*. The more he can *feel*, the more he can *feel* the joy that vibrates just behind the seeming reality of matter. Many of our PD patients have admitted to spending decades in dutiful, externally “correct” practice of so-called “spiritual” techniques while never *feeling* anything, least of all joy.

Did I explain anything?

That's the best I can do. While this explanation might not describe what "Feeling: the Noun," really feels *like*, it is probably as physiologically detailed as I can give or need to give to explain "feeling: how to experience it."

They say that it is impossible to describe the taste of an orange. This is because the taste of an orange is a feeling, and it is impossible to describe a feeling.

"But it's such a small thing!"

PDers are usually disappointed because the sensation that they experience after doing this experiment is small and somewhat subtle. Many PDers have protested, after feeling this difference, "It's not such a big deal" or "I didn't feel so anything that made me *blissful*."

I don't know what they were expecting, really. Cataclysms and soul expansion, maybe.

I have to keep reminding PDers that I am not teaching them how to be great mystics: I am reminding them how to be living humans. We're starting out small.

Over and over, PDers who have recovered *after* having been stuck for a while in partial recovery have been amazed when they learn to feel an expansion of this tiny sensation – and a wave of relaxation and physical suppleness. They protest, "But this is so easy! It can't be this easy!" Until they surrender, they imagine that something enormous has to shift in order for them to let go of their tension.

"It can't be this easy," is the common cry.

Also, sometimes, after briefly experiencing for the first time the relaxation that comes with focusing on the heart feeling, they are not able to feel it so easily the second time; they seem to have convinced themselves that what they need to feel *must* be something more arcane, more difficult to attain. Possibly, their wary hearts seem to get more guarded after having been unexpectedly breached.

Yes, the vibrant feeling can be small, and feeling it is simple. But it is not always easy to feel it, at first, even though it's simple. In retrospect, after one learns to recognize it and feel it at will, it will have been simple. It's easy when you know how – sort of like riding a bicycle. In retrospect, after learning to ride a bike, it's so simple. But to the frightened beginner cyclist, it may not seem as if it will ever be easy.¹

Summary of "feeling: the noun"

Getting back to the main idea of this chapter, "feeling: the noun," refers to a physical sensation. When a healthy person says, "I have a feeling that I should (or shouldn't) do something," he does not mean that he has *thoughts* on the subject. He

¹ When good teachers give instruction to their students, they almost always start by saying "Relax." Whether the master is teaching Tai Qi, Yoga, sitting meditation, swirling meditation, violin, or bowling, the first instruction is usually "relax." Relax is the first step. The teacher assumes that any student can relax. Most PDers can't relax.

In the treatment technique chapters I am *not* teaching PDers how to be great souls or how to experience breath-taking waves of vibrancy. I am trying to teach them how to attain "relax." Relaxation must be preceded by feeling safe in the heart. So I also have to include instruction on how to feel safe.

means that the vibrations in his heart or chest that direct his intuition have moved in a manner that lets him *feel* what he should do. Feel. Not think.

When a person says, “I feel for that poor person,” he doesn’t mean he is *thinking* about him. He means that a sensation inside of himself is moving or vibrating in a manner that he cannot ignore. Feel. Not think.

PDers might want to take careful note of the next fifty or sixty times that they read or hear sentences with the word “feel,” and ask themselves if they have usually misunderstood the word to mean “think” or “thought.” They might be surprised. If they try to justify this by saying that everyone uses the words “feel” or “feeling” to mean “think” or “thought,” then they get to ask themselves *why* they have made this assumption. The assumption was wrong.

Feel: the verb

To feel means to perceive or to be aware of through physical sensation. There are other meanings, as well, but this is enough to be getting along with. Feel does not mean “think about” or “analyze” or “try to cleverly root out the motivation behind something.” Feel means be aware of through physical *sensation*.

An infant feels with out analyzing. Non-verbal creatures learn about and remember the world around them by feeling it rather than thinking about it. Feeling is done with all five senses and with the sixth sense. A “hunch” that comes from the sixth sense is a palpable feeling – a vibratory sensation that resonates with the wave patterns of the heart and which, when conveyed to the brain, is translated into a thought.

As humans become verbal and take on the murky cloaks of the various world cultures, teeming with dos and don’ts, fears and rules, they begin to let the mind over-ride their feeling experiences. If they aren’t careful to keep feeling, they can become hardened, living solely on the basis of mental processes rather than in response to the feeling. Many PDers create a bizarre blend of feeling and thinking: they might retain their compassion for others while becoming unable to feel emotional responses to their own joys, sorrows, pains, or pleasures. Soft to the world, they can be gradually turn hard as stone with regard to their own sensory existence: I cannot even guess at how many PDers have told me that they have never been able to cry at a funeral or in response to the loss of a loved one.

As a human starts to imagine that he is his body, instead of his awareness of vibratory feeling, and begins to mentally apply all the cultural rules and fears to the actions of his body, he reduces his memory of what his actual vibratory self feels like. When he dissociates from his ability to feel, as well, he loses his awareness of what it really means to be alive.¹

¹ Many world religions use phrases like “You don’t need to go *seeking* God (or Truth or Love); you already have it within you: you only need to improve your awareness of the Feeling within which is already there.” For example, in the Christian gospel, Jesus is quoted as saying, “Reverse your outwardly oriented mind, and focus on what’s within, for the kingdom of God is inside of you.” In the English language, St. James translation from the ancient Greek, this phrase was translated as “Repent, for the kingdom of God is at hand.” However, in the original Greek, the word that became translated into the English word “repent” actually means “reverse.” And back in the days when the bible was first translated into English, the phrase “at hand” meant “right here, right now.” So Jesus was saying turn your thoughts inward and notice how you feel: the loving feeling, God, is right there, inside of you.

The first bicycle ride

One PDer worked at feeling the difference between inhalation and exhalation for several weeks. He was finally able to notice a faint difference. So far, so good. But in the next phase of therapy, in which a person has to learn how to *expand* his awareness of that feeling, he was not able to expand the feeling in his chest beyond a tiny spot, an area a mere fraction of an inch in diameter.

So he branched out into trying to notice other feelings. He tried to feel the difference between how he felt when he was with his very sweet daughter and when he was away from her. He was able to zero in on the *difference* between those two states. He felt that the difference between those two states extended over a large area: throughout his chest. He exclaimed, “Oh! It’s just *feeling*, without talking. Like when I rode a bicycle for the very first time.”

I told him I was going to use his bicycle comparison in my book. His “first time on a bicycle” comparison is a wonderful description of feeling as opposed to thinking. The joy that occurs when a person first masters the coordination of keeping a bicycle moving and upright is due to the experience of pure feeling and the concomitant drop-off in mental processing. The new cyclist is completely focused on the coordinated *sensations* to which he must pay attention. Keen focus on those physical feelings are required to keep the arms and legs and torso all moving in that peculiar, bike-riding rhythm.

Because the new cyclist is absolutely absorbed in the necessary physical sensations, his thought stream shuts down for a moment. The pure joy of that first flight on a bicycle occurs *because* the cyclist is aware of his purely physical sensations and is not paying attention to his mind. For that fleeting moment, before the movements become second nature and the mind turns back on, the cyclist has experienced the same sort of

A non-denominational quip that I like says the same thing: “If you feel apart from God, guess who moved.”

But even if a PDer scorns religion because he thinks “God” refers to some grey-bearded man enthroned in a dusty corner of the universe, or he blames the world’s religions for the strife and divisiveness that humans had perpetrated in the *name* of religion, he must still admit that we are permeated with vibrations. Everything from radio waves and television waves to brain and heart waves are throbbing through the universe – and through our bodies. Healthy people can feel some, a few, of these waves. All parasympathetic sensory experience is based on perceiving these waves and noticing how they resonate, or not, with the heart. Spiritual people of every faith work at reducing the interference of the ego-based mind so that they can better feel these vibrations and the love behind them. Through mentally focused prayer and projection of heart waves of compassion, they also participate in *creating* waves!

At the very first talk I gave on my research into Parkinson’s, when I addressed the Parkinson’s Support Group of Santa Cruz, a man stood up and said, “I’m leaving. I suspect a spiritual undertone to your ideas.”

At the time, I was only talking about Asian channel theory, so I was amazed that he felt so threatened. I had not yet learned to feel safe enough to speak from my heart while also including references to modern physics and examples from great spiritual teachers. So, at the time, I was unable to answer him even though I felt like saying in reply to his suspicion, “Is there any part of existence that does *not* have spiritual undertones? Isn’t every atom in the universe made up of vibrations?” But I remained silent. I understood at the time that he was wary of religious proselytizing. Now, ten years later, I understand that his fear ran much deeper. He was afraid of his heart, he was afraid of feelings, he was afraid of being able to perceive the vibrations within himself that some people refer to as “soul.”

immersion in pure physical sensation that an animal has, or that the infant has when nursing.

What does feeling feel like?

This PDer had been asking me repeatedly what feeling felt like, and I was of course unable to say anything other than “It’s just feeling.” I turned the tables on him as soon as he was able to feel the difference within by comparing how he felt when he was with his daughter or not. I asked him to describe the feeling that he’d just felt while thinking about his daughter, or the feeling of riding a bicycle for the first time.

He shook his head. He seemed to have temporarily lost his extreme ability to define and describe. “There’s no word to describe it. It’s just feeling.”

A drop of love

Another PDer who kept asking me how to feel instead of think finally got a sense of what I meant. She was able to feel the tiny vibration of something that occurs with breathing, but she was never able to expand it beyond a four-inch diameter sphere in her chest. I asked her to imagine the vibrant sphere pressing up against her physical heart, which was in the center of the sphere. The vibrant feeling could snuggle up against her heart, comforting it. She liked that feeling. Then I asked her to imagine that the vibratory sphere was liquid, and it was floating in a sea of liquid that filled her chest. I asked her to allow a drop of love to plop into the sphere of the vibratory feeling, setting off waves of vibration that moved outward through her chest.

She did this, and then said, “I could *feel* that.”

I asked her what it felt like. She shook her head. She was usually articulate about her aches, pains, and symptoms, but suddenly she had no details. “It doesn’t feel *like* anything except what it was: it was just a feeling. I stopped thinking. I was just feeling.”

Then she became more animated: “It’s so easy. Why didn’t you ever tell me to just not think?”

I paused for a very long moment before I said, “What?”

Then she laughed. For nearly a year I had been asking her to try and quiet her non-stop mind, to stop thinking, and just feel. We’d experimented with more than thirty techniques that help quiet the mind and enable a person to “feel.” I had used every word and synonym possible to describe what she needed to experience. She was also a meditation student and had been studying techniques for stilling the mind for a long time. So she laughed at herself. She continued, “I didn’t understand. It’s so easy. I just had to stop thinking and start feeling.”

I asked if she could be more explicit; was there anything she could suggest that might help another PDer learn to feel what she had just felt?”

After a thoughtful pause, she said, “No. They just need to feel what’s happening inside. Stop thinking; just feel. But I don’t know how you’d tell them in a way that they could understand. I didn’t understand. They just have to do it.”

Suppose the roof falls in

Another PDer had been determined that he would never be able to understand what I was talking about. He preferred exhalation to inhalation. The vibrant feeling that

accompanied inhalation made him nervous. He did not want to experience an enlarged version of that feeling.

I asked him if he felt safe. He said he did.

I knew darned well that he didn't feel safe. For one thing, he often reminded me that he was scared he could never recover from Parkinson's disease.

I asked him if he felt so safe that he'd be OK even if the roof fell in on him right now, right this moment.

He replied, "Of course not. That wouldn't be safe."

I told him to pretend that he would be OK even if the roof fell in and killed him. He paused for a moment, and then said that he would be OK even if the roof fell in.

I asked him to make himself feel really safe, so safe that even if something blasted his head off and his detached head caromed across the room, he would still be safe.

He said OK, and relaxed. He actually relaxed. I'd never seen him relax before.

A moment later, he said "Oh."

I asked how he was feeling. He smiled and said, "Safe."

I asked what that felt like. Instead of giving me his usual wordy answer, he said, "Just a feeling."

I asked him how big the feeling was. PDer's usually need to practice before they can expand this internal sensory awareness of vibrancy at will. Some PDer's can feel the vibrant feeling occupying only a spherical space of less than a sixteenth of an inch in diameter. They need to work at it for months before they can dare to let themselves experience that feeling over a larger area.

He replied, "It's spread over my whole body. I can feel it in my whole body."

I asked him what it felt like.

He shook his head as if to say no. "I can't describe it; it's just a feeling."

I have given the above examples, all of which ended with the PDer saying, "Oh. It's just a feeling. I can't describe it; it's just feeling," to make the point that feeling cannot be described. I am also including it because it shows I'm not asking people to feel something shocking or stunning. I'm just asking the PDer to dare to feel what's going on inside his own body.

Again, the feeling that the PDer needs to recall is not enormous, nor is it covered in glitter or rainbows. It's a tiny sensation, and it can be enlarged. It is related to the feeling that a healthy person can recall instantly to mind when he remembers that he is safe or which expands in size when he perceives something of beauty or grace and which can be invoked via recall, if one errs so far as to temporarily forget it.

Being able to feel this sensation will not, by itself, cure Parkinson's disease. But it's a necessary first step in learning how to feel safe enough that one can start to think about re-associating instead of dissociating.

I'd rather be dead

The first time a PDer told me, in response to feeling inhalation and exhalation, that he preferred exhalation to inhalation, I was alarmed. Basically, he was saying that he'd rather be dead than alive. Now I have become accustomed to hearing this, now and then, from some partially-recovered PDer's. If I ask why they prefer exhalation, they say

While digging in various resources to find some commonly used English words to refer to the numen (an older English word that means indwelling spirit, internal guiding force) I looked up aesthetic in the dictionary. This word means with feeling or perception, and is the root word for *anaesthetic* (without feeling). (The prefix “an” means “without.”)

But when I glanced up at the previous word, aesthete, the word had one line of type defining aesthete as “a person who perceives.” The entry had another twelve lines pointing out that the word is also “used derogatorily to connote effete-ness, decadence, a person who artificially cultivates artistic sensibility; a synonym for dilettante (used disparagingly to mean one who dabbles in the arts); virtuoso (sometimes used derogatorily to suggest faddishness); and so on. Nearly all of the meanings for the word aesthete, a person who feels, were negative.¹

I was intrigued. I looked up “sensitive.”

The first three meanings for “sensitive” were value-neutral. Then came the negative connotations: easily hurt, tender, raw; easily offended, disturbed, shocked or irritated, etc, as by the actions of others; touchy.

Ooh. These were not good connotations. No one would want to be considered “sensitive!”

I looked up “feel” and “feeling.”

The dictionary had sixteen meanings for feel and ten for feeling. Most of the meanings of “feel” were value neutral, such as “to touch” and “to perceive.” The meaning that I am using was meaning number two: the value neutral “to perceive or be aware of through physical sensation.”

Of the ten meanings for “feeling,” several had positive connotations such as “a kindly, generous attitude; sympathy, pity” and “the emotional quality of a work of art.” But also listed under “feeling” was “Synonyms: feeling: a subjective reaction that usually connotes an absence of reasoning; emotion; passion, including intense sexual love or intense anger; sentiment.”

In English, all of the words that we have to connote perception and feeling are somewhat or heavily weighted towards negative meanings; meanings that suggest touchiness, phoniness or out-of-control emotions.

One of the newest uses of the word feel is in the phrase touchy-feely. Touchy-feely means not rational, subjectively self-indulgent, or led astray by emotions. Calling someone or something “touchy-feely” is *not* a compliment.

I began to see why so my partially-recovered PD patients were able to distance themselves from or even become uneasy around the words feeling, felt, and feel. Their nearly perpetual reliance on the sympathetic system pushed them towards the negative meanings of any words related to feelings.²

¹ *Webster's New World Dictionary*, second college edition, World Publishing Co., 1970

² When I discussed with a Frenchman the English lack of a purely positive word or phrase to connote the feeling of expansion in the chest, he laughed out loud. Then, in patriotic support of the long-running, mutual, and somewhat joking love-hate relationship between the English and the French he

In chapter xxx of this book I mentioned the patient who would start trembling any time I said “feel,” “felt,” or “feeling.” After perusing the dictionary, I had a deeper understanding of his fear of feeling. Feeling can be a “bad” thing. In English, a person with even a mildly negative orientation will be well within the legitimate meaning of these words if he chooses to impute negative connotations for them.

The PDer who tends to get stuck in partial recovery, having such a predominance of sympathetic mode negativity, may understandably choose the negative associations for a word like feeling. And considering that many of them are selectively dissociating from their ability to feel, as if feeling is the problem, it becomes very clear that feeling is to be avoided. Feeling is bad, not good.

But in order to recover, a PDer needs to understand that he has *decided* that feeling is the problem. When he leans toward the negative meanings of the word “feeling,” he is making a choice. That choice has paralyzing consequences.

Chapter summary

Most of the words that PDers truly did not understand had to do with heart and the sensations that a person experiences when he stops thinking and feels the vibrancy in his chest.

We had spent years being baffled by PDers who listened carefully to us and then behaved in a manner that suggested they were doing the opposite of what we were asking. We only discovered, very late in the game, that PDers have formed weird, even opposite meanings for many words. The list of these words includes safe, neutral, calm, vibrant, surrender, heart, imagine, feel, and feeling.

When we finally realized that PDers honestly had no idea what we were talking about most of the time, we were baffled as to how to proceed. I spent several months asking partially-recovered PDers to define certain words in order to ascertain how broad and deep the confusion really was. Many spouses helped out, assuring me that the PDer spouse had no idea what was meant by words such as relax or feel. Some spouses told me not to waste my time asking the PDer “How are you today?” because the PDer wouldn’t know. Sure enough, many patients responded to this basic question in all sincerity with answers like, “Oh, I don’t know, I guess I’m fine...” or, if I pressed for details, they would say “How should I know how I’m feeling? You’re the doctor; you tell me.”

The spouses were often deeply relieved to be able to talk with someone about the supposedly brilliant PDer’s seeming inability to truly understand so many basic words that have to do with feeling. Shared feelings can be a delightful part of marriage. These spouses had often been struggling for years with the loneliness of sharing a life with a person who thought that “feelings” were *thoughts*.

Between the input from spouses, the stunningly incorrect word definitions of PDers, and the exercises that we did with the PDers, we began to appreciate the *depth* to which some PDers had dissociated from their own hearts.

This semantics breakthrough may seem unimportant, like “an exercise in semantics.” But for the Little Project, it was an enormous boon. We finally realized why

exclaimed, “Ah! That explains so much about the English!” He then threw his arms out in a bounteous gesture and proclaimed, “It is Love with a capital L!”

PDers didn't understand some of our most basic questions or homework assignments. Also, in terms of creating treatment techniques for overcoming heart dissociation, the semantics breakthrough was crucial: we could not use the ordinary words that psychologists or therapists used unless we defined them first in a manner that PDers could understand.

This discovery also added yet more weight to our steadily growing hypothesis that PDers had dissociated from their hearts.



SEMANTICS: PART II

“Keep peace in your heart”

Olga, a bubbly friend of mine, an elementary school principle and a recovered PDer (who recovered very quickly, *never* getting stuck in partial recovery), always ends conversations with, “Keep peace in your heart!” All the students in her school call out to her “Keep peace in your heart!” when she passes by.

I’ve asked some of my PD patients what this phrase literally means. One partially-recovered PDer thought about it for a moment before telling me, “It means be very careful to not let any strong emotions affect your heart.”

I was stunned at this bizarre and unique interpretation.

But as I asked other partially-recovered PDers what it meant, they too assumed it meant something along the lines of “block out emotions” or “remain unaffected.”

“Keep peace in your heart” does *not* mean “stay unaffected by emotion.” It means, “Be filled with the radiant, irrepressible joy of peace and *act* on that joy.” PDers in partial recovery had nearly opposite meanings from the correct one.

Just to be sure, before writing this chapter, I met with Olga, who said that I could use her real name. I asked what it means to “keep peace in your heart.” She said that it means to be filled with joy. It means feel the sensation of love bubbling up inside and express that joy. It means “don’t forget to *feel* your heart.”

Olga gestured with her hands as she tried to put her meaning into words. With both her upturned hands at the level of her chest, she gestured as if her hands were fountains, shooting good feelings of her heart up and outwards in all directions.

I asked Olga what word, in English, defined the feeling that she was expressing with her hands. She was puzzled. Then, because she is bilingual from infancy, I asked her to define, in English, “*Espirito Santo*” (the Spanish name for this feeling). She gestured again. Her hands flew to her heart, and they made rolling outward movements as she said, “Oh! *Espirito Santo*! *Espirito Santo* is, you know what it is: it’s this:” she rolled her hands more expressively, as if singing a Spanish love song with her hands. She looked at me askance, clicked her tongue in mock reprimand and said again, “*You* know what it is!”

I asked again, “But what is it called in English?”

She replied, still circling her hands in front of her chest as if to demonstrate an outpouring of love, “It’s *what* you are, it’s everything, it’s *how* you know. Everyone knows what’s meant by *Espirito Santo*.”

I asked again, “But what do we call it in English?”

She replied, “It’s Love, it’s happiness, it’s joy, it’s why we live, it’s *how* we live.”

And I said, “Yes, but what is the word that we use in *English* that is universally understood to mean specifically the sensation you *feel* that you demonstrate for me by moving your hands that way.”

She thought for a moment, still motioning with her hands. Then she stopped moving her hands and her eyes grew wide with pretend horror. “Omigod. There is no word for it in English!”

I suggested that the literal English translation of *Espirito Santo* has historically been “Holy Ghost.”

“Yes, that works, but...” she protested, “...I’m Catholic. When *some* people say “Holy Ghost” they say it as if it refers to some ghost floating around in space, or some academic philosophy. When *some* people say “Holy Ghost” they don’t mean what *we’re* talking about. But in Spanish, everyone know what *Espirito Santo* means; it’s this:” (she gestured again with her hands and closed her eyes as if in bliss). She continued, “Maybe there *is* no word for *Espirito Santo* in English.”

When we parted, Olga hugged me and reminded me, “Keep peace in your heart!”¹

Safe or at risk: a matter of emotional health

Emotionally healthy people do not try to address the infinitude of potential dangers lurking in the universe. Healthy people assume the relaxed heart feeling of safety *unless* they are in immediate danger.

And here’s the deeper point: emotionally healthy people know that they are *never* in danger. They know this because they think of themselves as their sensations of internal vitality. As Olga would say, her *Espirito Santo* is never at risk. She *is* her *Espirito Santo*. She is *not* her body. The PDer may not understand this at all.

An emotionally mature person does not think of himself as his body. Nor does he dissociate from his body. He appreciates and cares for his body even though his body is merely the vehicle for his real self. The real self is the consciousness that plays at driving the vehicle. The real self is always aware of himself as the vibrant sensations permeating the body vehicle. The driver of the body vehicle, which is *the overarching consciousness* that feels and is aware, is never in danger.

An emotionally healthy person is aware of the sensations generated by having his sensory awareness *inside* his body. Oppositely, many PDers perceive themselves as if they are outside their bodies, observing themselves from the outside, and feeling little or nothing of the sensations that accrue from having their spirit of joy inside the body. As mentioned earlier in chapter xxx, this perception is called depersonalization, and is a common side effect of dissociation.

An aside: depersonalization increases over time

A PDer may be able to say, “I think I know what you mean; I used to feel that way. But now I don’t.” In childhood and even later, PDers may have been able to feel themselves inside the body during activities that they liked. Over decades, however, the habit of depersonalization gradually becomes the PDer’s normal method for dealing with any potential risk. At some point, sometimes even prior to the obvious symptoms of Parkinson’s, a PDer may start spending a majority of his time in a depersonalized state.

Many PDers protest that they *used* to enjoy the sensations in the body while doing sports or some such, and claim that, therefore, they have never been dissociated. However, if they examine themselves honestly, they eventually admit that they have changed through the years. The compelling force behind this change has been their own constant cultivation of wary

¹ Just for the record, Olga is *not* a musician. She is, however, an avid dancer. She always teaches boisterous Latin American dance to the students of every grade. Dance is a part of every open house and festival at her school.

thoughts. The mind develops based on what thoughts and instructions the mind receives. A person who dwells on wariness will eventually have a mind that cannot let go of its negativity.

Then again, some PDeers have not developed a thriving mental culture of guardedness or self-pity. These people tend to recover very quickly – as soon as their foot injuries heal.

Back to emotional maturity

An emotionally mature person knows that his real self, the self that feels and is aware (consciousness) is never in danger. It is always safe. Except during brief spurts of adrenaline that occur during times of imminent danger to the body vehicle, the heart of a human feels the sensations associated with safety.

But even during emergencies, an emotionally mature human knows that his real self, his feeling/awareness, is ultimately safe. As it says in the Bhagavad Gita with regard to that consciousness, “No fire can burn it, no water can drown it, no wind can dry it, no rope can hang it.”

Of course, a worried person *can* learn to ignore his sensory awareness of his own life force and vibrancy. Or he can override that awareness with mind games: worries. If he has powerful mental focus and is highly intelligent (like most people with Parkinson’s disease), he can learn to dissociate his consciousness from his internal sensations of vibrancy.

In an emotionally healthy person, this wealth of sensations automatically accompanies the insertion of consciousness into a body. But a brilliant person who is afraid of feeling pain can learn to increasingly dissociate from his own vibrancy at the least hint of emotional or physical unpleasantness.

However, even if he does this to avoid *dealing* with physical or emotional pain, the pains will have actually occurred. Even though he pretends not to feel his pains by dissociating from them, these unprocessed pains will still be present in his body and in his subconscious. The dissociated pains can even fester and grow inside his body and his subconscious, throwing out spreading roots of physical pain, anxiety and negativity.

If a person is always on the lookout for pain so that he can guard against it, his mind perceives himself as being always at risk. He can never feel safe.

There is no way to make like safe. The body can be hurt in an infinite number of ways. There is no way to guard against all potential injuries. The body is a temporary vehicle. It *is* going to die. And, we live in an ever-changing world. If we are alive and nursing likes and dislikes, preferences and opinions, there *will be* physical and emotional pains.

An emotionally mature person identifies with his internal sense of vibrancy

An emotionally mature person deals with this by not identifying himself as his body and by being careful in his cultivation of preferences.

An emotionally mature person identifies himself with the heart feeling inside.

He does take good care of his body, just as he would care for a good tool or a well-loved pet. But he knows that he is not the ever-changing body. He recognizes that what he really is is the feeling inside himself – a feeling that most people can feel and for which we have no word in

English, a feeling that I am going to have to try and define before this chapter is done. For now, let me call it vibrancy.¹

This vibrancy is never at risk. One can temporarily lose sight of his internal vibrancy or even forget that it's right there, vibrating inside. But as soon as one remembers that he is safe, he can feel that vibrancy. Reciprocally, if he feels that vibrancy, he feels safe. Either way, when he feels safe or he feels his internal sensations of vibrancy, he perceives that he *is*: that vibrancy is what he *is*. No matter what is happening to his body, those vibrations – his real self – that he feels inside himself are still there.

Many PDers understand the above in an academic sort of way. Many PDers are very well versed in spiritual treatises and religious theory. However, many of them still don't seem to understand that the vibrations and "Divine Sparks" that are discussed in their books and theories are things that every person can *feel* – they are physical phenomena that can be tangibly felt inside the body.

PDers, due to dissociation, often have a sense of themselves existing outside the body. Except for pains, which they associate with the body, they tend to live in their heads or outside of their own flesh, observing their flesh as if an outsider. This is not mentally or emotionally healthy. A human is not supposed to live as if he exists outside of himself, or think of himself as the reflection that he sees in a mirror. A healthy human is *inside* his body. Unless he is dead or dying, he is not outside his body, observing himself.

Arguments: I DO feel safe, I'm NOT afraid

Many PDers whose symptoms worsen in times of stress have argued vehemently with me that they do *not* feel *unsafe*. They are certain that the problem of dissociation or not feeling safe does not apply to them. They insist that *stress*, not "fear," is the problem. This is semantics.

They readily admit that they tremor more, or their toes curl under, or maybe they become more slow or rigid in certain settings such as when they anticipate problems, or when they are out in public, when they are being put on the spot, etc. But they often insist, "I feel perfectly *safe* during those times. I certainly don't feel unsafe just because I'm out in public, even though it makes me tremor and tense up. I'm worse when I'm in *stressful* situations. But I have no fear."

¹ I *love* English. It is my own, my native tongue. I am in no way disappointed in the English language for this lack: other languages besides English lack a specific word for this feeling. I discussed this semantics issue with a famous Tibetan thangka (sometimes spelled "tanka") painter. I had been admiring his paintings at an art show, and commented to him on the "feeling" in his artwork. I instinctively gestured with my upward facing hands moving gently in circles held up at the level of my chest, while inhaling gently.

He volunteered that he loved this word "feeling," and as he said the word, he moved his hands the same way that I had moved mine. He had clearly come to know that this word, when combined with the hand gesture, meant a specific, very good, sensation in the chest. He then said that, in Tibetan, there was no word to mean this "feeling," and again, as he said the word feeling, he gestured with his hands.

I was surprised that there was no word for this in Tibetan, and I asked again to make sure. He was adamant. So I asked if there was no word comparable to the "*Kai Xin*" phrase in Chinese. He replied that the Tibetan phrase *Ning Jei* was somewhat similar, but it was not really the same. *Ning Jei* specifically refers to the feeling of compassion that flows *out* from the heart towards others. I asked him about the Tibetan word for happy, and he said that there were many, but none of them conveyed the same meaning that he had come to understand for "feeling" and he did the hand gesture again as he said the word. I found it delightful that this artist, who had only come to America in the 1990s, had, in response to his art, so frequently heard the word "feeling," combined with the hand gesture, that this difficult to define but oh-so-glorious sensation was his *primary* understanding of the word.

If I ask what the underlying emotion behind stress actually is, and suggest that stress is a state of mild fear they might answer, “No. I don’t think anyone’s going to hurt me. I don’t feel unsafe.”

If then I ask if their tremor, toe curl, slowness or rigidity *decreases* at certain times, they assure me that it does: when they are doing specific activities where they feel absolute comfort such as after they’ve gotten their pajamas on; or when doing the laundry; or some other highly specific task or timing.

So then I have to ask, “Is it possible that what you are calling “comfort” might also be the same as “feeling safe?”

To which they often reply, “But I don’t ever feel unsafe! I never think that I’m going to get hurt.”

We can go round and round in this discussion, in which the PDer says that he never feels at risk, and yet he admits that in situations where he is not utterly comfortable, his symptoms rapidly worsen.

The real problem here is semantics. The PDer may be devoted to living in such a way as to be constantly assessing and avoiding risk. Therefore, he is certain that he is always staying safe – so, logically speaking, he has nothing specific to fear.

When I say that the PDer doesn’t feel safe, the PDer may have a very hard time seeing himself in this light, because his *thoughts* are devoted to keeping himself safe and steady at all times, or even assuring himself that he is doing everything he needs to do to be safe.

However, his *body* speaks the truth. The reason he tremors, becomes rigid or has curling toes in certain situations *is* because he feels some undercurrent of risk in those situations.

Sure, he has dealt with the risk so that he doesn’t have to notice that he feels unsafe. The *way* he has dealt with the risk is to dissociate from his heart. Because he has dissociated, he feels safe from the pain of any physical or emotional event.

So when I suggest that he doesn’t feel safe, he vehemently denies it.

He is preventively girding himself against something unsafe. Whether he calls it stress or nerves or anxiety or whatever, the underlying emotion is fear. The underlying feeling that is causing him to shutter his heart and play dead is a feeling that he is not safe.

Feeling unsafe is OK

Feeling *not* safe once in a while is perfectly healthy. Healthy people are able to feel at risk at appropriate times. However, unlike PDers, a healthy person will deal with being unsafe by switching into sympathetic mode for the duration of the unsafe period.

The PDer deals with the unsafe situation by switching over into dissociated mode. What makes it even worse is that the brain learns by repetition. The brain gets better and better at dragging more and more of the body into “nearly dead and shaking” the more often it practices dissociation. The brain-body relationship learns via habit. Dealing with feeling unsafe by dissociating from the heart instead of rising to the occasion with adrenaline is unhealthy.

An example: “I like to pretend I’m a corpse – but I don’t ever feel unsafe...”

Many PDers don’t like to admit to feeling unsafe because they are *so* determined to keep danger at bay via never making mistakes or by not letting themselves feel pain.

One PDer who was adamant that she *never* felt unsafe also admitted that her trembling and toe curling got much worse when “there are people around” or if something was expected of her. But she told me that she never felt like other people were going to harm her or anything. As many PDers do, she tried to convince me that she didn’t feel *unsafe* around groups of people, even though her symptoms all worsened if people were around.

She went on to say that “Sometimes I do feel *really* safe: when I’m practicing the Corpse Pose (lying flat on the floor, whole body limp, as if dead) in yoga class. When I do the Corpse Pose, I feel *completely* safe. So I think you’re wrong. I *can* feel safe.”

I have to admit, I laughed out loud. “The safest you ever feel is when you’re practicing the corpse pose?”

“Yes. I feel fine when I’m doing the Corpse Pose. I can relax then. I pretend I’m dead, and my tremor stops completely.”

“You feel the safest when you pretend to be dead?”

“Yes, I love that.”

“Why do you love that?”

“I feel so safe. I can relax completely.”

“You can relax completely when you’re pretending to be dead?”

“Yes. So you see, you are wrong; I don’t feel *unsafe*. I can feel very safe.”

“And can you relax *all* the time? You don’t look very relaxed now.” (She was trembling badly.)

“No. That’s why I like doing the Corpse Pose: so I *can* relax.”

“So when you *aren’t* doing the Corpse Pose, you’re not as relaxed. Is that because you don’t feel as safe when you aren’t pretending to be dead?”

“I always feel safe. I feel even *more* safe when I’m lying down pretending to be dead.”

I tried one more time: “If you actually felt safe, you would be able to relax when you’re *not* doing the Corpse Pose. I suspect the reason you dissociate, also known as pretending to be dead, is the same reason that you really like to do the Corpse Pose: so that you *can* pretend to feel safe – because otherwise, if you aren’t dissociated or pretending to be a corpse, you *don’t* feel safe.”

I continued, “If you truly felt safe, you wouldn’t need these over-the-top coping tricks to enable you to feel safe. You’d feel safe all the time except for when you were in imminent danger.”

We went on like this for half an hour. She never did understand what I meant: that she was using coping mechanisms because, bottom line, she did not feel safe. Her coping mechanisms made her feel less at risk. Pretending to be dead allowed her to relax a bit. But no matter what, she was not *inherently* safe. However, she refused to say that she felt unsafe. Her words were rambling, but the intent was clear: she wanted to assure me that feeling unsafe was not good. She was good. Ergo, she did not feel unsafe.

One long look into her eyes told me that she did not feel safe and had not felt safe in a long, long time.

A wrong definition of spousal support

As I spoke with her, her husband frequently snapped at her, “I am *always* telling you that you need to relax!” or “You just don’t get it! You need to have more faith!” He gave me a look of commiseration, “I’ve told her a thousand times that she needs to trust God.”

I observed that he criticized her constantly, stabbing her relentlessly with his superiority and spirituality. He attacked her with a steady flow of “I told you this before; I’m always telling

you; what *you* need to do is (this, that, and the other).” Her tremor increased during his harangues. Each time after “correcting “her, he smiled over at me. He was expecting praise from me, I think.

I could see where some of her fear was coming from. Her husband, a very well meaning and loving know-it-all, had no idea that, from where I was sitting, it seemed as if he was brow-beating her. I’m sure he didn’t see it that way.

Possibly, if she was a brazenly confident woman, she could have borne his “loving advice” without flinching. Or better yet, with a swift rebuttal and a breezy “Have your lawyer call my lawyer.” Instead, each time he accused her of ‘not feeling safe,’ she seemed to freeze up inside just a little bit more while bleating in reply, “I know I am safe, I know that God is watching over me.”

Feeling safe from criticism

As I watched her tremor come and go in time with his interjections and “helpful” accusations, I could see that he was oblivious to her fear of criticism.

PDers are often terrified of criticism. They are sometimes more afraid of criticism than of actual, physical danger. They can respond to actual, physical danger by switching into sympathetic mode and doing whatever needs to be done.

They are often far more brutalized by small criticisms or seemingly insignificant slights, against which they may be utterly unable to defend themselves, than they are by physical injury from sports or accident.

Very often, they have a skill set for dealing with physical pain – so long as the pain was not acquired during an event that was emotionally risky. But they may only have two choices for dealing with imagined failure or the criticisms of others. One is to dissociate (feel no pain). The other is to launch into sympathetic mode: fight or flight. Because they have learned that sympathetic mode can be associated with losing temper, and is therefore emotionally risky, they are very often afraid of allowing themselves to slide into sympathetic mode – the only mode that might allow them to defend themselves against criticism.

Sympathetic mode feels risky for some PDers. PDers may let themselves go into sympathetic mode for a true emergency. They may be able to slip into sympathetic mode in response to an imaginary emergency, one induced in order to create a sense of urgency so that they can move when they have become stuck or rigid. But many of them learned long ago, often in early childhood, that stillness and quiet held their best chance for safety for most situations, especially anything involving emotions.

When their *emotions* are at risk is when they are most likely to slide into dissociation. They often are severely immature with regard to their ability to feel or deal with their own emotions. In response to emotional risk to self, they revert into dissociation. Hence, their inability to deal maturely with criticism.

Many PDers are terrified by the thought of potential criticism from others. They are also harried by constant self-criticisms. PDers must know that when I speak about *feeling safe*, I am talking about feeling safe in the heart despite any and all criticisms and emotional pains, as well as from all physical dangers.

Another aside: the “bad spouse” excuse

Mentioning the above scenario with the holier-than-everyone-else spouse reminds me of another spousal complication.

Many PDerers have complained to me that the reason they can't feel safe is that their spouses or family aren't perfect. While reporting to me that they had a bad weekend, movement-wise, they will explain it by saying their spouses or family didn't let them do exactly what they wanted to do that weekend, or the spouse or family was critical of them that weekend. So of course, they were trembling and rigid all weekend, and it's the spouse/family member's fault. "I couldn't have any dopamine this weekend because we had to spend all day Saturday with my wife's parents instead of doing what *I* wanted to do," is a not uncommon sort of whine.

Because the world did not wrap itself inside out to mollify their desires, they tell me that were unable to feel enough joy to release dopamine and thus feel safe. These people have it very, very backwards. They have the emotional perspective of a child.

Immature expectations

A child thinks that the world must please him so that he can be happy. An emotionally mature adult knows that he must be happy so that he can be pleased with the world.¹

An adult knows that feeling safe allows a person to relax. Being relaxed allows a person to deal easily and appreciatively with difficult situations.

PDerers are often extremely intelligent and are often deeply spiritual or philosophical. However, they can be extremely immature when it comes to knowing how to deal with emotional pain or emotional disappointments.

A PDer, like any adult, needs to learn how to feel safe in his heart *in spite of* external conditions. He should feel safe *whether or not* the spouse and/or family are being supportive or abusive, present or absent.

We feel safe because our hearts are at peace, not because circumstances warrant it. And if we lose track of that safe feeling, we can reclaim it by re-establishing a remembered calm heart feeling.

Feeling safe is calmness, steadiness, in the slowly or rapidly beating heart. It should only be disturbed in times of genuine danger to life and limb. Feeling safe has nothing to do with whether or not a person is having his way or having fun.²

Truly abusive: the exception to the rule

Then again, if a spouse is truly abusive, the PDer, or any person, might be better off leaving. Many years before I ever started this project, I knew one very, very early stage PDer who healed herself. Her symptoms gradually went away when she changed her life. She left her husband, becoming a single mom with two grade-school kids. She moved away from our town, settled into a new city, got a job, joined a church, and started doing daily Qi Gong (Chinese energy-moving exercises). While doing her Qi Gong, she realized that the energy in one foot was badly blocked. She worked everyday on feeling energy moving through that foot. She even spent

¹ "No one can make you unhappy if you are determined to be happy. No one can make you happy if you are determined to be unhappy." This is an *ancient* truism.

² One of my favorite prayers is "Change no circumstance of my life. Change me." From *God Alone: Life and Letters of a Saint*, the writings of Sri Gyanamata, published by Self-Realization Fellowship, 1984.

some time holding her own foot until, after a few months, the energy moved freely and correctly through it. (She was also an acupuncturist, and had working knowledge of the correct channel flow patterns.) She did notice that, after she got Qi moving through her foot, many of her physical problems, including cold hands and feet, went away.

I didn't know that she'd taken up Qi Gong when I decided to look her up about ten years after she moved away, after I had started this project.

I found her contact information through a friend of a friend, and we got together. After a little chit-chat, she volunteered why she'd left her husband. I'll paraphrase: "I was dying, being in that marriage. He was never cruel to me. He was brilliant. His academic life and the university were so important to him. It was like he never knew we were there, the kids and I. He probably was grateful when we moved out – if he even noticed. The children had been a distraction from his academic pursuits. He had no interest in the kids or in me – we interfered with his work. He had told me he didn't want kids, but I thought if we had kids he would change his mind. I was wrong. After ten years in that marriage, I was dying inside.

One morning, I woke up and I realized that, if I didn't leave, I was going to get really sick or die. I decided that it was up to me to make my life be the life I wanted to live. I have to shape my own life, every minute of it. I had been young and emotionally dependent when I married. I realized I had to outgrow that and become an adult. I had to take risks. I had to not be afraid of being alone, of making mistakes, of getting hurt. I realized that I had to grow up and take risks so that I could really live my life and set a good example for my two sons. It was the hardest and the best thing I ever did for myself – for all of us."

I started asking her about some of the health problems she'd had ten years earlier by inquiring if she'd ever found a good treatment for her Reynaud's syndrome (severe cold in the hands and feet that doesn't warm back up in a normal amount of time. Reynaud's is a not uncommon symptom of Parkinson's disease.) I never told her that I had paid sharp attention to some of her other physical symptoms during the few years that our kids had been in school together. She'd only mentioned her Reynaud's syndrome and her increasing stiffness. I had observed over several years how certain muscles on one side of her face moved less and less even though her eyes had remained sparkly. Also, one leg had been starting to drag and her arm on the same side barely swung when she walked. Her head had been beginning to pull forward and a hoarseness had been creeping into her voice.

When I saw her again ten years after she'd moved away, I could see that all these symptoms were gone. I didn't say so, but I had intentionally sought her out to see if she'd developed full-blown Parkinson's disease. She hadn't. She looked and moved like a very different person: a confident, relaxed, supple person.

She spoke a little bit about how returning to her Quaker roots had helped her feel safe and calm in her heart, no matter what happened to her.

Based on many observed symptoms, she'd very likely been en route to Parkinson's. Not knowing this, she had discovered and healed an old foot injury and remade her entire outlook on life so that she felt safe and calm no matter what happened to her. She'd done the two things that PDers must do in order to recover! And all of her PD-like symptoms were gone.

I am glad to share her story in this book. It makes two points. One, leaving an abusive spouse can be the right thing to do. Two, a person can recover from Parkinson's by himself, with

no outside help, by restoring normal channel Qi flow through his injured areas and by learning to feel safe and getting back in touch with his own heart.¹

Again, in the case of non-safety due to genuine mental or physical abuse, a person should leave.

But if abuse is not occurring and a person stills feels *unsafe* in response to his spouse or family, it's his own fault, not the fault of the spouse or family. *As long as a person is blaming others for his inability to feel safe, he will never feel safe.*

Many styles, one motive

The behavioral styles that PDers who get stuck in partial recovery have chosen in order to create the illusion of being safe in spite of the fact that, at heart, they do *not* feel safe, can vary. The *reason* that they instituted their behaviors does not vary. They may be control freaks or non-argumentative; self-contained or extremely desperate to serve others; always correct (whether by withering argument, gentle coercion, or stony silence); highly responsible; ever punctual; strong or disdaining of physical strength.

But at the heart of the matter is a striking similarity: they would rather be numb than experience the pain of failure, criticism, weakness, or losing self-control: the pain of being emotionally wounded.

If a person is able to temporarily feel safe *because* of some protective thing he is doing, it means he doesn't feel truly safe.

Oppositely, if he feels safe in his heart no matter what happens to him or what people think, then he truly feels safe.

If he behaves one way with some people and another way with others, he doesn't feel safe.

Oppositely, if he is the same all the time, and speaks his truth from the heart, whenever the small still voice within tells him to do so, whether or not that truth puts him at risk, he does feel safe.

PDers who feel safe

Some people with Parkinson's disease do feel truly safe. These people recover easily when their foot injury heals.¹

¹ I am constantly contacted by people who have read my writings and who are looking for a referral for an acupuncturist in their area, even though our website states very clearly that we do not give referrals. Also, I have tried to make the point in all my writing that acupuncture is not key for treating Parkinson's disease. Most acupuncturists are not familiar with my work, nor are they familiar with Tui Na – nor do they want to be. Most acupuncturists who trained in China do not even believe in channel theory. The Tui Na treatments that are used for healing the foot can be done by anyone – a friend or neighbor who is able to sit still and provide comfort while minding his own emotional business. The PDer himself must do the psychological work. This book has chapters describing treatment techniques. This book was designed as a *self-help course* as much as a book of theory and a chronology of our Little Project.

I'm making this point here, and I'll make it again elsewhere in the book. I am being redundant on this point because it is something that readers consistently seem to miss. They want to find someone to fix the problem. They often don't want to recognize that they can fix the problem themselves.

Hopefully, by including this case study, the point will be better made.

But many of our patients with Parkinson's, the ones that have gotten stuck in partial recovery, have never really learned how to feel safe. If we treat them and their feet heal but PD-like problems still come and go, they should look carefully: they will see that their problem times occur when they do not feel safe. As they swing back and forth between healthy movement and fear-based movement (tremor and rigidity), and as these swings become more severe, they will notice that the movement problems arise as rapidly as thought: they occur when they do not feel safe.

Summary of feeling safe

Feeling safe comes from inside. It is a sensation in the heart. It has very little to do with external conditions of safety.

The basic emotional position of a healthy person is "I am safe and my heart is at peace. Except during those short moments when I am in bodily danger, I am safe. As soon as danger is over, I am safe again."

The PDer may need to add to the above, "I do not need to play dead to my emotions in order to be safe."

Finally, by way of proof, I can state that even those PDers who have insisted that they *do* feel safe, that they *aren't* worried, that they are in control, self-contained, confident, strong, in-charge, etc., etc., etc., and that I am wrong, nevertheless *all* say the same sort of thing when they finally snap out of their long-term reliance on self-induced heart dissociation: something along the lines of "Ha ha! I just realized that I'm OK after all, that there's nothing to be worried about after all. I just needed to stop trying so hard to be safe from everything. I just needed to relax!"

Almost always, they add something to the effect of "Why didn't you just tell me to relax?" or "Why didn't you tell me to stop doing my self-control thing?" or "Oh! It's a physical sensation! Why didn't you tell me?"

You can imagine my bafflement and frustration at these sincere outpourings. And yet, it is also glorious to have a suddenly recovered PDer try to explain to me, using the very words I'd been using for months or years, how simple it is to feel safe. They tend to go on for quite a while, telling me the very things that I have told them a dozen or more times. Very often, they use the exact same words that I have used, with no idea that these were the same phrases they had failed to understand prior to making the switch.

I sometimes point out that I have said these same words in the past and failed to get my point across. I ask what I might have said differently, so that they might have understood me sooner. When they consider it in this light, they always conclude that there is nothing I could have said to make them understand. They had to *feel* it for themselves. When they were finally willing to surrender, to admit that they were OK, that they were safe, then they were suddenly

¹ One PDer suggested that the reason professional musicians heal so quickly is a person *must* feel safe if he's going to throw caution to the wind and embark on a livelihood as unpredictable as that of a musician. His idea was that only someone who is confident that the ravens are going to feed him can decide to damn the odds and commit to a lifetime of musicianship. Hard to say for certain, of course. Could be another one of those chicken and egg situations. Or reciprocity. Maybe a passion for music keeps the heart and brain of a PDer so attuned that calm heart, that is to say, fearlessness, is the default condition for everything but that one darned foot injury. Or maybe it's the other way 'round: the fearlessness in the heart keeps the music flowing. Doesn't matter.

fine. And until they were willing to admit that they were safe, there was *nothing* I could have said that they could have understood.

This chapter will end here, but the next chapter takes up where this one left off. The only reason for the chapter break is that there is too much material to fit into one chapter: the subject matter is continuous from this chapter to the next.



CHAPTER FORTY-THREE

SEMANTICS: PART III

Defining surrender

Recovered PDers often use the word surrender to explain the mental shift that occurred during recovery. For example, “I finally realized that I’d been fighting something my whole life. It felt so good to just surrender to the universe.”

And yet, many people who are stuck in partial recovery usually have a powerful emotional dread of the word surrender.

To start with, surrender does *not* mean die.

Surrender means stop fighting: send up the white flag; stop being so tough and alone; ask for help; stop pretending that you are the only thing keeping your family fed and sheltered; stop thinking that, if you let down your guard, you and your loved ones will be necessarily be hurt.

The next sentence may seem a bit harsh, but I assure you, I am speaking to many PDers when I say: stop thinking that your stone-hard sense of responsibility is what has made you successful in life and that people who are different from you are losers. In the big picture, if you are so afraid that you constantly tremor in fear and you are so out of touch with your own body that you can’t relax and restore yourself at will to an awareness of the vibrations of joy within yourself, how successful are you, really?¹

As I mentioned earlier, the first time I attended the local Parkinson’s support group I was stunned to my core when a sweet, eighty-plus year old woman shared this experience (I paraphrase): “Last week I was at the grocery and I couldn’t reach something on the top shelf. I hated to do it, but I asked a young man to help me. He was so pleasant, he seemed to take pleasure in helping me!”

The other the PDers in the group looked at her aghast, as if wondering how she could have so far forgotten herself as to ask for help.

Surrender. It means admitting that you don’t run the universe.

Many PDers, in response to the above sentence, have said, “Maybe I don’t run the universe, but I’m responsible for running my part of it to the best of my abilities. I should not make errors or let people down if I can help it.”

¹ Many PDers are captains of industry and highly honored powerhouses. But if they are not contented, how successful are they, really? All the honors and money that they have accumulated will stay behind when they die: the consciousness of imperfection and fear will accompany them. If a PDer does not overcome his inability to be relaxed and content in this lifetime, he will have to continue working on these problems after death and in his next life. I often ask PDers how many lifetimes they intend to have Parkinson’s before they decide to surrender their stoicism and end their dissociation from their own hearts. Of course, they always say that they never want to have Parkinson’s again. I ask them what they have been changing in their approach to life so that they won’t end up with the exact same problems next time. Anyone who does the same thing over and over and expects to get different results if he does it yet again is *not* behaving rationally. If one’s attitude has contributed to Parkinson’s, one will have to change his attitude if he hopes in future to not have the mindset that causes Parkinson’s.

This sounds somewhat reasonable on the surface, but the way that some PDers apply it is in the extreme, dissociated from their ability to know what sort of errors are reasonable or inevitable. Because they cannot feel their own heart, they cannot know their true conscience. Therefore, many of them try to be strangely perfect. At some point it becomes sheer vanity – and doomed to failure.

This tendency is so marked that doctors have, for more than a century, tried to define the Parkinson's personality. I will make an attempt at describing that "something" that makes PDers seem so different from other people.

The Parkinson's Personality

For many PDers, their personality is defined by an attempt to use a combination of adrenaline and a numb heart in order to make the world as safe as possible for themselves and for as many people as possible according to the guidelines of their imperfect intellect, while having no idea what their hearts are trying to telling them with regard to their own feelings.

Humility: a hallmark of surrender

Can anyone of us prevent an earthquake? Prevent the death of a loved one? Reverse the tides? Prevent an economic recession? We can do our best, but we can't create a world that will be free from pain to the ego or body: a "safe" world.

Our responsibility is to use our faculties to do our best. We are not supposed to be super-humans who can "make everything all right" or keep ourselves or anyone else "safe" from pain.

We are supposed to love ourselves, and others, and do what's right to the best of our abilities. But we aren't supposed to be unreasonably perfect or knock ourselves out when we turn out to be no better, in the big picture, than anyone else. In fact, if the goal is to be virtuous, one of the greatest virtues is humility.

A humble man is perpetually surrendering to the will of the universe. Humility does its little job to the best of its ability and then leaves the rest to play itself out.

A humble man doesn't hurt himself by lying awake at night thinking of retorts, or hurt himself by running a constant critique of himself or of the rest of humanity in its head. Humility is always grateful.

I am not talking about being grateful or humble to *others*, although that is admirable. Many PDers come across as *very* humble and appreciative towards *others*.

I am talking about the way a PDer has to learn how to treat himself – the person he's become dissociated from. The PDer needs to be grateful and humble towards himself.

Humility can mean being grateful for having a body and prove it by taking the time to feel the body and feel oneself being inside his body. Humility can mean surrendering in the sense of *admitting* that various forces have wreaked havoc on the body and emotions, *feeling* those pains, and humbly *helping* the body by holding the pain-filled areas with internal vibrations of love and support when it asks for solace.

PDer, take a load off; give up the fight. You may discover that you have been spending most of your energy fighting enemies that you are creating in your own mind. Surrender. Stop the fighting. You may well find that most of your enemies were imaginary.

Surrender. It doesn't mean die. It means stop the fighting. Be at peace. Enjoy the physical sensation of calm that your heart feels when you remember that you are safe.

Defining calm

The discussion of feeling safe can be related back to the heart-nerve that were discussed in chapter xxx: the feeling of safety is associated with a relative calmness in heart rate variation.

“Calm heart” means “*regular* heart rate variations.” Calm means orderly: not chaotic. A rapid heart rate can still be calm. For example, a person who is having a glorious time leaping to catch Frisbee or gaily galloping down the beach at full tilt might have an *elevated* heart rate that is nevertheless calm and orderly.

Some people want to misunderstand the phrase “calm heart” to mean *slow* heart rate. This is incorrect.

If a person feels safe, his heart rate variations will not be chaotic. Even if the heart beat is going fast to accommodate increased oxygen needs, the “feeling safe” heart wave variations can be patterned in such a way that they are regular and somewhat steady: steady enough that the brain and heart are entrained and working together. “Calm heart” does not mean minimally active heart – it means heart without chaos, without fear.¹

A minimally active heart occurs during the pre-death state of automatic dissociation. In this condition, heart rate is minimized. However, this is not the same thing as a *calm* heart, one that is joyfully integrated with the mind and sensory awareness. A dissociated heart is one that is detaching from its ability to perceive sensations: preparing to die.

The minimal activity in the heart of a person who is dissociating is *not* the same as the minimal activity in the heart of a person whose sense of feeling has become vast through meditation and practicing compassion. The former has a slow heart rate because he is nearly dead or pretending to be nearly dead. The latter has a slow heart rate because he is living via the neutralized vibrations of energy that he *feels*, instead of sustaining himself with oxygen. There's a substantial difference between the two. Many PDers have deluded themselves on this point.

Defining “neutralize”

Most PDers, and many modern English speakers, have come to think that the word neutralize means “destroy,” as in, neutralize (kill) the enemy. Actually, that usage is a euphemism. Neutral means, more exactly, not taking sides: impartial.

For example, during World War II, the Swiss remained neutral. This does not mean that the Swiss were dead. It means that they tried to not get caught up in the madness.²

¹ A frequent admonition of Paramahansa Yogananda is “Be calmly active and actively calm.” In other words, even when active, the heart should be calm, not chaotic. And if, during the rush of activity, you temporarily lose your sense of calm, then actively work at feeling calm and safe again.

² I know, I know. Historians are making the case that the Swiss were not in fact neutral. I merely use this familiar political stance as an example. For that matter, historians' jobs are to *not* be neutral. Their job is to *interpret* the past so that we can learn from it. But to be most effective, they must integrate some illusion of neutrality into their work. This is what makes the job of the historian an art and not just a listing of dates and places. Historians must present their cases as if they are based on truths when, in fact, all perceptions *not* rooted in absolute Truth are delusory. And by bringing up the subject of history, I get to segue into a tangent that gets to the very root of our ability to be neutral: perspective.

As we say in physics, all observations vary depending on the location and velocity of the observer. Only when one perceives all things from the perspective of infinity can he perceive the Truth. Or as they say in Chaos

Perceiving a sensation with a neutral attitude allows it to be perceived as neither good nor bad, but simply as what it is. When, in the previous chapter, I said that pain becomes neutralized into mere sensation when one simultaneously feels the pain *and* the indwelling vibrant feeling, I did not mean that the pain is eliminated. I meant that the pain becomes mere sensation, without being tinted by fear, by notions of good or bad or a desire that it increase or decrease.

Sensory neutrality

When a person beholds a beautiful sunrise unfold, as he feels the sensations of color vibrating in his eyes and resonating in his chest, he will perceive it most fully if his perceptions are neutral.

Oppositely, if he analyses the sunrise to decide whether it is as good or bad as yesterday's sunrise, he's actually missed the whole experience. If he mentally says, "Nice work God, but I think last Tuesday's sunrise, featuring mauve and less canary yellow, was more effective," then he hasn't really *experienced* the sunrise – he has only judged it.

A person gazing at the sunrise should be able to feel neutral towards the sunrise. He can accept the sunrise for what it is: a sunrise. He *feels* the sunrise by noticing how his heart's vibrations are influenced by the colors and shapes. He does not taint the experience by deeming it "good" or "bad."

When one stops applying the murk of personal opinion and preference to an event, he can perceive the event as it truly is: he is a neutral observer. This is also known by another term: relaxation.

Oppositely, the evening news is designed to make a person feel strong opinions: *not* feel neutral. The TV viewer may feel aghast at the images of violence that are shown. He may feel angry or opinionated. Unless he is deeply wise, he cannot maintain his neutrality and relaxation as he is subjected to the over-dramatized events of the day.

One PDer told me that, in her teen years, after attending several music concerts with her friend, her friend said, "Why don't you ever just sit back and enjoy the music? You're always looking around at everyone, trying to see everything that's going on." The PDer continued, "When she said that, I had no idea what she was talking about."

What the friend was trying to say was, "Why don't you just sit back and enjoy the sensations of the music? Let the neutral – neither good nor bad, utterly unanalyzed – waves of music wash over you."

theory, in order to be able to know exactly what is happening, what has happened, and what will happen, you need to know everything, and you need to start your measurements at the moment all actions were initiated.

For example, to *truly* predict or understand the weather, we need to measure vibrations starting at the moment the universe began. If we have that Cosmic perspective, we can know what the weather will be and – getting back to the point – we can be emotionally neutral about what happens. When we know all the past, we can see all current events as repercussions of cause and effect. If we step back one step further, perceiving the love that *initiated* the universe, we can even perceive love amidst the fluctuations of seeming light and dark, pain or pleasure. We can see all events as neutral demonstrations of the law of cause and effect, originated and sustained by Love and moderated by Forgiveness.

We are never victims of randomness: we live in a universe that is ultimately fair. We are safe. We can rest assured that the exquisite perfection and balance in the universe is in force even when winds of change tear at the ever-shifting, delusory flesh. We can stand, emotionally unmoved, amidst the crash of breaking worlds: vibrating in time with the universe; feeling the song of every glorious atom. We can feel everything, feel the Love behind everything, and be joyously, truthfully *neutral* (without prejudice, without personal preferences, likes and dislikes).

Move to the music, housefly

Music is particularly glorious if one doesn't try to analyze or judge it. The other evening, while proof-reading the previous chapter in my living room, some friends were across the room playing a Hayden piece for violin, viola, and cello. The music was written in waltz time (oom pah pah, oom pah pah).

A housefly was walking around on the table next to me. For the duration of the musical piece, the fly paused at every downbeat. Pause skitter skitter, pause skitter skitter, pause skitter skitter, pause skitter skitter – in perfect time to the music. The music was vibrating through the housefly, and the fly was *feeling* the music. He was experiencing the music. His movements were being influenced by his vibratory awareness, his *feeling*, of the music. He was *not thinking* about the music; he was feeling it. His feeling of the music was emotion-neutral and vibrantly alive.

This fly brought to mind a particular problem. When I've told partially-recovered PDers that they need to learn to *feel* the Something Within, as it is called in gospel music, or the vibrations in the chest, or the expansion of the chest, they have often complained that I expect them to attain to sainthood. They bitterly point out that they shouldn't have to be a saint in order to recover from Parkinson's; *lots* of people aren't saints, but they don't all develop Parkinson's disease.

Some partially-recovered PDers have gone on to make the point that they know lots of people who are real jerks, who are far less spiritual than they are, who don't have Parkinson's, so how dare I make a connection between PD and the inability to feel "something within."

When I assure my patients that they don't need to be the least bit perfect or saintly in order to feel, or to feel safe, they shoot me a look that says it all: they remain convinced that I am asking them to aspire to something rare and nearly unattainable: a Gandhi-esque level of fearlessness and God attunement.

I thought of this common complaint as I watched the fly moving in time with the music, feeling the music. The fly could be another example for them: a PDer doesn't need to turn into a bodhisattva or prophet in order to recover; he just needs to experience neutral feelings – like a housefly.

Neutralizing consciousness

The great sage Patanjali (circa 200 BC) wrote in his Yoga Sutras (spiritual insights) that, in meditation, the waves of consciousness should be neutralized. He didn't mean that people should pass out and become unconscious – a common western misunderstanding. He meant that the waves of consciousness must be fully perceived, but with impartiality rather than opinions and analysis.

In other words, turn off The Internal Judgment Machine and feel what you can feel. Be human; come back to life. Be as safe as a mouse can be; be as perceptive of vibration as a housefly.

The neutrality of babies

When a tiny baby sucks his mother's milk, the baby's experience is neutral. The baby is not thinking about whether or not the milk is richer or tangy-er than last time. The baby is not

worried about whether or not the supply will run out. His isn't wondering as to his mother's motives.

The baby experiences the feeling of the milk flowing into his mouth, and the taste of the milk running across his tongue, and the smell of the mother's skin, and the feeling of his own tummy filling with milk, and the feeling of pure being that he senses inside the casing of his body. What joy!

A tiny baby doesn't take sides while nursing. He experiences his life without judging it: he is neutral; he is content; he is safe. When he isn't safe or when he is in pain, he cries or screams. When his pain ceases and his needs have been met, he is safe again. Safe is the fallback condition. Neutrality (sometimes known as gratitude) is the stance that lets one feel safe.

Defining gratitude

As mentioned in an earlier chapter, many PDerers have told us that the "breakthrough" occurred when they realized how much they had to be grateful for.

Because of this insight, we often advised PDerers to do a gratitude exercise that consists of feeling grateful for five things every night before going to bed.

We finally came to realize that it wasn't the gratitude, *per se*, that ended the dissociation. When we asked carefully about what the person had been grateful for in that moment when the brain suddenly sprang back into normal function and the Parkinson's permanently ceased, as if turned off by a switch (if the foot injury had already started to heal), we found that it wasn't due to the act of mentally *listing* things for which they were grateful.

It was when people stuck in partial recovery suddenly were able to *feel* grateful (a sensation, a heart feeling) – and they felt grateful for *everything* – that they suddenly felt profoundly safe. When they managed to change the drift of being grateful for the things they *liked* into an overarching gratitude for everything, even things that had seemed terrible or unpleasant at the time, they simultaneously felt safe.

Any immature person can be grateful for getting what he wants. Emotional maturity is being grateful for whatever happens.

How does this relate to feeling safe? Logically speaking, if *everything* that happens is OK, then there's nothing to be afraid of. If there's nothing to be afraid of, we're safe. Period.

As soon as PDerers remembered that everything they'd hated or feared, even pain, had been helpful, had been a learning experience, had been fair and reasonable, maybe even *deserved* (in some impossible to understand, karmic sort of way), or had turned out OK in the end, they suddenly had to admit that everything, even the Parkinson's, was obviously going to turn out to have been OK, no matter how it turned out. And the tremor and the rigidity and the other mood-based symptoms all stopped.

In other words, when the gratitude became neutral, it became *real* gratitude. Real gratitude is gratitude for *everything* instead of gratitude for things that were favorable or desired. As soon as the PDerer was neutral in his gratitude, his gratitude became directed at the universe in general instead of things that he particularly wanted or liked.

Which is the same thing as saying he became glad to be alive and a part of the great play of cause and effect and love that runs the universe. And a person who is truly glad to be alive and feels safe no matter what no longer needs to dissociate from his ability to feel.

When he ceases to dissociate, his brain flips off the switch that activates dissociation. He climbs back into his body, to stay. He no longer needs to play dead. The long-standing risk of potential emergency is over.

No exceptions

If a person is trying to experience the fullest expression of the neutrality type of gratitude for everything in his life, there can be no exceptions. As long as a person is nursing a grudge, he's not really grateful.

So long as a person is blaming anyone or anything other than himself for the events of his life, he cannot know peace in his heart.

The PDer will benefit from practicing gratitude for experiences that seemed painful at the time. Then he can remember and feel the pain of those experiences (re-associate). Then, he can neutralize the pain and be done with it.

If he simply can't find a way to be grateful for some bit of bad fortune, he can use the approach I learned from a dear friend: "When you can't understand why something has happened, assume the kindest possible explanation and you might be closest to the truth."¹

For example, suppose you come down with a dread disease. Try and come up with the kindest possible explanation. Is it possible that in a previous lifetime or in this one, you prayed for a friend or child who was sick? Maybe you even asked that the illness be transferred to yourself. Is it possible? If the sick person recovered against all odds and now you are sick – maybe it's because your prayer was answered. In this case, this might be a very kind explanation, reflecting well on the your magnanimity. And it suggests so much to be grateful for.

If a PDer is nursing a grudge against some perceived unfairness, he might try to be more original and creative in understanding how he himself might have set the mess in motion. Or he might want to consider how he has learned and benefited from his "troubles" in the long run. He can turn the problem into a blessing or an answer to prayer. And voila! Gratitude.

Or, he may need to learn that something that he perceives as unfair to others is actually, in some incomprehensible, "big picture" sort of way, actually fair. One PDer, trying to explain why people should keep their hearts blocked from all emotions told me, "If you don't, you'll go crazy; you'll see inequality in the world and you'll fret over it. You might even kill someone." I asked her if possibly the seeming unfairness in the world might actually be manifestations of cause and effect. She said that it didn't matter what the cause was. She assured me that the best way to deal with it, for a big-hearted person like herself, was to keep the heart blocked off from any emotions.

Some tips on learning gratitude are included in the chapters on Treatment Techniques.

Defining vibrant

In an upcoming section, am going to use the term "vibrancy" to mean the sensation in the chest that I've been struggling to find a word for.

¹ Thanks to Lendie Bliss

The words “vibrancy “ and “vibrant” are related to the word vibration. In addition to referring to vibration, vibrancy can also mean resonant, throbbing, radiant, sparkling, or vivacious (full of life).

Possibly because many PDers tend to *look* at things, including themselves, instead of *feeling* things, including the insides of themselves, they often assume that the word vibrant is primarily associated with colors. They think that vibrant means bright or fluorescent. This is a very limited meaning of the word vibrant.

And, although we *can* use the word vibrant to describe colors, it refers not to brightness, but to the way that colors sometimes seem to vibrate when you rest your eyes on them.

For example, a relaxed person, gazing across a mountain lake at the trees in the far distance, may notice that the colors of the trees and the sky and the lake are not a fixed, specific color. The colors seem to shimmer, as if they are rapidly vibrating between one color and another, or the colors alternate between sparkles of color and sparkles of pure light. These colors are vibrant – they are vibrating.

The PDer may not know what I am talking about, so I will explain. When a person sees the vibrations of light and sparkle when he gazes at colors in nature, he is perceiving some of the vibratory energy that is being generated and/or reflected that is not in the normal visual range of red to ultraviolet. This occurs when he registers visual perceptions using his heart’s resonance instead of his brain’s analytical processes. His heart’s electromagnetic field can resonate with lightwaves that are outside of the usual retinal perceptions. He “sees” vibrating color tones.

On the other hand, if a person is deeply depressed, colors may utterly cease to vibrate. If a person is depressed, the colors in a landscape may appear flat and matte: without any inherent vibration.

When colors are perceived as vibrating, they can be referred to as “vibrant colors.” Fluorescent colors have a greater likelihood of being perceived as vibrant. Still, no matter what the actual color, the emotional state of the observer plays a large part in the degree to which colors are perceived as vibrant or not. That’s enough about color.

Because I am going to use the word vibrant quite a bit, I just needed to make it clear that vibrant refers to *more* than colors. When I use the word vibrant, I am talking about a vibratory feeling, a small, gentle, physically felt sensation that resonates, flows, and swirls in response to stimuli that arrive via sight, hearing, taste, touch, smell, or the intuition.

In closing: reviewing the superscript quotes

Since we’re defining terms that might have been misunderstood due to lack of experience, I want to make clear the meanings of some of the quotes that I’ve inserted above some of the chapter titles.

St. Teresa of Avila said, “Pay no more attention to the mind than you would to the ravings of an idiot.” Mother Teresa of Calcutta said, “No one ever got into heaven (found happiness) by being clever. And the Mahatma Gandhi reminded us that “The mind is the attorney for the devil.”

In every case, these great souls were reminding people to turn off the relentless jabbering of the undisciplined mind and instead, feel the heart. Make the mind subordinate to the resonances of the heart. Feel, don’t think. Let the mind be the perfect servant of the heart feeling.

Years ago, I learned the following affirmation and prayer: “I shall reason, I shall will, and I shall act. Guide Thou my reason, will, and activity to the right thing I should do.”¹

This simple quote shows the correct relationship between heart and mind. The heart feels the vibrations of the land, sea, and air, and of the hearts of others. It also feels the voice of inner guidance. The mind’s job is to reason, will, and act in response to those vibrations. A person who has chosen to not feel will not be able to live. He will become increasingly mechanical, and then, like the denizens of Dante’s lowest circle of Hell, he will become frozen. His only movement may be the unending tremor of fear – the wordless call for comfort that even infants make when their hearts and brains are not working in tandem.

So feel.

I hope these word definitions have been helpful.

Redundancy

Some of my proof-readers have pointed out, correctly, that my writing suffers from redundancy. Many PDers, on the other hand, have told me that, in the beginning, they *think* they understand what I’m writing about, and it helps that I present the same information over and over again, in different ways, so that they eventually realize that they do *not* understand what I am talking about.

Some recovered PDers have told me that they honestly didn’t understand all that “heart stuff” in my book until *after* they recovered. Many have admitted that they skipped over the chapter about the feeling role of the heart in their hurry to get to the “important part.” But they did begin to suspect that there was something about the heart that they didn’t understand when, due to my redundancies, they realized that the bits that they tended to just skip over were the ideas that they truly did not comprehend. So, I apologize for the redundancies. Then again, if after reading this chapter even one reader is able to smack his forehead and say, “Oh! She means an actual physical sensation!” it will have been worth it.

There have been many instances of patients who have been told repeatedly, verbally, all the bits in this chapter. And when, after a spurt of gratitude that results in feeling safe, they suddenly notice a physical feeling of expansion in the chest, they usually say to me or email to me something to the effect of, “Why didn’t you tell me it was a *physical* sensation?!”

¹ From a talk by Paramahansa Yogananda (1883-1952), who has been described as “The father of yoga in the west.”



Dear reader. The following chapter was slapped together very quickly so that I could include it with the summer 2008 posting to the recovery website. Please forgive the hundreds of weaknesses in the writing.

A more readable version of this chapter, and the rest of the chapters on techniques for healing from dissociation, will be posted as soon as possible. However, because my schedule is rather frantic for the next five months, I do not anticipate posting the remaining chapters, the index, and the fine-tuning of these currently posted chapters prior to spring of 2009, at the earliest. Thank you for your patience.

* * * * *

There's something within me that holdeth the reins; There's something within me that banishes pain; There's something within me that I can't explain; All that I know is, there's something within."

Lucie E. Campbell, aka The Mother of Gospel

CHAPTER FORTY-FOUR

"SOMETHING WITHIN"

PDers dissociate from pain because they do not know how to deal with it. They don't realize that "there's something within that banishes pain."

The correct method for dealing with pain is to surround the painful area with the feeling that might best be referred to as "the feeling of expansion in the chest." The feeling that can best be described as "expansion *in* the chest," not expansion *of* the chest, is also the same "feeling" that allows the body to resonate with physical sensation. The feeling of expansion in the chest resonates with all physical sensations and is the basis for the feeling capacity of the five senses.

We learn in school that physical sensations are electrically transmitted to the brain via the sensory nerves. We aren't always reminded that physical sensations also create electromagnetic *vibrations* that interact with the electromagnetic waves that are produced by the heart.¹ After physical sensations have been transmitted to the brain and analyzed, these brain thoughts are also transmitted to the heart. The heart is thus the body's primary location for sensory input.²

¹ In western medicine, we say that the heart creates the electromagnetic field. In traditional Asian medical theory, one can say that the electromagnetic field creates the heart. In terms of understanding the bigger picture, the latter is more correct. However, the cause and effect situation *can* go either way: an alteration in the physical heart can affect the field, and an alteration in the field can affect the physical heart.

² Confirmation of this fact, long asserted by the religions and cultures of the world, is now available from science. Heart transplant recipients very often manifest the sensory preferences of the donor. Well-documented case studies abound. Not only are sensory preferences retained by the harvested heart, but in some cases, specific, usually recent, memories are retained in the heart as well. Cases exist in which, for example, a heart recipient was able to sing songs recently written by the donor or, in another example, remember events that recently happened to the donor. In one of the most dramatic cases of transplanted heart memory, a five year old heart recipient was able to describe the events that led to her donor's murder.

As the heart field resonates, or not, with the sensory vibrations, a person experiences *feelings*. The type and quantity of resonance, that is to say, the feelings, are transmitted, via nerves, back to the brain. The brain, relying mostly on habit or innate, animal reflexes, then creates a response to the heart's feeling. The response may be a thought, or it may be a physical action.

In other words, the brain responds to both nerve signals and the heart's responses. The heart responds to vibrational feelings and to the brain's analyses.

The combinations of responses are supposed to be under our conscious control. If we can consciously control which set of inputs we respond to, we can choose, at any given moment, to be more in touch with the brain or more in touch with the heart. We can focus on analytical thoughts when we are at work and we can focus on the feelings contained in nature and art when we relax.

Our self-controlled consciousness, and not the circumstances of our life, is supposed to determine the extent to which we allow the mind's responses or the heart's responses to dominate at any given time. We are the healthiest when we maintain a balance between the two sets of responses. If we are using both heart and mind, we can be calm inside while active on the outside, and we can also be actively feeling and thinking on the inside while we appear to be sitting motionless, in a physical state of perfect calm.¹

To attain this balance, a person needs to be able to regulate, at will, the amount of heart feeling that he is using at any given time. We are designed to be able to feel as much or as little "expansion" feeling as we choose. In other words, the sensation of expansion in the chest that automatically occurs when a healthy person beholds a beautiful sunrise or hears a magnificent piece of music *can* also be conjured up even in the absence of those more obvious stimuli. This concept is crucial and bears repeating: a human is supposed to be able to produce, at will, the feeling of expansion in the chest.

Using the expansion feeling as a healing mechanism

The expansion feeling, which enables us to detect a portion of the electromagnetic field that shapes and defines the physical body, *must* be available to us, when needed.

The terrifying but precise recall of the abduction, abuse, and the location where the brain-damaged donor had been left for dead, as well as a distinct description of the abductor, led to the discovery and conviction of the perpetrator.

For millennia, the heart has been considered the primary interpreter of the senses. A short-lived error in recent "scientific" thinking created the hypothesis that the brain was solely responsible for sensory awareness. Modern medicine is forcing a return to the correct understanding, while giving a nod of appreciation to the supporting roles, including sensory memory *storage*, that are played by the brain.

¹ One of the goals of religious attunement is constant, conscious awareness of the electromagnetic field of the heart: constant, conscious awareness, constant, conscious feeling. The individual's heart vibrations are part of the larger Universal Heart vibration, in the same way that a wave is a part of the ocean. By practicing attunement with one's own heart feeling, the spiritual aspirant can reduce one's identification with one's physical body and increase attunement with the Universal Heart. As a side effect of this attunement, spiritual adepts are able to exercise heart-based, loving control over their minds and bodies: they may be perceived by others as being profoundly calm inside, even when they are in the midst of activity.

The expansion feeling is not merely used for pleasure: the expansion feeling serves as a crucial stabilizer for the physical body. It can be consciously used to maintain stability in our physical body's electromagnetic system if the body becomes destabilized from injury or fear.

The "expansion feeling" enables us to feel the injury or fear, and fix it. By applying the expansion feeling to the destabilized (painful) area, the correct electromagnetic pattern contained in the heart's field can restore the destabilized pattern.

The heart's field contains the "instructions" for our physical make-up. If we are emotionally healthy, the vibrational field is the stabilizer that consoles us when we are frightened, or that comforts us when we are injured.¹

When the heart's resonance-with-feeling messages to the brain are disabled, an event that occurs, temporarily, during an emergency, the brain responds to sensation without the benefit of the heart's input. The brain is capable of making very fast but fairly crude responses to sensory events. In a *healthy* person, the brain is the primary respondent to sensation *only during emergencies*. During emergencies, the awareness of the chest expansion feeling, also known as the heart feeling, is shut down.

An emotionally unhealthy person, for example, one who has learned to selectively dissociate from his ability to feel pain, has also, inadvertently, learned to selectively prevent his heart from feeling. Whenever the dissociation kicks in, the brain becomes the primary respondent to sensation. During these times, the heart just serves as blood pump – the sensory message activity of the heart is temporarily disabled.

¹ I read in a book of yogic essays a list of names for the feeling that accompanies the expansion in the chest. I cannot find the reference, but I recall that the list of names included Divine Mother, the Comforter, the Protector, Love, Chit, Holy Ghost, Om, and Amen, among others. In the glossary of *Journey to Self-Realization* (a collection of talks and essays by Paramahansa Yogananda, published in 1997 by Self-Realization Fellowship), Divine Mother is defined as "The aspect of God that is active in creation; the shakti, or power, of the Transcendent Creator. Other terms for this aspect of Divinity are Nature or Prakriti, Aum, Holy Ghost, Cosmic Intelligent Vibration." This vibrational power can be felt. Saints and sages have confirmed that the feeling that accompanies the expansion of the chest, a feeling which occurs spontaneously when perceiving something of great beauty or grace, is a tiny taste of the Universal Vibration. The closer a person comes to attaining spiritual attunement with God, the more he is able to feel that vibration. A great saint is, after all, not a person who has become numb: a great saint feels deeply.

Many religions point out the dangers of *identifying* with sensory perceptions: when a person can make himself believe that he *is* his sensory events and his thoughts, he forgets that he is actually a soul – an intelligent, immortal, vibratory power. As long as a person feels sensory perceptions for what they are – expressions of nature – his soul is not deluded. The goal of the spiritual life is *more* feeling, not less – so long as those feelings are not tainted with personal likes, dislikes: preferences and attachments. The desire to not feel pain must be considered a "dislike:" an "attachment." When sensations are greeted with judgements and dissociation, they become problematic. If sensory experiences are perceived correctly – the way that God perceives them – they are never a problem.

As noted in Paramahansa's translation and commentary on the Bhagavad Gita's verse 40-41, "With enhanced perceptions rooted in a knowledge of the unending joy of God, the advanced yogi ["yogi" means one who has union with God], far more than the worldly man, is able to enjoy the sensory world – its people, its roses, its skies!" (*God Talks With Arjuna: The Bhagavad Gita*, Self-Realization Fellowship, 1995, page 160).

When a person with Parkinson's is dissociated from his ability to feel pain, that is to say, when he is trying, physiologically, to behave as if dead, he may relegate nearly the entire job of sensory perception over to the brain. This reliance on the brain alone, without the input of the heart, results in a greatly diminished ability to smell, taste, and perceive touch. It also creates altered perceptions of visual and auditory input.¹

People with Parkinson's do not think that they are numb. However, their spouses and children may quickly affirm that the PDer does not have normal sensory responses, normal "feelings." Even those PDers who admit to having reduced senses of smell and taste have no concept of the extent of their actual numbness. During recovery from Parkinson's, one of the most frequent surprises for the recoverer is the extent to which he can suddenly experience his five senses.

PROBLEMS WITH THE ENGLISH LANGUAGE

Strangely enough, there is no word in English to describe the feeling of expansion in the chest. I spoke with many highly educated English speakers. They all knew very well the sensation that I was describing, and none of them knew an English word for it.²

I asked a Catholic Spanish speaker if she knew the Spanish word that is used to describe the feeling of expansion in the chest. She said promptly that the feeling was called "Espirito Santo:" literally, the Sacred Spirit.

In *English* Christianity, this same concept has been poorly translated from the Latin as "Holy Ghost" – a translation that strays far from the original meaning, and which incorrectly suggests a disembodied entity floating around in space, possibly wearing a white sheet).

I asked a non-denominational French speaker for the French word that describes the feeling in the chest. He said (in English) that the feeling was called, "Love, with a capital L."

When I started pestering friends, looking for a word to help PDers understand this concept, a recovered PDer helpfully informed me that, in ancient Greek, the word that meant "heaven" did not refer to a *place*: the word that meant heaven was synonymous with the Greek word that meant "expansion."³

¹ Please see appendix xxx for the specifics on how visual and auditory input, in particular, is altered when a person is not in parasympathetic mode. Western research has confirmed that sensory perceptions vary depending on the mood of the subject.

² The fact that we have no word in English for this core feeling of heart expansion, that is to say, the feeling that allows us to feel all other sensory events, may be related to the historically austere English concept that sensory feeling and self-awareness are two mutually exclusive processes. Oppositely, the founders of the great world religions explained that feeling (the heart's response to physical sensation) and awareness are the same thing.

While it might be acceptable for an Englishman at the funeral of a dearest friend to shed a few tears of a "not unmanly emotion," he has also been advised, historically, to keep a stiff upper lip. I had to wonder if the English inability to even name the feeling of expansion in the chest might be related to the high percentage of people from the "sceptered isle" (where every prospect pleases, and only man is vile) that develop Parkinson's disease.

³ In ancient Greek, heaven was not a *place*, it was an expanded feeling. I suspect that it was the obvious feeling: the feeling of expansion in the chest that occurs when one experiences something glorious. Eastern religions' and original Christianity's understanding of heaven also recognize that heaven does not

I work in English. Other languages have sublime words like (I translate) sacred spirit (soul energy), Love, and heaven to describe the wondrous, joyous sensation that is experienced when a person opens up the door of his chest – a door that opens onto his own vibrations of soul. In English, I have to settle for “chest expansion:” a feeble phrase that didn’t begin to express the glory of this feeling.¹

Certainly, this English vocabulary problem was not going to make my job any easier. However, terms like heaven and Love weren’t going to convey a *sensory* meaning to a person with Parkinson’s; a phrase like “feeling of expansion in the chest” *might*. And so, with a sigh of regret, and a hope that I was making the right choice, I decided to use the phrase “expansion of the chest” to help PDerers learn about the underlying basis of all heart-based sensory experience.

USING THE FEELING IN THE CHEST

We realized, in 2008, that a person stuck in partial recovery from Parkinson’s needed to stop being dissociated from his ability to feel.

In order to *cease* his dissociation, we suspected that the PDer would have to feel the original pain from which he had dissociated. We based this guess on the fact that many PDerers, when they first learned to open their hearts, suddenly felt physical pain. Sometimes, this pain had an emotional connection. As they experienced what seemed to be a pain that had been held in suspended animation for decades, they were able to sit with the pain, feel it, and then say something along the lines of “that wasn’t so bad, after all.” With the wisdom of aging and the safe environments that they had created, they now

exist in a location, *per se*. “Heaven” is a feeling: a sublime feeling, but a feeling, nonetheless. As Jesus said, “The kingdom of heaven is within you,” and “The kingdom of God is at hand (“at hand” meaning, “you already possess it”).

When a person dies and is no longer encumbered with the physical, it becomes easier for him to perceive the subtle vibrations of feeling that made up his unique nature. The after-life time period is sometimes described as “heaven.” In this meaning of heaven, heaven refers to an increased ability to *feel* the vibrations of one’s soul. When a body dies, the intelligent, vibrational field and its derivative, the electromagnetic field that directed the body, no longer are physically connected to the atoms that derived from the electromagnetic field. The intelligent, vibrational field of the soul is then free to move through space and the ether, just as a radio wave is free to move through space. Astral “worlds” do exist and can be referred to as “heaven,” but they do not have a physical basis. These worlds are vibratory realms in which one’s individualized consciousness, if so inclined, can enjoy increased *feeling* of that “world’s” energetic phenomena.

¹ A patient shared with me this maxim: “The goal is not to feel God in your heart. The goal is to feel that you are in the heart of God.” Certainly, if one feels himself to be physically cradled in the heart of God, he will be able to use that presence of Divine harmony to influence his own electromagnetic fields, should he become injured. PDerers should also note the use of the verb *feel* throughout this paragraph.

While writing this footnote, I suddenly remembered the words of a rollicking song, a spiritual, which I had sung at Girl Scout camp – without understanding the words. The words proclaim, “Rock my soul in the bosom of Abraham!” This refrain was about placing one’s heart in the larger heart of God and being cradled there, rocked, just as a body is cradled in the arms of the loving mother. The world can be a painful place. To survive and heal from the pains we must always be able to feel that the love we can draw on is greater than the pain. As Paramahansa Yogananda said, “In this drama of life, our love must be greater than our pain.”

had a skill set that enabled them to deal with a pain that, decades earlier, had been too traumatic to address.

But only some PDerers had been able to feel these heart-awakening pains. More of the PDerers didn't even know what we meant by the word "feel." They also, for the most part, had no idea whatsoever as to how an adult *correctly* deals with pain. A PDer who couldn't feel and who didn't know what to do with painful feelings, should they occur, had two things he needed to learn. He had to learn what *feeling* actually is *and* he would have to know how to correctly deal with pain.

By carefully analyzing the physical and mental processes by which adults process physical pain, we were able to figure out a very specific vocabulary that even PDerers could understand which allowed them to learn how to feel, and how to deal with painful feelings.

We started teaching PDerers how to feel the "expansion in the chest" sensations while he simultaneously experiencing his long-ignored pain.

By so doing, they were able to render the pain non-threatening, even boring. The pain, having been reduced to mere sensation, and no longer associated with fear or destabilization of the body, could then be processed and healed.

The next problem was getting the PDer could figure out the pain memories that would be most efficient in turning off the dissociation. Most PDerers have suppressed many, many pain memories. But we hoped that he might not have to consciously call up every single one of them. If he could process the most *crucial* pains, that is to say, the pains that might have been involved when he first started his lifetime habit of selectively dissociating from his ability to feel, he might open his heart enough that the other, coincidental pains might get processed easily, maybe even without conscious effort.

Recalling the feeling of "something within" – or not

When we realized what the PDerers needed to do, we started asking them to conjure up in their chests that physical sensation of expansion that occurs in the chest when you experience something beautiful or glorious.

Some PDerers who were stuck in partial recovery *were* able to understand what we meant by "the physical sensation of expansion that occurs in the chest when you experience something beautiful or glorious." Others had no idea what we were talking about. So far as they knew, they had *never* experienced the sensation that we were talking about.¹

¹ As the ancient Vedas point out, God has three qualities: He is "Sat:" ever-existing; Chit: all-feeling (also expressed as all-aware, all heart, all Love); and Ananda: ever-new bliss. When a person learns to feel all the feeling, awareness, or "love" in the cosmos, he has the same "universal feeling" that God has. This is heaven. Oppositely, when a person has no feeling, but merely submits his sensory experiences to mental analysis, he is in a hell: unable to feel the vibrations of sensory-based joy that make life worth living, and unable to feel the vibrations of love that connect us to the rest of the universe.

Of course, mankind has passed through a terrible Dark Age, during which humans had almost no capacity to imagine anything having to do with "consciousness or awareness." During this dark time, the western religions, for the most part, and even some of the eastern ones, came to assume that "heaven" was a physical place – similar to earth, only without the pain.

Some remembered having felt it in the past. A few of them were still able to feel it, now and then, under certain conditions, such as during yoga class or while attending an opera, or other, highly specific environments. The situations were always ones that the PDer considered to be “safe and good.”

But for the most part, PDers who were stuck in partial recovery either didn't even understand what we meant by “feeling of expansion” or, if they did know, it was a part of their pre-Parkinson's pasts.

Why did we ask PDers to feel this particular sensation?

The feeling of expansion or contraction in the chest in response to sensory stimuli is actually the *basis* for all parasympathetic feeling. Most of our PD patients actually thought that *analysis* of sensory input was the same as *feeling*. But *analysis* of sensory input is how the brain responds to sensory input when a person is in a state of emergency. During an emergency, the heart-brain entrainment ceases. During an emergency or a near-death condition of dissociation, the heart starts working like a mere pump; the heart stops *feeling* sensory input. During an emergency, the brain, not the heart, deals with sensory input.

Although I've mentioned it already, I'll say it again: it's easy to understand why the heart stops feeling during dissociation or an emergency: after almost bleeding to death *or* when being chased by a lion, a person does not want to be distracted by the sight of a glorious sunrise, the song of the skylark, or the nagging pain of a blister on the big toe. During dissociation or an emergency, the sensory experiences are not *felt*: they are analyzed. During these times, physical sensations are not recognized by the heart: they are processed solely by the brain.

Many PDers have inadvertently turned off their hearts by selectively dissociating from their ability to feel pain. Because of this, they didn't even know what we meant by the word *feel*. So we figured out a way to teach them how to *feel*.

NOTICE THE SMALL EXPANSION IN YOUR CHEST

The healthy reader may not believe that an exercise of this simplicity has the power to turn off the dissociation that causes Parkinson's disease.

On the other hand, a person who is stuck in partial recovery from Parkinson's disease may not believe that anyone ever feels a small, physical sense of expansion in his chest which feels good and which is distinctly separate from the chest's rib and muscle expansion that occurs in response to inhalation.

But the key to turning off the selective dissociations that can prevent recovery from Parkinson's is this simple:

- 1) Notice that, when you feel deeply moved, your chest experiences a sensation of expansion. It may be a very small feeling, or it may be big enough to trigger goose bumps on the skin.
 - 2) Practice imagining that feeling of expansion being bigger or smaller.
-

If a person can do the above two steps, he can recover from mentally-induced symptoms of Parkinson's disease.

If the PDer feels nothing, a later chapter offers techniques in learning how to create and feel this sensation.

After a person learns to feel sensation in his own chest, he can move on to the next step: using those sensations to guide his thoughts, instead of allowing his fear-based thoughts to inhibit his ability to feel.

In order to practice using the chest sensation to guide his thoughts, a person can ask himself simple yes or no questions, and noticing the expansion that occurs in the chest if the answer is "yes." The question needs to be very simple, such as, "Can this technique actually work?" (Do not ask questions for which the answer doesn't matter. For example, don't ask, "Should I have the soup or the sandwich for lunch?")

The healthy reader may be thinking to himself, "Huh? Is it humanly possible to get through the day without checking in with the "heart?" How can a person know how to behave if he isn't constantly feeling that "something within?"¹

The reader who has Parkinson's may be thinking to himself, "Huh? I never heard of anyone noticing whether or not his chest was feeling expanded, or using that sensation to tell himself what to do!"

The reader with Parkinson's might be surprised to learn that obedience to chest expansion is how healthy people recognize their "voice of conscience." If they notice a faint, pleasant sensation of expansion or an increase in "comfort" in the chest, it means that they are thinking about doing something that agrees with their conscience.

The PDer may be surprised to learn that conscience is *not* a logical, word-based rational process. It is actually a feeling. This is why so many people refer to conscience or intuition as a "gut feeling." Though, actually, it's not actually a gut feeling; it's a chest feeling.

One of the more advanced steps in recovering from Parkinson's (if one becomes stuck in partial recovery) is "having a conversation" with the chest feeling; asking it simple yes or no questions. A physical sensation of expansion or comfort means "yes," and no change means "no."

As a person gets more familiar with "conversing" with chest sensations, he will be able to get more advanced answers to questions: answers that involve words other than "yes" or "no."

¹ A famous gospel song refers to "Something within me that banishes pain, Something within me that I can't explain." The song's writer was inspired when overhearing a Memphis street musician explaining why he only played Gospel music, and had just turned down a paying request to sing a blues song. He explained his refusal to sing anything but Gospel by saying, "All that I know is there's something within."

Just for fun, and because it makes the above more pointed, I'll add that I read in a 2008 interview of famous blues musician B.B. King that King started playing blues because, when he was a street musician, he quickly figured out that street musicians don't make money when they play Gospel. Blues players make money. So the "Something Within" speaks more loudly than money – in some people.

For example, at some point, the PDer may need to practice feeling a specific pain from which he has been dissociated. How can he know which particular physical or emotional pain is the “right one?” If he has learned to get answers from his chest sensations, he can ask his own heart which pain he needs to work on.

He will ask his chest what he needs to *feel* in order to do the necessary healing. Whatever *immediately* comes to mind is the thing that he will need to feel. Also, it will be the thing for which he needs to feel grateful – but more about that, later.

Of course, many PDers don’t like whatever it is that the heart suggests. In fact, many PDers, after asking their heart, or chest, what old physical or emotional pain they need to *feel*, so that they can process the pain instead of having it hidden away, have a sudden thought. Then, they dismiss that thought, often saying something along the lines of, “I thought of something, but it’s not important,” or “My heart thought of something but I’ve already dealt with it, so I’ll think of something else.”

This habit of wanting to *think* of something instead of listening to the heart needs to be overcome. The PDer must not only learn to feel the sensations of his chest, he must learn to trust them. If the heart (chest) tells you that you need to work on a particular physical or emotional pain, you can be pretty darned sure that you do need to work on it.

After realizing what it is that needs to be felt, the PDer then needs to feel it. He needs to imagine himself experiencing the actual pain that he has been avoiding for so many years.

Then comes what can be the hardest part of the healing process: being *grateful* for whatever painful event your heart tells you to work on. This can be the most difficult part of recovery. Very often, the heart has suggested working on a very painful situation, one that may have rankled in the bosom for decades or which might have been a life-threatening pain at the time it occurred. A PDer may feel that the event is too painful to inspire gratitude, or that the instigator of the pain was so wrong, so cruel, that gratitude is impossible.

Chapter xxx will share techniques on learning how to be grateful for horrible things. For now, just keep in mind that any greedy child can say words of gratitude for getting the things he *wants*. Only a truly wise person can be grateful for a chance to play a part in the drama of life – even if things happen that he doesn’t like, doesn’t understand, or are painful.

Finally, the PDer must increase the diameter of the sphere of “good feeling” that accompanies the subtle, physical sensation of expansion in the chest; he must make the “feel good” area large enough to encompass the physical pain that is being remembered. This sensation can then be transferred to the physical area that is hurt. The “good feeling” must completely envelop the pain area.

Three factors need to be going at once: The physical pain needs to be recalled, a mature, circumspect attitude or gratitude needs to correct the old fear-of-pain attitude, and the “feel good” sensation of the chest needs to be literally larger than the area that is feeling the pain.

Once all three of these processes are in place, a person just concentrates on both the feeling of the pain *and* on the good feeling that is larger than the pain. If his mind starts to get in the way with any sort of distraction, the mind has work at figuring out how to be grateful, in retrospect, for the painful experience or the life lesson that might have been associated with the pain.

If no pain had been felt at the time, the PDer can recall the event and linger with the memories, until the pain of the event begins to manifest as physical sensations.

When the lump in the throat, the burning in the eyes, the knot in the stomach, or whatever the pain *should have been* begins to appear, the PDer needs to *feel* the pain.

I'll be redundant here. As soon as the PDer feels the pain, he must simultaneously feel the good sensation that is associated with the sensation of chest expansion. He must make the area of "chest sensation" bigger than the area that is feeling physical pain. The chest sensation must become so large that it encompasses the physical pain. If the pain is covering the whole body, then the chest sensation needs to be bigger than the body.

"In this drama of life, your love must be greater than your pain."

As the person sits quietly, feeling both the pain and "good feeling" that comes from chest expansion, a "miracle" occurs. The pain becomes less intense. After a few moments, or possibly forty five minutes, the pain becomes just a sensation – it is no longer able to induce fear or pain. The pain becomes less interesting; it becomes boring. At this point, the body is able to regard the pain objectively and determine what steps need to be taken to heal the pain.

That's it. A PDer may need to do this many times, repeating the experience with the same event several times, or addressing many different events. A PDer may only need to do this once. Some PDers have found that they only need to do this once and they don't even need to recall a specific event; by merely feeling the physical sensation in the chest and combining this sensation with free-floating gratitude, they "flip the switch" and come back to life.

Chapters on treatment techniques xxx will share tips and insights that might be helpful for a person who is struggling to feel that faint expansion in the heart – what the poet James Montgomery called "the motion of a hidden fire that trembles in the breast."

These chapters will also provide help in understanding how to feel gratitude for some *aspect* of what may have seemed like, and may have actually been, an atrocity.

Disdain for dredging up the past

For now, I just want to address one point that many PDers bring up. They say that it is not good to dwell on the past, and that there is no point in dredging up old pains. This is true. However, for the PDer, the pain is not yet in the past. The pain, even if it has been dissociated or not remembered, is still very much in his present. The pain has been put on hold, and is silently setting off alarms in the subconscious, while being told that it will be dealt with at a later time.

Now is that time.

Keeping the still-current pain hidden via the trick of instructing the heart to feel no pain has a dire side effect: it can set in motion the illness known as Parkinson's disease. This illness can cause far more long-lasting, more painful symptoms than the original pain ever could have done. So it is time to finally address the pain and be done with it.

This technique is not about dredging up old pains and dwelling on them. This technique allows a person to address a pain that is still sitting in his "In" box, which has not yet been looked at and dealt with.

Of course, as soon as the pain has been processed, one will not need to dwell on the pain. Then again, if, during the exercise, one has truly become grateful for the experience or *something* about the experience, then it will be OK to dwell on it: dwelling on the experience will activate gratitude, after all.

There is no harm in recalling, with true gratitude, an event that helped shape one's life.

I don't believe it

The reader with Parkinson's disease may be thinking that the above technique is far too simple to reverse the processes that result in the mentally-driven symptoms of Parkinson's. And he may not yet have any idea what I am talking about when I refer to the small sensation of expansion in the chest that, in a healthy person, accompanies relaxation or joy.¹

Then again, the truly mature reader may be absolutely stunned that the PDer never had a clue about how to deal with "the thousand natural shocks the flesh is heir to."

¹ For many years I had received flak from PDers who were adamant that they were not using the adrenaline-based sympathetic nervous system because they always kept a tight lid on their emotions. They were insistent that any syndrome involving adrenaline must also involve hot-headedness and violence. Many PDers are proud of their ability to not get angry. Research has shown that PDers actually do angrier than most – they just hold it in. The article "Anger in Parkinson's disease: A Case-controlled Study", Macias, Y, et al, *Movement Disorders*, 2007 Nov; 23(2): 195-199, explains how 126 PDers compared to 126 age- and gender-adjusted control subjects in a study of anger, as measured by state-trait anger Expression Inventory-2 (STAXI-2). The PDers scored lower on anger *expression*, but scored higher on *inner* anger levels, *control* of outward expressions of anger, and *control* of inner expressions of anger. In other words, PDers are angrier than most, but they don't allow themselves to express it outwardly, or even manifest it internally, in terms of physiological changes. In my limited experience of listening to hundreds of PDers justify their often bitter attitudes towards events or people in their past, I would have to say that many of them have allowed anger, resentment, and self-pity to roil and fester in their brains for decades. However, those who seem (to me) to harbor the *most* anger are *very* likely to tell me that they don't *have* any anger – because they never *show* signs of anger.

One PDer told me that her sister warned her, "If you intend to recover from Parkinson's, you're going to have to get over your anger." The PDer was astonished. So far as she knew, she had no anger in her at all. She was puzzled by her sister's statement, so she went into her bedroom, shut the door, and prayed intensely: "If there is any anger in me, let me see it." She was horrified to sense black clouds roiling out of her body, filling the room with sooty darkness. As she felt this darkness, she also felt enormous waves of anger. It was directed at her school teachers, her parents, her siblings, her friends, her husband. She felt and remembered countless incidences of anger involving nearly everyone she knew. At the time of each incident, she had pretended to feel no emotion. But those angry feelings were evidently still inside her, waiting to be expressed.

And the immature reader may be wondering why the PDer hasn't availed himself of the most popular American method for dealing with pain: focusing all his attention on the pain, amplifying the pain, yelling about it, crying over it as often as possible, demanding pity, demanding retribution, yakking about the pain to anyone he can collar, cultivating outrage for as long as possible, and generally acting like a spoiled, three year-old child.

But no matter what the reader thinks of all this, this technique has proven to be a helpful exercise for teaching PDers how to deal correctly with the pain that occurs when they stop dissociating from the hearts.



PART V

APPENDICES

“There is something more hideous, more atrocious than war or than death; and that is to live in fear.”

– *Eve Curie, French author*¹

APPENDIX I

THE ADRENALINE-DOPAMINE RELATIONSHIP

Some of the subjects addressed in this appendix are redundant, and repeat concepts that were addressed in the main text. However, so many questions arise over the role of dopamine that I wanted to include this extra material, and repeat some of the basic ideas.

ADRENALINE – MORE THAN JUST FIGHT OR FLIGHT

Certain physiological events have long been known to occur in response to an overwhelming surge of adrenaline. Whether the adrenaline tsunami comes from within the body or is administered via a needle, the main events are: increased heart rate, increased bronchodilation, increased urination, and opening the pores for increased sweating. Aside from these symptoms, known for nearly a century to doctors, we have the more general aspects of adrenaline, the ones that are better known to the man on the street. We all know that, under the influence of adrenaline, we all think and move differently than we do when we are relaxed.

The first section will describe behaviors that occur in various arenas when the adrenaline system is being used for that particular body function. I will include examples of adrenaline system behaviors at high, medium, low, and exhausted levels of adrenaline.

The second section will describe behaviors that occur in the same arenas when the dopamine system is dominant.

Adrenaline in the various arenas

Speed of thought

Adrenaline-based thought is *fast*. Consider the example of a bicyclist who has just been hit by a car. While he hurtles through the air, his mind switches over to the adrenal neuropathway in the brain. His adrenaline levels surge, filling the thinking arena. This creates the following well-known change in his perceptions: time expands. He is able to distinctly process hundreds of thoughts. Sometimes, his “whole life passes before his eyes” before he hits the ground. He may be able to consider dozens of moves that might protect him. This tremendous rush of thoughts is able to be processed because, when the adrenaline neuropathways in the thinking arena are fully loaded, they do not dilly-dally around skipping from one side of the brain to the other, trying to balance logic and art, intuition and reason. Heck no.

¹ From a speech to the American Booksellers Association, New York, April 9, 1940.

When one is about to crash head first into a windshield of a car, floods of adrenaline send the thoughts racing straight down the center of the brain to the frontal lobe allowing for a supreme amount of information to be directed to the center of will and focus. The maximum levels of adrenaline blast another group of brain neurons which dive directly to the midbrain areas, the areas that regulate primal survival instincts.

Even if the emergency is not purely physical, if the “danger” is coming from the competitors in an academic contest, the person who is using the adrenaline pathways for his thinking function and keeping high amounts of adrenaline gushing through this part of his system will be able to think faster and more brilliantly than the competition.

Now, if the adrenaline system is being used (as opposed to the dopamine system) but the adrenaline *levels* are low, a different scenario occurs. The mind, the guiding force of the adrenaline system, is still the director of the show, but the low levels of adrenaline allow the mind to scramble around quickly from one thought to the next, often getting nowhere: the mind in a pointless whirl. This frame of mind can manifest as unfocused rambling, confusion, or even anxiety. These behaviors are mind-driven, but are not productive.

When the adrenaline system is dominant (the dopamine system is not in use) but adrenal *levels* are extremely low or exhausted, the mind simply doesn't know how to be effective. Depression or panic may ensue.

The intuition is not available in the thinking arena when the adrenaline system is dominant for that particular function. In all cases, whether the adrenaline levels are high or low, charging ahead, dithering in confusion, or moldering helplessly in depression, *dopamine and the intuition do not kick into the thought arena while the adrenal system is in use for that arena*. Instead, the negatively oriented, fear (adrenaline)-based mind controls the reins of the body chariot.

Again, when using the adrenaline system to perform certain thought tasks, the mind is in charge and the intuition is silent with regards to those thought tasks. Fear is the motivator that activates the adrenaline system. The mind-driven self-preservation instinct, whether raging at lightening speed in self-defense or just potheringly worried about everything in general, dominates when fear is present.

Motor function

When the adrenaline system is dominant (the dopamine system is turned off) and adrenaline levels are extremely high, the normal brakes on motor function are absent. We all know stories of a person who performed a superhuman feat, such as ripping the door off a burning car to rescue a child trapped inside. Such a powerful act of unnatural strength can occur when the adrenaline *pathway* to the midbrain is employed and the adrenaline *level* is set at maximum. At such very high levels of adrenaline, the self-evaluative, self-critical parts of the brain are bypassed; a person ceases to know that he cannot perform great feats of strength.

When the adrenal path is dominant for motor function but adrenaline levels are low, the pathways directing the body to keep moving are still in use. But without an overriding sense of adrenaline-induced purpose, a person may be merely restless instead of effectively dynamic.

When the adrenaline system is dominant for motor function but adrenal levels are exhausted, a person may feel internally restless but can barely stir himself to move. He

must almost create a pretend mental emergency to stir up enough adrenaline to get moving. His balance may be poor due to lack of a strong sense of direction and focus.

Note carefully: *just because adrenaline is exhausted, dopamine pathways will not automatically kick in.* The mental attitude determines which path is used. If fear is dominant with regard to motor function, the adrenaline path will be selected to govern movement even though adrenaline levels are set on empty.

Sensory perception

The adrenal pathway has neurons going to the incoming senses of sight, sound and smell. The neurons devoted to the rapid, fear-based processing of this information go more to the more primitive, self-defense areas of the brain than to the pondering, musing areas. Sensory information may be evaluated more in terms of what it means rather than just experiencing it.

When the adrenal pathway is dominant but adrenaline is at low levels, sights, sounds and smells are more likely to be perceived as annoying instead of provocative.

Pain

When the adrenaline neural pathways for monitoring pain are activated, pain can be ignored. Any of us might know of someone who has performed an impossible action such as fleeing a maniac by running a mile on a broken leg, or winning an eighteen mile bicycle race with a broken collarbone. One feels no pain as long as the adrenaline pain recognition pathway is in use and completely saturated. The neural circuits used when the adrenaline system is supremely dominant and topped up with adrenaline connect only tangentially with the pain centers.

When adrenaline levels are low but the adrenaline pain recognition system is the dominant (the dopamine system is not in use), a person might perceive pain, but might still hide his pain from others. The extent to which the adrenaline system or dopamine system is dominant in the socializing part of his mind may determine whether or not the person in pain tells his friends, his co-workers, the world – or even himself – about his pain.

When the adrenaline pain recognition system is dominant and adrenal levels are exhausted, anything or everything might hurt. His response to the pain might range from being worried to being scared.

Immune system

The adrenaline pathway sends repression signals to the immune system. How many of us don't get sick as long as some crucial bit of work remains undone? And then, when the workload lightens or we get to take a vacation, the adrenaline levels drop and the suppressed illness surfaces.

When the adrenaline system is dominant but the levels of adrenaline are low, the result can be frequent illness, allergies, or malaise.

Digestion

The adrenaline pathways give short shrift to food metabolism and digestion. When we are keyed up with fear and rage, the adrenaline system pathways to the stomach say, "Shut down" and the signal to the large intestine is often, embarrassingly, "Abort!" It

is not so much that adrenaline itself causes the digestion system to stop functioning: the entire collection of adrenaline systems work together to prioritize body functions when adrenaline levels rise. Digestion is at the bottom of the priority list when one's life is on the line.

When most of the adrenaline pathways are dominant but adrenaline levels are low, one might feel a desire to stay busy via eating, but might not actually feel hunger. This can lead to pointless, incessant snacking, as the person tries to sedate with food the anxiety caused by choosing to use the adrenal system during times of low adrenaline.

Temperature regulation

When adrenaline levels are high, the adrenaline system for temperature regulation bypasses the conscious awareness of temperature. During an emergency, a person might work valiantly for hours in the blazing sun or bitter sleet, not affected in any way by the dangerous levels of heat or cold. Hours later, when the emergency ends and the adrenaline levels drop back down to normal, he may collapse from the heat or shiver violently as his body starts to assess his temperature imbalance.

When the adrenaline system is dominant but adrenaline levels are exhausted, temperature control of the body is compromised. One might suffer from a tendency towards extreme heat or extreme cold, or even go back and forth.

Social interaction

The adrenaline system for social interaction, when fully charged with adrenaline, does not integrate well with the nerve centers that govern nuances of socializing. Instead, they go directly to the danger assessment area. This pathway will interpret a stranger's behavior patterns in light of whether or not the stranger is a safety risk. It probably will not determine whether or not the stranger's feelings have recently been hurt.

The areas of the brain that process sophisticated emotions are not well traversed when the adrenaline levels are high. When the adrenaline system is dominant and adrenaline is surging, a person can be very aggressive and defensive, dangerously so, towards anyone whose bona fides are not certain.

When a person's adrenaline level is moderate, but the adrenaline system is turned on in this arena, he may act in a manner that is called "harm avoidance." Harm avoidance means that one is careful to not anger others and avoids situations that might lead to high drama. The underlying reason for the harm avoidance is not so much the obvious reason of avoiding bodily harm. The actual reason is this: if a person is locked into using the adrenaline system, when situations that are fraught with tension arise, the resulting increase in adrenaline can make him act out uncontrollably, in a primal manner, with unbridled fear or rage.

If a person is suppressing a tremendous amount of rage or fear from previous life events and is primarily adrenaline-system dominant, he may be very afraid of any social situation that will elevate adrenaline levels.¹ His fears may have two prongs: the profound

¹ These fearful previous life events may include anything from a parent's epileptic seizures to violent beatings by an alcoholic step parent. They may have been events which, to an adult's mind, are not terrifying, but the events might have been terrifying to a child. The child's mind can be the one that determines neurotransmitter pathway choices, choices that may remain in place through adulthood. Even if the adult is able to consciously create less fear-driven pathways, it may be that certain events will

fear of provoking anger in the other person and the fear of evoking in himself an uncontrollable rage or terror in response to that person's anger.

Such a person may work furiously inside to harmonize his social surroundings. He may use his quick-working adrenaline system pathways to be always testing his surrounding emotional waters. He may use his rapid intelligence to manipulate social situations to minimize the level of stress. He may be a "people pleaser" or he may be a peacemaker. He might always be trying to anticipate and sedate any possible social tension.

On the other hand, to relieve himself of the moderate, but never ceasing level of adrenaline that courses through him, he may involve himself in intense sports. He may perform very well; using adrenaline, he may be able to move stronger, harder, and faster than his peers. He may even enjoy extremely dangerous sports: paradoxically, the relief he gets as he works off adrenaline can be calming.

If he is competitive, he may teach himself how to summon rage-level amounts of adrenaline during competitions, making himself into a primal, uninhibited competitor who is literally fighting for his life during his sports events.

Still others who are adrenaline dominant with moderate to low levels of adrenaline are merely suspicious and emotionally distant from most humans, especially people outside their immediate circle.

When the adrenaline system is dominant and adrenaline levels are exhausted, one usually avoids groups of people and may even avoid close contact, both emotional and physical, with loved ones.¹

Logic versus Intuition

The adrenaline system pathways in the brain do not rely on intuitional hunches. The primitive brain centers of fear, rage, and hunger are the main connecting points along the adrenaline system neural pathways. Logic sometimes also plays a role, but it is not the sophisticated logic of higher thinking, but the primitive logic of "I did what I needed to do to save my life (or the life of my child)."²

immediately throw him back into the fears learned in childhood. This is particularly the case if, through mental suppression and disassociation, he has ongoing unhealed injuries.

¹ A fascinating study on brain function and susceptibility to addiction in male primates who become alpha males suggests that their brain switches from dopamine system -dominant to adrenaline system-dominant. They also switch from addictable to non-addictable. Even if the primate is supplied with dopamine-enhancing drugs, his behavior and brain patterns remain those of an adrenaline-dominant alpha male. See: *Nature Neuroscience* 5(2): 169-174, 2002, Mike Nader.

One significance of this study, for our purposes, is that it suggests that neurological preferences for the adrenaline system or dopamine system are not hard-wired in, based solely on genetics or innate personality. These neurological shifts from one system to the other occurred in these primates in response to a change in their external environment: a change in social status or in danger levels. In the study, a shift in social standing to an alpha position caused a neurological shift towards the adrenaline system. This shift helps maintain constant vigilance, wariness, and an increase in physical strength. This point is very important to keep in mind for our discussion of Parkinson's disease. Most PDers point with pride to their extreme focus and self-control. They are often displeased to learn that their quick mental faculty, their self-control and will power are, for the most part, the result of their constant mental immersion in fear or other negative thought patterns. Their "superiority" is, to a large extent, a result their unhealthy, abnormal habit of performing all body functions via the adrenaline system.

² Those who are adrenaline dominant often consider themselves to be supremely logical. However, in my experience, most self-proclaimed adherents of logic are actually quite illogical. Their main source of

When the adrenaline system is dominant but adrenaline levels are low, a person may be extremely, pedantically logical, maybe analytical, but not particularly insightful. Mere listings of facts and old knowledge are what some people call “bean counting:” not real wisdom. Insight requires intuition. The adrenaline pathway does not intersect significantly with the intuition-directed, dopamine system pathways for logic.

When the adrenaline system is dominant and the adrenaline levels are exhausted, a person might imagine himself to be determinedly logical, while, in fact, his memory may be flagging and his thoughts might not be particularly cogent.

Breathing

Adrenaline is closely associated with diaphragm function. From a western perspective, we might agree that breathing is fear-based: it is fear of suffocation that causes a person to inhale every few seconds, every moment of his life. The greater the level of adrenaline, the deeper and faster are the breaths.¹

When the adrenaline system is dominant but adrenaline levels are low, the breathing may be slow and relaxed. However, if the adrenaline system for the mind is racing, however, or if one is anxious, angry, guilty, or fearful, the breathing may be rapid and shallow, or uneven. This is a case that shows how one adrenaline arena may be influenced by patterns occurring in another adrenaline arena.

When the adrenaline level is exhausted and one is emotionally depressed, with the mind stagnating, breathing may be slow and heavy, even difficult.

Gravity

When adrenaline levels are at their highest, they impart the power to jump higher, run faster, and last longer than most: defeating gravity, you might say. When the adrenaline system is dominant but adrenaline levels are low, movement may feel somewhat clumsy and uninspired, and only by summoning up extra mental will power, thus briefly increasing adrenaline levels, can one attain the focus needed to move easily. If the adrenaline system is dominant but adrenaline levels are exhausted, one feels greatly affected by gravity. If one is depressed, moving can be an effort. It can feel as if gravity has increased. The legs feel dense, heavy. Movement is slow and requires mental effort.

“logic” is “That’s the way I learned it so it must be true.” They tend to be fairly closed to new ideas, and particularly distrustful of positive thinking. This distrust can be so severe as to be illogical.

An example of this illogic is the following example of a not uncommon attitude: “I admit that if I meditate for fifteen minutes on having a stomach ache, I can give myself a stomach ache. But that doesn’t mean that if I meditate on feeling good, or if I pretend to feel good, I could make myself actually feel better. Negative thinking does create a negative result, I admit, but positive thinking doesn’t do anything *real*. I don’t want to waste my time on pointless “feel-good” thinking – it can’t possibly work. And even if I did feel better, it wouldn’t be *real*, so I don’t want to try it.” Some people, including me, consider this attitude to border on illogical.

Many PDers feel a sense of pointlessness, or even shame, if they indulge in truly positive thinking. However, they are usually brilliant negative thinkers, especially when it comes to self-criticism.

¹ Interestingly, in Asian medicine, it is the water element (the element of the kidneys and more particularly the *adrenal* glands) that governs inhalation. The metal element (the element of the lungs) only controls exhalation.

Shock

Shock can occur as a response to a potentially fatal blow or emotional event. Head injuries in particular can set in motion a shock type of response. When a healthy person goes into shock, it causes an unaccustomed high level of adrenaline to course through the system, filling all pathways. When the first rush of adrenaline wears off, the body may still be on the adrenaline system, even though the adrenaline molecules themselves have gone back down to their normal level.

In a case like this, the person may behave just like the descriptions earlier in this chapter for the various arenas when “adrenaline system is turned on but adrenaline levels are low.” In other words, thinking may be stunned, not making coherent thoughts. Movement may be impaired, the person in shock may wish to just lie still and try to sort things out. The body may be sweating or shivering uncontrollably. There may be a sense of ineffectual panic, with trembling and holding the body in a fetal position.

DOPAMINE – JOY AND INTUITION

Dopamine is the neurotransmitter of joy. The neural pathways of the dopamine system are the normal, default pathways of brain processing. The dopamine system is the one that humans automatically use when they are not performing a fear-based response.

In a healthy person, the level of dopamine present is simply this: enough to match how he feels. If a person feels mildly content, a small amount of dopamine is released. If he feels pretty good, more dopamine is released. If he lets himself feel great, even more dopamine will be released. Dopamine can also respond to expectation: just as adrenaline can be released in anticipation of a fearful situation, dopamine can be released in anticipation of pleasure. Loving, wise, grateful, and humble thought patterns trigger the dopamine systems.

Dopamine is an expectation-dependent neurotransmitter. Dopamine is discharged from its brain-vesicle holding tanks according to how good you feel or how good you think you are going to feel. Although most PDers do not realize this, a person does not have to work at feeling good. Dopamine and contentment are the default systems in humans; they occur automatically. Fear must be created. Joy is already present.

In order to perceive joy and experience the release of dopamine, one has to stop thinking fear-based thoughts: one must stop anticipating that one is going to feel bad. Even more than that: a person must feel safe.

- In a healthy person, when dopamine is released, it automatically floods the dopamine systems. The dopamine systems are always potentially available, always turned on. Dopamine itself is not released into the various systems, however, when their parallel adrenaline systems have been activated.
- The amount of dopamine available to stimulate the dopamine systems is good-mood and expectation dependent. To the extent that one feels safe or, following that, anticipates joy or a good result, appropriate levels of dopamine will be automatically released. If an adrenaline system is activated, dopamine will not be released for that system.

- In Parkinson's disease, dopamine cannot be released – even during positive thinking – because of incorrect signals in the brain that suppress dopamine release.

As an aside, the fact that the dopamine system neuro pathways are always potentially available explains why a stressed (fearful, adrenaline-dominant) person can take dopamine-enhancing drugs such as nicotine, methamphetamine, opiates, cocaine, or alcohol and soon feel the flow of false (due to drug-induced distortions) intuition and temporary joy: the pathways are always switched to the On position, just waiting for some dopamine to come along and activate them.

The activation of the adrenaline system, as well as the release of adrenaline into the adrenaline systems, on the other hand, is activated by choice. The choosing of fear instead of joy is sometimes a conscious choice and sometimes an unconscious choice. Sometimes the “choice” is choosing to follow an established habit.

Habit

A person can develop the habit of using the adrenaline system for some activities, and turning the adrenaline system off during others. For example, some people can relax deeply the moment they get home from work. Others only feel really joyful, turning off their fear, when they are singing or being creative. Others, including people with Parkinson's, tend to have a very narrow range of circumstances in which they dare to turn off the adrenaline systems.

Of course, this description is grossly oversimplified. When both systems are running, which is the norm, adrenaline is being released for some activities and dopamine is being released for others. For example, in a normal person, there is always some amount of adrenaline being released into the breathing system. If this adrenaline pathway is shut down, a person will stop breathing. Therefore, if a person is feeling deeply relaxed in other aspects, he will still have some adrenaline in the breathing system.

Adrenaline above and beyond the levels necessary to maintain certain life-sustaining fear-based activities such as breathing can cause adrenaline prioritization of the flow of blood and energy throughout the body.

For example, a person who is eating but whose thoughts are worried will have excess adrenaline in his system. Even though this person may think that he is physically relaxed, adrenaline in the thinking pathways causes a diminution of activity in the digestive tract. Digestion is a lowest priority function when excess levels of adrenaline are in place. Therefore, because the adrenaline levels in the total system are causing a decrease in blood to the stomach and an increase in blood to the skeletal muscles, the dopamine levels in his digestive tract may not be optimal.

Summary of the one-or-the-other relationship between adrenaline and dopamine

Again, activation of the adrenaline system, regardless of the level of adrenaline, turns off the release of dopamine. Even when a person is in a condition of adrenaline exhaustion, if he is fearful, if he is choosing the adrenaline system, he will not be able to access the dopamine system. As long as an adrenaline system is the system of choice *or* if

there is excess adrenaline floating around in the area, dopamine will not be released: the dopamine system will just sit there, unused.

However, in the opposite situation, excess dopamine in the system, from drugs, for example, does *not* turn off the release of adrenaline.

The dopamine system is the default system; it potentially can run all the time. The dopamine system runs when everything is normal and healthy, and there is no fear. The onset of fear triggers the adrenaline system and an appropriate level of adrenaline for the situation. The release of dopamine into the dopamine system is then turned off. The dopamine system is stymied to the extent that the adrenaline system is turned on. One does not need to turn the dopamine system on; it is on automatically to the extent that one is fearless.

Adrenaline-blocking and dopamine-enhancing drugs

When the adrenaline system is turned on, as it is in Parkinson's, flooding the body with dopamine-enhancing drugs does not turn the adrenaline system off; the drugs cause both systems to run simultaneously. This is what happens when PDers take dopamine-enhancing drugs; dopaminergic drugs *mask* some of the symptoms of adrenaline exhaustion. The adrenaline system is still turned on full blast.

However, there are certain drugs that can sedate the adrenaline system. Anticholinergic and antihistamines somewhat sedate certain parts of the adrenaline system: the muscle and immune sections, respectively. However, in a PDer, this turning down of adrenaline does not turn the dopamine system on; the dopamine system in PDers has been electrically turned off at the source.

Marijuana turns off parts of the adrenaline system: the digestion, pain, and mental processing arenas of the adrenaline system are turned off in response to this herb. In healthy people (non-PDer,) this allows dopamine to flow in these areas. This accounts for the surge in appetite, decrease in pain, and a somewhat realistic sense of joy while under the influence of marijuana.¹

The effects of the drugs and herbs that turn off the adrenaline system in various arenas are slightly different from the effects of the dopaminergic drugs. Dopaminergic drugs include cocaine, alcohol, nicotine, methamphetamine, the opiates, and of course, the current antiparkinson's medications. The dopaminergic drugs do not turn off the adrenaline system, they simply mask the adrenaline symptoms with symptoms of excess joy. Also, the dopaminergic drugs induce dopamine at unnaturally high levels.

Since these latter drugs only mask adrenaline, an appetite suppressant, they cannot increase the appetite. Appetite is suppressed if the adrenaline system is employed. Because the levels of dopamine are unnaturally high, the illusions of joy under the

¹ Because marijuana very possibly turns off adrenaline rather than supplying dopamine directly, marijuana may be less neurologically addictive than the drugs that directly enhance dopamine. Addictive in this sense means that the amount needed to produce an effect increases with usage. Marijuana is not neurologically addictive in this sense. However, marijuana may still be emotionally addictive even though the need for ever-increasing dosage does not occur.

influence of dopaminergic drugs are even more distorted than they are under the influence of the drugs and herbs that reduce adrenaline.¹

Dopamine in the various arenas

What happens when the adrenaline systems are turned off in various arenas and the dopamine pathway is allowed to dominate? The following section will list these changes. I will only describe what happens when dopamine is flowing at the higher levels. I will not list the ways in which dopamine functions at low, medium and high levels. By describing how dopamine works at the highest levels, the reader can extrapolate what happens when the dopamine system in any given arena is only partly topped up.

Again, in order for the dopamine system to be on, it is *not* necessary to create a background of love or joy. That background already exists. The dopamine system, and dopamine itself (in a healthy person), is already up and ready to go at all times; it is only *inhibited* to the extent that adrenaline systems are being used instead.

A spiritual analogy is that one doesn't need to find joy; one only needs to stop being scared. More to the point, one doesn't need to find God: one only needs to stop avoiding Him. God, like the dopamine system, has always been there right along.

Speed of thought

Along the dopamine neural pathways, the speed of thought processing can be leisurely, integrating both sides of the brain. The thoughts find themselves pondering, daydreaming, and creatively combining information from both its logic and aesthetic sides. Thoughts developed using primarily dopamine may include information from memory and feelings of the heart. The thoughts may be directed by the feelings of gratitude, joy, humility, loyalty, or devotion.

During deep meditation, when the adrenal pathways are shut down and the dopamine pathways maintain homeostasis of the body through quieting all the body processes, one can hold the mind still on one thought at a time for as long as is desired. It is as if time ceases to exist – just the opposite of adrenaline, when time seems to expand.

But quick knowledge is possible via the dopamine system: when using dopamine, the intuition, no longer shrouded by the adrenaline system, is exposed. Wisdom, the knowing of what is right via the intuition, is instantaneous. The process of creatively processing thoughts with dopamine may be languorous.

At the highest levels of dopamine flow, thoughts are not directed towards self-preservation: when intuition is supremely dominant and no fear is present, there is no self-preservation instinct. A person in this state intuitively perceives himself as one with everything: there is no “self” to preserve.

¹ This section on drugs is extremely superficial. For more details on the mechanisms of the various dopaminergic and anti-adrenaline medications and supplements, please read Walton-Hadlock, JL, *Medications of Parkinson's Disease or Once Upon A Pill: patient experiences with dopamine-enhancing drugs and supplements*. Parkinson's Recovery Project, 2003. This book is available for free download at www.pdrecovery.org.

Motor function

Motor function using dopamine is pleasant. The body moves, not so much because the mind commands it to do so, but because of the sheer joy that comes from having a body, from being alive in every vibrating cell. Motor function using dopamine feels very different from motor function from adrenaline. Some recovering PDeers have described the novel – to them – sensation of moving via the dopamine system, with naturally occurring dopamine, in this way: “It was like a miracle. It was like brain waves taking form as movement.” Or more straightforwardly, “I was sitting on the sofa and I thought about getting up, and suddenly, I found that I was standing up! I didn’t have to actually think about what I was going to do, I just thought about the *idea* of doing it, and it happened.”

Motor function from dopamine feels as if brain waves are manifesting themselves as movement, with no middleman of grim determination or stern resolve required, and no self-critic watching from a distance making sure that it is done correctly. Dopamine movement is manifested in the way that a cat stretches, or the unselfconscious movements of a child, or the brilliantly fast reflexes of a joy-filled athlete.

“Dancing with Glen” – an aside

If you will forgive me a short aside, I will share with you a quick story that makes the point. A member of our PD Treatment Team shared this story with us. She had been a dancing instructor for years and years before she became an acupuncturist. She has an expression she uses for describing how our recovering patients move. When they suddenly start moving with dopamine, she says they are “dancing with Glen.”

“When I was a ballroom dancing instructor, I had, for several years, an instruction partner who was the best dancer I’ve ever known. Everyone loved to dance with Glen, both men and women; he was ambiguous in his sexual preferences and had many gay friends. But gender had nothing to do with it; it was all about the music.

“When Glen danced with you, it was as if there was no gravity. There was no labored thinking, no analyzing. If you just surrendered to the music and trusted Glen to lead you, you could dance forever, floating on air. It was pure joy, directed by music, expressing itself as movement.

“But Glen would only dance with you as long as you trusted his lead and followed him. If you started thinking consciously about what you were doing, if you started trying to remember logically which foot should come next, which direction you should spin in, Glen would immediately sense the change in the dynamic and would promptly walk you to the side of the room and pick up another partner.

“As long as you were willing to trust him and fly in time with the music and the spirit of the dance, he would keep dancing. The moment you started to have logic or doubt enter into it, he would know it, and stop dancing with you. He always said he simply couldn’t dance, really dance, if the partner was distracting him with mental stuff.

“I loved dancing with Glen, of course, so I worked at training my thoughts. But the more I tried to discipline them so that they wouldn’t show up, the more they intruded, and Glen always knew. So I had to train myself the other way, by training myself to choose trust. Not so much choosing trust, but *being* trust. When I surrendered my logic and just flew with my heart, time ceased. I could have kept dancing forever.”

Moving with uninhibited dopamine, with the adrenaline system turned completely off, is like Dancing with Glen.

Sensory perception

Dopamine system pathways to and from the senses connect more with the memories and creative processes than they do with the emergency warning systems. They go more to the sides of the brain and not so much to the primitive centers at the core. When using the dopamine system's neural pathways, smoke from the barbeque might evoke memories of long summer evenings and cooking outdoors. If the adrenaline system pathways are in use, the smell of smoke might evoke the response, "Did I remember to recharge the fire extinguishers?"

Pain

The dopamine system's network of nerves perceives pain not necessarily as a horrible thing, but as something to be dealt with. When a person is most fully engaged with the dopamine system rather than the adrenaline system, pain is perceived as a helpful signal. The pain signal is acknowledged and removal of the pain source is initiated. This might mean taking one's thumb off a hot stove or it might lead to gently stretching and bending a foot with a cramp in it.

In extreme dopamine mode, pain is perceived impersonally, with no negative connotation at all. Fire walkers and people in ecstatic trance often experience events that should, in a normal person, result in pain. However, if the dopamine system is fully operational, the body might not respond to the pain, and the mind may transcend it.

Transcendence versus denial

Note carefully here the difference between transcendence and denial. A person with Parkinson's might have been in denial about receiving an injury, and therefore registered no pain in response. His adrenaline system's neural pathways will support him in this: by cleverly keeping the neural paths on the "Emergency" circuit, the normal pain recognition process is bypassed and the pain is put in a holding tank, to be dealt with later.

A person of even-tempered mental control and wisdom, on the other hand, experiencing a situation that would ordinarily be considered painful, might be able to understand the universal electromagnetic forces at work in both his body and in the pain-inducing action, and even the greater dynamic of ultimate cause and effect.

By focusing on the light and electricity behind the atoms and molecules of the body, one might see the painful action as occurring, not to the ego-identified body, but to the body that is a physical manifestation of one's thought waves. A person of supreme presence of mind can simply restore the thought waves that constitute body awareness back to the correct pattern, so that the interaction of the body and the pain-inducing event is not a harmful one. This is the opposite of denial of pain, this is transcendence of pain.

True transcendence is not an activity that we see frequently, but it is one that the great saints and sages can employ when performing healings on themselves and others.

Suppression of pain does not equal spiritual superiority

Some PDers imagine that they are somehow emotionally, mentally, or even spiritually superior when they deny and suppress their pain. The true man of wisdom, however, acknowledges all events and intuits the wisdom behind them, or at least that there is a wisdom, however unfathomable.¹ He stands unmoved amidst the crash of breaking worlds, not because he pretends it's not happening, but because he perceives the perfect love and trusts in the ultimate perfection that hides behind the movements of the swirling cosmos.

On the other hand, if a truly wise man does feel pain, he expresses it. An example of a great soul expressing his pain is found in the gospels of the New Testament. In response to the sisters' sorrowful mourning of their beloved, recently deceased Lazarus, "Jesus wept."

I am reminded of another example of this principle. The close disciples of Saint Francis observed that sometimes Francis would go for long periods of time without eating. They tried imitating what they thought was his tremendous self-discipline. After the disciples went a few weeks, suffering pangs of hunger, Francis asked them what they were doing. When he was told that they were trying to imitate him, he laughed at them good-naturedly, saying, "If I was hungry, I would eat. Go, feed Brother donkey (the body)."

Immune system

Under the influence of dopamine, illness is less likely to occur. When one is feeling peaceful, or radiant with joy, he is much less likely to become ill than one who is feeling out of sorts and disgruntled. In the case of illness, both high levels of dopamine and high levels of adrenaline keep an illness at bay, and low levels of both make a person susceptible.

But unlike the situation with adrenaline, in which the body holds off fighting an illness until such time as the adrenaline winds down, a person who is flush with dopamine is not postponing the illness until a later date. Instead, his well-tuned immune system can work with an exquisite accuracy so that pathogens are killed off with few obvious signs of ongoing illness.

Digestion

In high schools, students are taught that adrenaline regulates the sympathetic, "fight or flight" system. They are also taught that the opposite of this system is called the parasympathetic, or "cud chewing" system. When the parasympathetic (dopamine

¹ I heard an unsubstantiated report that India has the world's lowest incidence of Parkinson's disease. A national characteristic of the people of this great country, home of the world's most sophisticated and profound philosophies, is their expressiveness of their emotions. Though I generalize here, the people of India are known to readily laugh, cry, and openly express their joys and sorrows. Some westerners regard their behavior as "childlike," even while crediting them with an advanced degree of compassion and spiritual depth.

It is curious to note that the British, known for their stiff upper lip, have a high rate of Parkinson's disease. This is particularly significant because, for over a century, many British and native Indians lived in the same Indian environment, sharing many of the same external influences. If this report of low incidence of Parkinson's in Indians can be proven, it suggests an interesting direction for future epidemiological studies of historical data on PD.

system) is turned on, the neural signals in the eating arena instruct the stomach and digestive tract to report for duty. The best digestion occurs when the dopamine system is dominant in a maximum number of systems.

When the adrenaline system is turned off, the appetite increases. Many PDers lose weight when they begin taking dopaminergic drugs. Despite the dopamine, their appetite does not increase. This is because their adrenaline system is still turned on. The drugs merely mask the situation in the adrenaline system, they do not turn it off.

Temperature regulation

Under the influence of high dopamine, one does not notice temperatures. Like with the pain and the immune system arenas, high levels of either dopamine or adrenaline render one oblivious to temperatures. However, when the dopamine system is working well, one does not notice the temperature because the body is automatically doing whatever it takes to maintain homeostasis. Temperature regulation with dopamine is done with elegant use of the body's resources so that a person does not get too hot or too cold, nor does he notice the temperature.

Examples of this are young children at play, who never seem to notice the weather as long as they are laughing. Another example is the way in which a person might not notice the cold wind strafing him when his clothes fit like the paper on the wall, his haircut turned out just right, he got the new job he was hoping for and the woman who owns his heart has just agreed to marry him. In a case like this, he might be singing in the rain, but he won't get a chill. His dopamine-saturated dopamine system will see to it that his internal body temperature is regulated properly, making automatic adjustments to compensate for the weather.

Social Interaction

The dopamine system's neural pathways for social activities connect the dots between social cues, memories, and logic. This integration makes possible the correct interpretation of a smile, or remembering – and caring – about someone's birthday, and knowing what sort of gift would be perfect. Even public speaking to a room full of critical strangers, when performed fearlessly by the dopamine system, is a joy rather than a trial.

Logic versus intuition

Great scientific insight, poetry, and art, all work of heart-expanding beauty, is inspired through a high level of attunement with intuition. Intuition is at its most perceptible when dopamine levels are highest. In combination with intuition, which contributes joy, gratitude, humility and devotion, the dopamine system's neural pathways for logic meander through the brain, making connections between analytic thought, creativity, art, and memories. These connections and combinations can trigger the sensation of the chest swelling with joy, a sensation that we associate with love, and which expresses itself as art.

Breathing

When the dopamine pathways are in full dominance and all the adrenaline pathways are completely turned off, one can consciously stop breathing. Great yogis, Zen

masters, Sufis, and Christian mystics all practice devotion and/or practical techniques to still the restless, adrenaline-driven mind. Only in this way can the intuition, speaking through the language of the heart, answer the deepest questions of the soul.

As the adrenaline system's pathways to the lungs (diaphragm) and fear centers are shut off completely, the body reverts to its default state, using only the dopamine neural pathways. These pathways, when followed without any fear-based mental distraction, allow the cells of the body to remain quiet, alive but not metabolizing, and the breathing stops for as long as one desires. Verified accounts exist of yogis being observed in the breathless state.¹

Weeks or months later, they resume breathing. They suffer no physical damage during their deep stillness.

Gravity

Probably most interesting of all is the relationship between gravity and dopamine. I have already mentioned that, via adrenaline, one can combat the forces of gravity with super strength. But when the dopamine system for movement is completely saturated with dopamine at the highest levels, one no longer needs to combat the forces of gravity: one can fly.

Saints and sages of every faith have found that, when their thoughts are too saturated with love to be borne, they cannot keep their feet on the ground: they levitate.

If this example is too spiritual for you, let me get a little earthier. Michael "Air" Jordan, a supreme basketball player, earned his nickname because of his ability to soar into the air and hover there, even fly towards the basket, radiant with joy, while he lined up his shots. Magic Johnson got his name the same way. Other athletes have been known to perform "antigravity" feats. In athletics, this is called "being in the Zone."

One of my Olympic athlete patients (not a PDer) describes being in the Zone as what happens when the laws of physics disappear, the sun is on your back, and all things are possible. "That's why I swim," she says. "I swim for those moments when reality shifts, when there are no limits."

When one's wings are filled with such pure joy, one can perform feats that defy physics. At these times, breathing is minimal, coordination is effortless, and one can

¹ A study designed by French scientific researchers in the 19th century recorded the underground burial of Sadhu Haridas, a yogi who had agreed to put himself in a breathless state for two months. Two months later he was exhumed and his inert body was immediately pronounced dead. A few minutes later, his eyelids fluttered and he began breathing. He quickly resumed normal activities.

Zen master Shunryu Suzuki said, "Concentrate on your breathing and it will go away," alluding to this phenomenon. *To Shine One Corner of the World: Moments with Shunryu Suzuki, Stories of a Zen master told by his students.*

The great Christian mystic Paul attested "By the rejoicing that I have in Christ Jesus, I die daily."¹ Corinthians, 15:31. This mystic "death," during which the body becomes motionless and breathing ceases, is the result of the supreme peacefulness and joy that is felt when, during God-attunement, all fear-related thoughts, even the fear of death, are turned off.

Mystics of every faith speak of this breathless state, the deathlike stillness of blissful union with God. An Islamic mystic, the poet Rumi, wrote in this conversation with God (in my source book God's voice is italicized): "I would love to kiss you. *The price of kissing is your life.*" (*The Essential Rumi, Translation by Coleman Barks.* Castle Books. 1997. p. 37.

In the Jewish faith this relationship between utter cessation of body function and attunement with the Divine is simply stated in Psalm 46:10, "Be still, and know that I am God."

almost hear the sound of the universe singing. Such a person can feel in his heart the answers to his questions, and he knows that he is love, that is he is loved.

Watching athletes when they are in the Zone confers an almost breathless joy onto the fans. The same applies when truly great singers fearlessly soar up to the high note, or sing from their wide-open soul – the audience feels “uplifted” on the shared wings of the singer.

Swimming lessons – a story

Writing here about the swimmer reminds me of a wonderful example she gave me of how to separate the adrenaline/fear people from the dopamine/joy people.

“I used to teach swimming for little kids,” she said. “On the first day of lessons, the kids hang on the side of the pool and practice blowing bubbles in the water or kicking. All the kids can do that. The next day is when they separate themselves out. I tell the kids that they are going to float. I tell them that the water will hold them up. They won’t need to do any work. I tell them that they just need to lay back, spread their arms and legs, and float on the water.

“Some of the kids get sparkly eyed and happy, saying, ‘The water will carry us? Oh boy!’

“Other kids grit their teeth and I can just see that they are thinking, ‘The water is going to hold me up? What? No way will the water hold me up. Maybe the other kids, but not me. I’m going to have to work like a beaver to keep from going down!’

“So then I get them started floating. The kids with the faith and wonder lie back on the water and float, their faces beaming. The scared ones start pumping their arms and legs. Thrashing, kicking, beating at the water, they do everything they know to force themselves to stay on the top. And they go right to the bottom like a stone.”

This reminds me of another story, a story of the great ballet master, Nijinsky. He could take leaps that defied all the laws of physics. Like Michael “Air” Jordan, he could simply fly through the air. The orchestra director had to hold the note, watching him, and only resume the flow of the music when Nijinsky came down.

He could not perform those leaps, however, if he was dancing with a partner. The partners’ negative thoughts always pulled him down. There was only one person he could dance with: his wife, a skilled ballerina. However, even she would keep him bound by gravity unless he first hypnotized her. Only after she was hypnotized into a state where she was unable to doubt his magic could he then perform his flying, lighter-than-air leaps with her by his side.

Parallel pathways: a summary

The brain has two neural systems that regulate thought, metabolism, and perception. These two systems, the adrenaline and the dopamine, are somewhat parallel. They regulate all the same functions, but they do it with different motivations, different neural connections, different fields of emphasis.

They both regulate speed of thought, pain awareness, temperature regulation, hunger and digestion, the immune system, time perception, and breathing. All of these body functions are necessary for the maintenance of life. The manner in which life is maintained, one might say the quality of life, depends on which pathway is used.

The trite old freeway metaphor

If these two pathways were roads, they would start and end in the same place (“being alive”) but one would be a superfreeway with no speed limits and only a few specific exits. The other would be a rambling road that had different scenery every time you traveled it. The freeway route would get you there faster, and when you arrived, you’d be keyed up with fear and pride. The slow route would fill you with joy and gratitude. As for how long the slow route might take, it wouldn’t matter: with the beautiful slow route, there is no sense of time.

If we wanted to get poetic about it, we might say that the dopamine route is that Road Not Taken, the road less traveled by, that we read about in high school.

As you can deduce from the above, a happy and healthy person uses primarily the dopamine systems. He only shifts over to the adrenaline pathway and lets loose with elevated adrenaline levels, with a concomitant closing off of the dopamine pathways, when a condition of high stress or emergency is occurring. As soon as the emergency is over, the adrenaline levels, in a healthy person, climb back down and dopamine goes back up.

Ideally, as the dopamine and adrenaline system usage fluctuates back and forth, their respective neurotransmitter levels adjust automatically up and down as well.

And don’t forget: a person can be using both of these systems simultaneously. Most people simply use the system that corresponds to whichever activity and mood is dominant at the moment for a given arena. Someone might use the adrenaline system for his eyes, ears, hands and feet while driving, even while he is laughing merrily, using the dopamine system to choose his words and move his lips as he converses with his best friend in the passenger seat.

Only people with extraordinary mind control, such as saints and PDers, can will themselves to use primarily one system or the other.

Wrapping up the adrenaline/dopamine section

The mind-body relationship is altered when adrenaline is dominant: under the influence of high adrenaline, the ego- or fear-laden mind is the unquestioned master of the slavishly obedient body. Under the influence of very high dopamine, a unity of body and joyful intuition is maximized, while the mind serves as the obedient switchboard, monitoring and controlling the flow of incoming and/or outgoing sensations to and from the consciousness.

In normal people, dopamine is dominant during eating, enjoying pleasant company, dancing, and doing sports while in “the Zone.” Adrenaline is dominant during driving, interfacing with testy strangers, combat, or performing extreme sports.

How does this relate to Parkinson’s disease?

Most people with Parkinson’s, especially those who develop Parkinson’s in their early years, prior to their mid-seventies, have an extraordinary level of mind control from an early age. Due to fear, they use this mental control to choose the adrenaline system as their perpetual system of choice.

In people with Parkinson’s, the adrenaline system always dominates a majority of activities. Until energy begins to flag due to aging or adrenal exhaustion, the adrenaline

levels are kept as high as possible. This high level of adrenaline and reliance on the adrenaline system corresponds to, “I’m injured and I can’t deal with my emotional and physical insults and injuries right now; I’m in danger.” The brain’s dopamine system, for the most part, is in perpetual hibernation.

Even if this pre-PDer consciously tries to turn off his fearful thoughts and relax, it is very difficult to do: his lack of dopamine availability (an electrical, physiological pathology discussed in chapter three) prevents the normal feelings of joy from rising up even though fear is absent. The normal reversion to joy simply doesn’t happen. Instead, the pre-PDer finds, over time, that to keep his blood stirring and his mind focused, physical and mental restlessness is a more likely source of reliable stimulation than the deep inner joy that eludes him more and more.

Let’s look once again at the PD development sequence. First, a person’s fear-based will power, self control, and personality determined his ability to ignore his injury and ignore his fear, and maybe even ignore his whole body. Over time, when the dopamine signals in the brain got shut down via the electrical resistance/backwards flow, the physiological deficiency in dopamine began to direct his behavior, his personality: since dopamine was not available, he chose more and more to create a sense of urgency and restlessness to keep himself going. This in turn triggered the release of more adrenaline, the neurotransmitter that is associated with danger and fear. His fearfulness increased, ultimately, because of the injury, which in turn had been retained because of fear.

In the PDer, the interconnectedness of mind and body has congealed perfectly, as it always does, to create a body and behaviors that reflect his thought patterns. In his case, those thought patterns are usually powerfully focused, desirous of helping others, negative with regard to his own worth, and based on fear rather than joy. Over decades, despite his best efforts to hide his emotions behind his actions, his body will proclaim them: his body will begin to resemble the shrinking, trembling body of a person who is, deep inside, too frightened to move. If his trauma was profound enough and he lives long enough for the PD symptoms to become advanced, his trembling rigid body may begin to resemble that of a person in shock.

SUMMARY

The adrenaline-dopamine relationship is a cornerstone upon which Parkinson’s, a neurotransmitter-related illness, is built. One might want to say that in the absence of dopamine, a person must use adrenaline. However, we think it is more apropos to say that, when one chooses to use a fear-based model for living one’s life, making adrenaline dominant, dopamine levels must necessarily decrease. When, through electrical confusion set in motion by a foot injury, the dopamine unavailability eventually transmutes into cellular change and dopamine availability is no longer a matter of choice, this transition is merely solidifying a choice that was already made, long before. I propose that the sum of the fear-based attitude, the injury, and the sequelae of both, constitutes the energetic blockage that causes Parkinson’s disease.

Our in-depth meetings with hundreds of PDer, in which we discussed personality, responses in all the various arenas, and attitude, provided thousands of clues that suggest that an incorrect adrenaline-dopamine relationship is at the bottom of the

Parkinson's disease problem. We will need to meet with thousands of PDers before we can be certain.

But until that time, we can say this: all of the PDers that we've asked have all laughingly admitted that, when they tried to learn to float, they sank like a stone.



“A bad habit never disappears miraculously; it’s an undo-it-yourself project.”

- Abigail Van Buren (author of the advice column “Dear Abby”)

APPENDIX II

HEART-SHIFTING TECHNIQUES

This appendix is a collection of techniques for reinforcing the heart-mind connection and for shifting the predominance of one’s internal dialogue from negative to positive.

This collection is not linear: one needn’t go through this collection methodically, practicing all the techniques in the order presented. Instead, read all the suggestions in this and the next appendix. If one or more of the techniques makes sense to you, try them.

Breathing techniques

Observing the breath for a few minutes and/or taking long slow breaths can be very helpful at shifting the mood from frantic to calm.

Hundreds, if not thousands, of breathing techniques have been written up over the past few millennia, especially in Asia.¹

Most of them involve sitting up straight. Most of them make the point that, during an inhalation, the abdomen should expand outward, the chest should expand outward, and the shoulders should rise up a bit, allowing the maximum amount of air into the lungs. During exhalation, the abdomen should, if possible, move gently in and up, the chest might move inwards and, if it is comfortable, the shoulders can drop down.

Other breathing techniques do not dwell particularly on the physiology of breathing, but rather ask the breather to simply “observe” (feel) the sensations or rhythms associated with the incoming and outgoing breath without trying to regulate or control the rate or volume of breathing.

Another technique is to imagine the feeling that the incoming breath is a stream, or waterfall, flowing down, down, down into a chasm of great depth and beauty. The outgoing breath is another stream of whatever seems appealing: mist or steam rising up out of an almost infinitely deep gorge.

Yet another breathing technique asks the breather to inhale while counting to a certain number, then hold the breath for the same number, and then exhale to the same count. It doesn’t matter what the count is except that it should be comfortable. While doing this, he should notice how he feels throughout his body when his breathing is measured.

Still another suggests that a person feel the breath while making no attempt whatsoever to control or regulate the breath. This focus is non-judgmental. The focus should not be on the previous breath or the upcoming breath, and never wondering “how much longer or shorter should this breath be?” The point with this technique is to practice being in the present, in “the now.”

¹ When confronted with the word “Asia” most Americans think primarily of China and the countries along the western Pacific rim, but most citizens of the British Isles think primarily of India and Pakistan. When I use the word Asia, I am referring to both groups of countries.

These various breathing exercises can be powerful assists in learning how to stop the internal monologue and allow awareness, rather than words, to be dominant in the mind. Don't try to do all of them; find one that appeals to you and practice until you begin to realize how difficult it actually is. After you've realized that, then work at it until you master it.

As for which of the above techniques are the most effective, I refuse to say. To the experienced eye, they all look the same. They can all turn the mind's focus onto the experience of feeling the breath. By focusing on feeling, one turns off the critical voice.

Going through the motions: misunderstanding the techniques

No matter how powerful and effective these breathing and other techniques can potentially be, they can always be sabotaged by a person who is determined to stay in defensive mode. Consider a person who perfunctorily does one of the above breathing exercises, methodically counting off the seconds of his "wasted time" while doing the exercise, and wondering all the while whether or not the technique is "working." He might as well not even do the exercise. He will do the deep breathing, and when he is finished, he will brush his hands as if to say, "Thank God *that's* over with...am I better yet?" When he realizes that the brief moment of slow-breathing-induced calm has already disappeared, he might even say to himself, "Hah! It didn't last. I knew it couldn't be that easy."

A wiser person, one who actually understands the point of this, and all the other techniques, will have a different approach to going through the motions. He will understand that this technique, like all attitude-altering techniques, merely provides a structure within which one can practice noticing how he feels: he can pretend, for a little while, to be gently cradled in the bosom of love. In this pretend state of being not only removed from all worldly cares, responsibilities, resentments and anxieties, but proactively loved and supported, the latent, usually beclouded or veiled peacefulness of feeling, of pure consciousness and intuition, is able to rise to the surface of awareness. The state thus attained is a state of awareness and pure feelings rather than a state in which streams of words are relentlessly evaluating anything and everything.

For people who meditate regularly and correctly, the joy of this wordless state is the motivator, the positive reinforcement, that encourages one to do the self-changing work that is necessary to – guess what – *increase* his ability to remain in this pleasant state during subsequent meditations and even during his busy day. If, on the other hand, these exercises are done with an eye on the clock and a relentless word-based consciousness of "How'm I doin'?" Sheesh. Am I done yet?" the entire point of the exercise will have been lost.

Seeing faces in the clouds

Again, as noted earlier, word-based thinking is nearly always a fear-based director of judgmental, adrenaline-dominated consciousness. Wordless awareness, in comparison, is not even, technically, thinking. Awareness simply perceives what is happening and, if appropriate, lets the heart decide whether or not the perception needs to be acted on. For the reader who is completely baffled at this point as to what I mean by awareness as opposed to mental dialogue, a quick example might be helpful. Most PDers recall that, at

some point in their childhood, they were able to look up at fluffy clouds in the sky and see them forming into sheep, ships, and shape-shifting faces.

This “seeing” was never a word-based process. This pleasant “seeing” of imaginary shapes is an emotionally joyful act of playing with awareness. Many PDers are stunned to discover, as a part of their recovery process, that they are once again “seeing” faces in the leaves of trees and the clouds are once again forming fantastic shapes. There is no income-generating, efficient purpose in the mind while it is playfully forming these images, and, yet, even the most “practical and logical” PDer finds, during recovery, that seeing these images is accompanied by simple joy.

Feeling safe and seeing faces

The reader who is reading between the lines of the breathing exercise will know that the exercise is a framework in which he can stop what he is doing for a few minutes and enjoy focusing on his sensory awareness in a wordless sort of way.

The experience of feeling peaceful or even feeling loved, even if induced, at first, by sheer pretending, will be similar to the experience of seeing faces in the clouds. It’s a simple, gentle recognition of the mind’s capacity for awareness of self and Self.¹

The fear-drenched reader who is determined to “do these techniques and exercises correctly” will want to know exactly how long to hold each breath, should he make a noise with his breathing, should he breathe through his mouth or his nose, etc., etc. The answer to these questions is “none of those things matter.” What might matter is one’s ability to pretend that, when he sits down to do a breathing technique, all the cares of the world have ceased. He can even pretend he has died to the world. (This is actually very helpful! It can be refreshing to realize anew, every day, that, the moment we die, all our worldly cares will cease and, no matter how important we think we are, everyone *will* find a way to go on without us. Why not practice for this relieving inevitability?)

In the pretend life-stoppage or sense-of-time-stoppage that can occur during the breath observation exercises, one can wordlessly observe the breath. Now, a person might choose to visualize some pretend image or sensation that accompanies one’s breathing – something fun and beautiful, something creative and loving. That’s fine too.

Or after while doing some breathing techniques, why not notice how the heart is feeling? Is it in your throat? Heavy? Broken in two? If your heart doesn’t feel quite “right,” why not spend a moment gently talking to it, asking it what it wants, and treating it the way that you wish you’d been treated as a child.

Then again, if I include something in the instructions to the effect that visualizations during the exercise are a good idea, a fear-based person who lives his life trying to “follow the rules” will most likely counter this with “But I don’t like to pretend or visualize,” or even “I can’t do that kind of stupid stuff.”² And then he might not even

¹ The capital “s” Self is a writer’s convention: “Self” signifies the greater self, the superego, superconsciousness, the individual soul, and sometimes, the larger manifestation of soul: the universe or even that which created the universe. When one gazes at the clouds and turns them into sheep and ships, he is playing games with creation: the self is playing games with the Self.

² Many PDers have convinced themselves that pleasant activities are a waste of time. I am frequently told by really stubborn PDers something to the effect that all of the things that contented people do are “stupid.” It is tempting to point out that at least these “stupid” people haven’t created so much fear avoidance that they can’t even walk. It does seem to me that wallowing in emotional rigidity to the point of

try to do the exercise. And since the very point of the exercise is exploring where your unique heart takes you when the worries are turned off, the very act of my suggesting what to do with your heart in your silence is bound to be incorrect and misleading. *I can't know where your heart will take you. Try the exercise with a childlike wonder, and see where you end up.*

The very essence of all of these techniques is turning off the fear of being judged and turning on the awareness of omnipresent love. However, the people who most need to do this may, quite likely, perform all these techniques as if the performance is being judged or measured – thereby inhibiting the joy. Therefore, as I have mentioned, writing up these exercises may very well be an exercise in futility. The very people who need to do these techniques are the ones who are most determined to do them correctly, but the whole point is that there is no such thing as doing them *correctly* – the essence of the exercises is doing them as if they were fun.¹

I think this quote from Sir James M. Barry, the author of *Peter Pan*, puts it very well: “You must have been warned against letting the golden hours slip by; but some of them are golden only because we let them slip by.”

Moving right along, now let's look at a technique that does the opposite of silencing the nagging mind: a technique that awakens the positive mental voice.

The technique of chanting

Chanting, the steady repetition of a word or phrase, silently or out loud, is a way to gently and deliberately retrain the consciousness. By chanting, one trains the unruly, undisciplined mind to move in the direction of your own choosing. The technique is extremely powerful.

For those PDer's who think they have strong mental faculties, I suggest that they try to chant one gentle or loving word for a solid minute, holding the mind on that one

becoming physically rigid is also a bit on the “stupid” side. When I was a child, this was called “cutting off one's nose to spite one's face.”

Recently, I was reading *The Ardent Birder*, by Todd Newberry, PhD in Biology and professor emeritus at University of California, Santa Cruz, and I was laughing so hard I nearly fell off the sofa. The text is *not* intending to be broadly comic, it is gentle and sincere; I was laughing because I was imagining how most of my PDer patients would respond to this book. I'll describe and paraphrase this book briefly. The book tries to describe the joy of sitting in an overgrown field for hours, hoping that a bird will come along. The writer points out, over a pleasant, meandering ten pages, that there is pleasure if a bird shows up. He even suggests that one go with a friend once in a while, thus doubling the pleasure, should it occur. The writer further bubbles that, at those moments when a bird stares at the birder who is staring at the bird, “it is life touching life.” I can just imagine the frustration and even anger of a PDer who has been assigned to read this pleasant and sincere discussion of why it is important to “unexpected the expected” while birding. “What's the point?!” screams the PDer to the absent author. “Where are you going with this?!” “Why am I wasting my time reading this?!” “I have better things to do than sit in a meadow and hope that a bird comes along!!!” “I have better things to do than read a book about the pleasures of a thermos of cocoa!”

¹ My colleague, Chris Ells, shared this story with me. A fellow Tai Ji student had been hired to teach a Tai Ji class. Due to polio, this student's right leg was considerably shorter and weaker than his left, so he always skipped over the one-legged portions of his Tai Ji form. When he got the teaching job, he asked his Tai Ji teacher if it was OK to skip the one-legged parts of the sequence, since, “If I do the one-legged parts, I will fall over.” The teacher said to him, “Then fall over.” The teacher's point was that the essence of the exercises (joy) had nothing to do with the skill of execution.

thought without being distracted into other thoughts. These “strong thinkers” are often amazed at the actual lack of control that they have. As long as their mind is looping over and over in some ego-prized memory of self-pity, anxiety, or obligation, their mind may be able to stay in a particular arena indefinitely. But they find that they cannot keep the mind steadily on a subject of their own conscious choosing for more than a few seconds. Even a few attempts at this mind-disciplining practice will show one why all great men, from Alexander the Great to the great saints and sages, have agreed that taming of the mind is the most important and most difficult undertaking that a man can pursue.¹

To practice chanting, choose a word or very short mantra, or phrase, and repeat it whenever you are not using your mind in a specifically productive manner. That’s the whole technique. How hard can it be? (Answer: very hard.)

Practically speaking, when you are driving the car, talking to someone, or adding up a long column of tricky numbers, you might not want to be silently, in a focused way, repeating your chant. But if you are eating alone, resting, working in the garden, bathing, dressing yourself, cleaning the house, or performing most of the activities of daily living, you can be trying to keep your mind focused on repeating your chosen word or phrase.

The more famous mantras usually have a strong spiritual emphasis. Some classics are, “For God” (meaning, “Whatever work I am doing, I am doing it for God”), “I love you” (the word “you” in this context is directed towards the chanter’s largest possible understanding of the cosmos), “Divine Mother,” “For Love” (meaning, “I am doing this work for the Universal Love”) or simply, “You.”

The psychological process is obvious; a person who is saying “You!” with all the focus of his mind cannot simultaneously be saying “Me!” Since “me” is the wily demon-lover, the ego and its partner, fear, the constant repetition of the word “You!” can slowly, eventually, effect a change of mental orientation towards peace of mind and away from fear. And please, don’t go saying “That’s so simple! It won’t be hard to do,” until you’ve tried silently chanting for ten straight minutes while keeping the thoughts ever turned towards the subject of your chant. This technique is simple, but there is nothing “easy” about it.

Other suggestions for chants include Om-Tat-Sat (Hinduism), Om Mani Padma Hum (Sanskrit form of a Buddhist prayer, also known as Om Mani Peme Hung in Tibetan), Father-Son-Holy Ghost (Catholic and some protestant Christian religions), Allah Akbar (Islam), any of the many Hebraic variations on the name of God or a few words from a favorite Psalm, or, mixing and matching, one can use God-Christ-Guru, or anything that makes a person focus on something other than self. All of these ideas draw the mind away from the little “me” and focus it on the greater Self.

Because I receive many complaints about this technique from people who do not believe in God or follow any particular spiritual path, I want to point out that belief in

¹ Thayumanavar, “The Silent Sage” of southern India (1706-1744), wrote this poem:

You may control a mad elephant;
You may shut the mouth of the bear and the tiger;
Ride the lion and play with the cobra;
By alchemy you may earn your livelihood;
You may wander through the universe incognito;
Make vassals of the god; be ever youthful;
You may walk on water and live in fire:
But control of the mind is better and more difficult.

God is *not* a requirement. Many people, due to the cruel images of God that they learned in their youth, have a strong aversion to the word “God” or any organized religion, per se. Such people need to know that the phrase “You!” can be directed to Love, Wisdom, or the Force that permeates the universe and which sets the universal ball rolling. For that matter, it can be directed towards a beloved deceased grandmother, or any person who you think of as a great soul. The Mahatma Gandhi, Mother Teresa of Calcutta, and Martin Luther King Jr. are all very good subjects for “You!”¹

One elderly monk said to me that, when he began his spiritual seeking, he wasn’t sure what was meant by “chanting with his heart.” He wanted to speak to God with his heart and not his mind, but he wasn’t sure how. So he imagined a little mouth with lips on his heart; that mouth said the words.

By creating this image, he was inadvertently accessing the playful part of his mind – the part that releases dopamine. This simple act of imagination was bringing him closer to using his intuition and simultaneously shutting the door on potential negativity.

He told us that, using this image, his chanting increasingly came more from the heart and less from his brain. His heart was successfully able to open up to the joy therein.²

It’s the thought of the thought that counts

Again, as with the breathing and all the other exercises, the way in which chanting is practiced does matter. If a person perfunctorily repeats “I love you World” for a predetermined number of counts and then says to himself, “Thank goodness *that’s* over with,” or if he is mindlessly chanting “You, You, You” while multi-tasking his brain to

¹ A study done in 1985 suggests that people who do not credit anything religious or spiritual can still be powerfully affected by exposure to a “spiritual” stimulant. A group of Harvard students, many of whom were deeply cynical, even adamantly anti-religious, watched a movie about Mother Teresa. After the movie, their saliva was tested. Even students with hard-core anti-Mother Teresa sentiments had a change in their saliva: a sharp increase in IgA (an immune system component that can *increase* when a person feels good and which can *decline* with stress). This study was included in an article about scientific experiments measuring whether spiritual and meditative practices elevate (improve) certain immune factors. (The answer was “yes.”)

The study with the Mother Teresa movie was done by psychologist David McLelland, Harvard University, and reported in *American Health* magazine, July/August 1985.

² Many PDers have convinced themselves that they cannot create mental images. Can you see how many layers of difficulty these PDers have created for themselves? They will not be able to imagine a mouth on their heart – something a child can do. That’s another problem with literal mindedness and overly “sophisticated” thinking: it detracts from, does not add to, our innate abilities.

My favorite experience dealing with innate ability occurred when I was doing acupuncture on a nineteen-year old. She had brought her nine-year old sister along to watch this “new” form of medicine. As part of the treatment, I put needles in both ankles of the patient. When the treatment was over, I asked the young girl if she had enjoyed herself. “Oh yes,” she replied. “I especially liked watching the blue-green sparks, like electricity, going back and forth between the two ankle needles.”

Now, I have learned in my fairly advanced and arcane researches that the color for the particular type of Qi that I was accessing is “blue-green” or “dragon’s green,” and that the paired currents arc back and forth while coming to a healthier equilibrium. However, I have never seen it. This young girl, not knowing any better, was able to see it easily. Not knowing what to say, I merely agreed, “Yeah, that’s great.” All too soon, no doubt, sophisticated, “rational” thinking will begin to cloud her innate ability.

simultaneously remind himself that his mother was not affectionate or his father should never have remarried, while alternately worrying pointlessly about the economy, next year's weather, or the ailing home sprinkler system, he will receive *no* benefit from this technique. Such a person may arrogantly think that he is demonstrating terrific mental faculty because he is thinking of so many things at once. However, it would be more honest and accurate to say that the owner of such a mind is a victim of his undisciplined, runaway thoughts.

The whole point of chanting is to take the mind away from chronic immersion in the "I, me, mine" mentality and break the pathological cycle of endless worrying. For example, it is fine to plan one's day – once or twice a day. If the process of day planning repeats itself pointlessly, that's pathological. Focused chanting can regain some healthy control.

It is important to have some sort of direction as one goes through life, but chronic worrying about taxes, traffic, or "what is the other guy thinking of me" is pointless. Such ego-based, fear-based thoughts diminish the quality of one's life. If one has Parkinson's, or even if one has recovered from PD, such habits can do more than diminish quality of mental peace: they can be physically debilitating.

To be sure, such looping, impractical immersion in worries conveys self-importance to the ego and is therefore gratifying, but if a person has enjoyed this type of thinking to the point that he can no longer walk easily or swing his arms, maybe it's time to try a little change of mental direction.

St. Francis and Brother Lawrence

A more advanced version of simple chanting is to direct all of one's silent thoughts and awareness towards the "You." To perform this technique, one steadily directs his mind to acknowledge the Love or divinity in everything he touches, smells, hears, and in all his actions and thoughts. St. Francis of Assisi felt an affinity with all creation; he loved Brother Sun and Sister Moon. He beheld divinity and affinity in all things and actions. He did not *think* about God so much as remain ever aware of Him.

This technique is sometimes called "practicing the presence of God." It has been practiced for millennia in the East, and has been practiced by mystics of various faiths even in the West.

Brother Lawrence was an uneducated, 17th century French monk who, having no academic skills, was assigned to his monastery's kitchen duty. As he performed his daily chores, he focused on seeing God in everything around him, in the very stones of the floors that he scrubbed and in the water that he carried in from the well. He was constantly aware of and talking silently to the ever-present God. Eventually, he was able to say "The time of business does not with me differ from the time of prayer; and in the noise and clatter of my kitchen, while several persons are at the same time calling for different things, I possess God in as great tranquility as if I were upon my knees."

As an aside, I want to make it clear yet again that no one needs to become transformed into a saint or hero in order to recover from Parkinson's. A recovering PDer need not become a sage-like master. However, he must learn to, at least once in a while, float along with the universe instead of trying to control it. To this end, even the simple act of staring at the stars once in a while is a very worthwhile activity. It is hard to

maintain the idea of self-importance while gazing out across the Milky Way. Of course, a truly determined fear-lover can use the darkness of the infinite abyss as a source of terror. Still, gazing at the night sky might be a helpful technique for a person who is ready and willing to put his anxieties into perspective.

To summarize the technique of Practicing the Presence of God (or the Presence of Love, if one prefers), one should try to be constantly aware of the love or mystery or magic going on around him and be ever talking to it in the language of one's heart. Although this technique seems ridiculously simple, it can provoke a ferocious tug of war between the mind's long-term habit of negative stream of subconsciousness and the novel experience of the mind being guided, directed, by positive, lovingly disciplined consciousness.

When, eventually, through formation of new habits, the mind becomes more focused and guided by the intuition instead of by fear or negative emotions, the results can be profoundly life-changing. Both chanting and practicing the presence can, when practiced diligently and lovingly, eventually bring about an orientation towards sweetness and gentleness even in a heart that has long been disconnected or enmeshed in a trap of worries and concerns.

Re-tuning the heart radio

I first read about this yogic technique in a write-up of a lecture given in the 1930s.¹ The modern information about electromagnetic fields emanating from the heart was not yet available. However, this yogic technique for tuning out the “fear channel” and re-tuning the heart to a channel that broadcasts love is clearly based on the electromagnetic characteristics of the heart.

The technique is as follows:

Place the right hand over the heart. Gently draw the hand towards the midline of the body (towards the sternum) and then let the hand rest for a moment between the two breasts. Then, lift the hand off the chest and replace it over the heart. Repeat. (Repeat as many times as you like.)

While doing the hand gesture, say out loud or silently, “Tune out the fear in my heart radio.” Repeat this statement every time you move the hand across from the heart to the midline.

About this technique: notice that this technique does not try to pretend that there is no fear. This technique is not a top dressing that merely masks the fear. The power of this technique is acknowledging that the heart is facing in a direction that tunes in with fear. The affirmation and the physical movement both assist to redirect the receptors, the antennae, of the heart radio.

(It is important to note that this technique does not seem to work in people who are pretending that their hearts are not connected to their minds (see chapters 22 through 27. One who has historically been disconnected from his emotions might want to first make sure that he has “reconnected before spending much time on this technique.”)

¹ I found the text of this lecture, originally given by Paramahansa Yogananda (1893-1952) in, I think, the 1930s, in a late 20th century issue of *Self-Realization* magazine. I do not recall which issue of the magazine had this technique. I have since heard a yogic monk refer to this exercise; he said, “I know it sounds ridiculously simple, but it really works.”

Why it works

In Asian medicine, the line of energy that runs up the center front of the body from the pubic bone to the lower lip is called the Ren channel. Ren is often translated as “Conception,” and is considered to be related to fertility and reproduction. A deeper understanding of this channel is that it resonates with the creative electromagnetic signal that underlies the creative properties of the universe. This channel is considered to be “Yin,” materially creative or “feminine.”

The Ren channel is associated with the *physical* manifestation of the universe, the quanta, if you will, as opposed to the causal, or *thought-based* forces that keep the universe in play. The more physically extant forces in the body and in the universe are considered more Yin, the thought-based forces that drive creation and that provide individual consciousness are considered more Yang. These Yang forces happen to resonate with the line of energy that runs up the back of the body, the Du (translated as “Governor”) channel. The Du channel is an energy source that plays a major role in shaping the spine and brain and providing consciousness.

Getting back to the point of this exercise, the Ren channel resonates with forces of created matter. We can call this force Divine Mother, Quan Yin, Maha Prakriti, Mother Earth, Mother Nature. On a more human level, this is the force that has found embodiment in Mary, the mother of Jesus, and other saints – male and female – whose love takes primarily a nurturing turn. The universal materially-creative force (physically creative and nurturing, as opposed to the forces that generate the *concepts* of creation, growth and change) manifests, among other things, as an electromagnetic field that, in humans, resonates with the Ren channel.

Heart orientation

In an emotionally healthy person, the electromagnetic receptors of the heart are oriented towards this channel. A well-tuned heart is thus always receiving signals from the nurturing love that permeates and guides the material, corporeal, physical aspect of creation. This is the love that can be transmitted via the sensations: the bliss of standing on the ocean cliffs in a pouring rain, the invigoration from the smell of fresh sage, and the sweetness in the touch of a baby’s tiny fingers.

When fear is present to the extent that one loses his connection with joy, the radio-like receptors in the heart can rotate outwards, towards the front chest wall, pointing towards the source of the danger, instead of pointing towards the midline. For example, in the newborn child, the shock and fear of breathing and being in the physical world often cause the heart receptors to be rotated outwards, facing out through the chest wall instead of towards the Ren channel. This is why a mother usually holds her frightened child to her left breast; the infant’s outward-facing heart, thus positioned towards the mother’s heart, can receive the relayed nurturing signals of the Ren channel via the outgoing broadcasts of the mother’s heart. The mother, hopefully, has her own heart receptors turned towards the Ren channel. In this manner, an infant that cannot as yet control his own fears can have them assuaged by the relayed signals of his mother’s heart “radio.”

As the child matures, he hopefully learns inwardly to attune himself with the radio signals of love that are always present in the universe. Outwardly, he affirms joy by learning to sing, play, and relate to himself and others in ways that build upon the already

present love and contentment. These inner and outward manifestations of joy help keep the heart radio tuned towards the Ren channel, towards the joy that resonates through the cosmos.

Sometimes, it is necessary for the electromagnetic field receptors of the adult's heart to reorient and face outwards temporarily. When danger is imminent but the brain is asleep at the wheel or preoccupied, the intuitive heart may still be able to sense that danger is lurking. In such times, the heart receptivity can be on guard, watching for signs of danger. However, after consciousness has been alerted to the danger, the heart should revert back to the position in which it faces the midline of the body.

Sometimes, a person who is overwhelmed by or chooses to live in anxiety or fear will find that he is incapable of mastering his own terrors. His fears, or his fear of fear, can dominate his mind. At such times, the heart has usually rotated outward and become lodged in that danger-anticipating position. This person can be said to have lost touch energetically with the underlying love in the cosmos. This person is, terrifyingly, on his own, in what seems to be an uncaring or even a hostile universe.

Also, a person who decides that he, and not the universe, is the source of his own strength may find it increasingly more difficult to feel joy. This is because the wariness associated with ego-directed self-reliance is fear-based. This fear can serve to reposition the receptors of the heart to face outward. The simple technique described above, which serves to reposition the heart reception into its correct alignment, can be very powerful.¹

Of course, one who practices conscious control of his thoughts is less likely to be a victim of habit. Then again, habit aside, many PDer's have a history of consciously or subconsciously choosing self-damaging attitudes.² To recover from Parkinson's, they may need to overcome – when dealing with new incoming sensory information – both their habits and their willful determination to choose negative thought patterns. Only so will they be able to tap into enough heart-joy that their brain-directed motor area will be able to, once again, move easily.³

¹ Although the signals emitted by the heart appear to be holographic (the same in all directions), it seems that the reception *into* the heart from outward signals is directional. I find it fascinating that the heart, historically considered to be mostly muscle with a self-contained electrical drumbeat, turns out, in the 21st century, to be an endocrine gland (releaser of hormones and neurotransmitters) as well. Not only that, but 60 to 65% of the heart's cells are neural cells, not muscle cells. Also, "the heart produces and releases a major hormone, ANF (atrial natriuretic factor), which profoundly effects every operation in the limbic structure (the emotional brain, also known as the primitive brain, or the "lizard brain"). The limbic area, in addition to regulating non-reason-based responses, also has an effect on memory, learning, and the hormone centers.

"Approximately half of the ANF released by the heart helps to integrate the rest of the body, allowing its parts to perform as a whole. The other half works with the brain; it can "carry on a twenty-four-hour-a-day dialogue between the heart and the brain." This information is from Chris Mercogliano, Kim Debus, "Does the Heart Have a Brain? An interview with Joseph Chilton Pearce," *Self-Realization*, Summer 2000, pp. 42-44.

² Many PDer's have said that fear of self-absorption or superficiality leads them to choose behaviors that are "correct" rather than "fun." And yet, by specifically shunning "fun," they usually inadvertently close off the door to joy. Hence my reference to "self-damaging" attitudes.

³ These attitudinal choices will be discussed more in chapter xxx. The attitudes that can be particularly damaging for a PDer include cynicism, resentment, self-pity and the fear of being judged.

A heart radio case study

I worked with a recovering PDer who was terrified of the dentist. Although this fairly well-recovered person could chase his nephew merrily down the beach or chase his wife around the dining room table when feeling frisky, he was now finding himself reduced to a heap of violent tremors when confronted with the thought of the dentist's chair. He had developed a dental situation that was going to require several visits over several weeks. The first time, he had to leave the dentist's office, procedure undone, because he was trembling too violently for the dentist to proceed.

I taught him the heart radio technique. He did it prior to his next dentist visit and even continued the silent mantra ("Tune out this fear from my heart radio!") throughout the procedure. The attending nurses, who had seen him shaking violently just a week earlier, were shocked at his new, relaxed mien.¹

When and how long to do this technique

The heart-radio technique can be beneficial when a person is anxious or fearful. It may not be of benefit, may not produce a noticeable shift, if a person is already feeling calm.

How many times should one drag the hand across the chest? How many minutes should be spent in this exercise? Sometimes, relief can be felt after five or ten movements. One time, I moved my own hand across a PD patient's T-shirted chest for longer than half an hour and neither of us said any mantra at all. I did it slowly and steadily until his body seemed to relax deeply. When he suddenly went uncharacteristically limpish, I asked him how he felt inside, in his heart, and he said with rare serenity, "Content."²

¹ This case study is interesting because this PDer clearly showed significant signs of recovery: his nearly-lifelong problem with constipation was healed. His facial expression returned. However, the return of suppleness and arm swing remained intermittent: he was able to move increasingly easily when he was unthreatened, but stressful situations were almost always able to induce trembling, weakness, and even foot sticking and difficulty in initiating movement. Even though he had success in the short term with the heart radio exercise, he soon stopped doing it. Though he insisted that he wanted to recover, he did not seem to be interested in doing the work required to make a consistent, lasting change in his make up; by his own admission, he practiced this technique steadily for several weeks, culminating with the triumphant success in the dentist's office – and then he lost interest in the technique, and stopped doing it.

Despite tangible, lasting changes in many of his body functions, he was certain that the benefits of treatment and mental techniques were placebos and that his behaviors in time of fear and stress revealed his "true" condition. He kept waiting for us to find the physiological kink that he felt was preventing him from being happy *all* the time. We could find no such physical problem and often reminded him that he could once again move normally, even athletically, when he was having fun or when he was relaxed. He dismissed these long-lasting periods of healthy movement as being "psychologically" induced. When we asked him, "Why not induce them all the time?" he answered that, in his heart, he knew he was still damaged by his stepfather's brutal assaults. He was determined that healing this underlying bitterness towards his stepfather was our job, and could and should be done via needles, massage, or some other physical or talk-type therapy. He was adamant that he himself could not change his attitudes. He felt he had no choice in the way in which he dwelt on the life-shaping traumas of his childhood or in the way that he collapsed into short-term episodes of PD-like symptoms when he was frightened or concerned about his condition. He finally stopped treatments and, two years later, applied for disability insurance.

² I want to mention that, when I saw this person a month later, he said he was doing very poorly: he was depressed and preoccupied with his conviction that his arm was never going to swing again. I asked

So, in answer to the question, “How long should I do this?” I will say, “As long as necessary.”

For long-term relief, a person needs to make a steady habit of attuning his or her consciousness in with the love that is radiating in the universe. To first feel the result, a calmness spreading over the chest, a healthy person may need to repeat the hand motion, with or without words, for less than a minute. PDers first starting out may need to do it for over an hour.

Concluding this section on the heart radio exercise, this exercise should be done as both a practical, physical technique, and also as an experiment in watching what happens in the body when the heart is rotated towards the midline. Do this sometime when anxious, depressed, or feeling fearful. Take some time to do this exercise when you have the time to really watch and observe the changes in yourself; do not wait until you are on the verge of a fear-collapse.

This heart radio technique is just a start. It can provide steadily increasing relief from fear if the exercise is done with a willingness to tap into the love that is vibrating behind every atom. If this exercise is done with a cynical conviction that there is no such thing as love, the exercise may only work for the short term. But even in the short term, it may provide a glimpse of heart attunement with joy.

Other techniques in this chapter and the next may prove helpful in solidifying this attunement.

Judge not, that ye be not judged

This next technique, like the others in this chapter, appears to be so simple that people refuse to believe it can work, until they try it. It consists of not caring about or not being afraid of the horror of being judged by others.

Many PDers, particularly the ones who remain stuck in fear even after their foot injury is gone, have a lifetime dread of being judged. Some are afraid of being “wimpy” or weak, others are afraid of being considered lazy. I have met PDers with a deep, compelling fear of, for example, being either a moron, a bad housekeeper, a bad dresser, not-manly, or bad-at-math. The list goes on and on.

Of course, it is perfectly normal for a person to doubt his abilities in some realm of life. But these PDers have taken their special fears and made them the cornerstones on which the edifices of their lives are built. The irony is that, in most cases, the PDer who

him if he'd done the heart radio exercise. He said, “Done the what?” I reminded him of how good he'd felt after I'd done the heart radio exercise. He remembered that he'd felt good but, although he was an extremely intelligent and quick-minded man, he had no recall of how to do this simple exercise. I showed him again, but after he did it for a short while, he told me, “You know, I'm not going to change. I'm just who I am. I'm never going to be able to be happy.”

So, as an experiment, I replied by telling him that I had read about a great saint who lived in the Himalayas. This saint was purportedly so elevated that merely saying his name with reverence would bring one a great spiritual blessing. I asked the PDer if he would like to know the name of this saint so that he could say the name. Without pausing to consider, the PDer said, “No, I'm not interested. Even if I experienced some miracle, I would need to rationalize it away because I am not interested in anything that I cannot rationalize.” I did not bother to remind him that he had been unable to rationalize the effectiveness of the previous session's heart radio-shifting exercise and, yet, it had worked.

I was not surprised at any of his responses; I have heard similar protests from so many PDers.

has a fear of, say, being bad at math, has usually made himself into a powerhouse of math and logic in the eyes of everyone around him. However, the PDer with the bad-at-math complex still finds himself trembling violently whenever a situation arises in which he needs to add a few numbers, such as balancing the checkbook. It seems as if the PDer latched onto the idea of some personal failing at an early age (possibly the age when the foot injury occurred) and cannot overcome the oppressive fear associated with this perceived failing.

The Stopping the Judge technique

This technique has to do with simply announcing to yourself and anyone who is interested that you *are* a failure in this particular area. (You may have to search far and wide for anyone who is truly interested; most people don't actually care about your self-conscious fears.) We have learned that doing the opposite of this technique (for example, the politically-correct treatment of combating weakness by stating "I *am* strong, I *am* strong," when you know darned well that you are not strong, is as useless as trying to force love and sweetness into a leg or arm that, according to the mind, needs to be dark and empty.

Merely stating something that you *know in your heart* not to be true is simply dribbling chocolate over a piece of coal. You are lying to yourself. It doesn't work.¹

Therefore, we suggest the opposite of some feel-good affirmation that will, inevitably, be subconsciously perceived by a PDer as deceitful: we suggest bringing the problem to the forefront of consciousness and then learning to play with it. For example, a person who is an overachiever because his mother always shamed him with statements about his laziness might say: "I *am* lazy and that's just the way it is!" or one who was bombarded with guilt about his inability to perfectly clean the house and therefore became an obsessively perfectionist housekeeper could say: "I am a bad housekeeper and maybe I always will be."

Several things might happen when you do this. First, the obviousness of the lie – after all, the PDer has doubtless taught himself to be a spectacular housekeeper, or a stupendous overachiever – might make the statement seem somewhat funny, if not ludicrous. Bringing the problem out into the open so that it can appear ridiculous is very powerful. The old adage "the devil hates to be mocked" is very true.

¹ Actually, affirmations *do* work for most people. But many PDers, even those who are "determined" to recover from Parkinson's, are so locked into the idea of the impossibility of true healing of their emotional and psychological wounds that an approach at variance with the classic "positive affirmation" format seems more helpful. What works best for these folks is to admit to the fear. Then, the fear needs to be brought to the forefront and vigorously addressed, even mocked. When the fear is out in the open, the PDer can work at abolishing it. Without first rooting out the fear, his attempts at positive thinking, as in this case, claiming fearlessness while actually sitting on masses of subconscious fears, are perceived by the PDer as lies, and not as true proclamations of positiveness.

As noted in an earlier footnote, the currently popular Emotional Freedom Technique, in which a person admits that he has a problem and then verbally states that he loves himself anyway, does not seem to work on PDers. I wonder if this is because they are so deeply attached to their noble idea of non-love of self or other variations on stoic suffering that their verbal statements to the contrary are not believed by the core being. By including the idea of self-love into the acceptance of the problem being addressed, it may be that the EFT technique raises barriers of its own for the PDers who have tried it. This may be an interesting avenue for further research.

Another option is, if the fear is something enormous, such as “I’m helpless to prevent people from dying,” then stating the obvious instead of being ashamed of it will at least bring it out in the open. Once the fear is out in the open, it becomes far easier to realize, “Of course I can’t stop people from dying! No one can!”

Basically, this technique consists of admitting that the fear of weakness or the fear of the problem is there – it does exist – and that all the vigorous anti-lazy work or all the good housekeeping in the world is not going to change the real issue: the fear of being judged and coming up short. The next step then is the admission that *no one* can be a perfect housekeeper or a perfect automaton of achievement. From there, one has to state to oneself, “I do a reasonable job. I refuse to be afraid or ashamed any longer that my work in this regard is imperfect. I do a fine job, a good enough job, and that’s the end of it. I can keep doing my best, but I no longer am willing to be afraid or ashamed if I don’t do a superlative job.”¹

The next case study is an example of how simple this process can be.

*Lady of Maine, I adore you*²

Roxy came out to Santa Cruz for a second round of treatments by the PD Team. During her first visit, six months earlier, she had said that she could not and did not want to change her personality in any way; she was the “responsible one” of her extended family: if it weren’t for her, no one would organize the Thanksgivings, no one would make the decisions about grandma, etc. Also, her friends had told her that they wanted her to recover so that she could resume her role as The Competent Organizer in their skiing circle. We discussed the necessity of “letting go” a little bit, of needing to at least let go of the tension that was holding her foot in that twisted position. If, in addition to letting go of the ankle tension, she could also let go of some of her onerous burden of social responsibilities, well, so much the better.

During her second visit, she seemed like a changed person. She laughed more easily and she didn’t seem concerned any more about whether or not she could change; she already had. She had been receiving FSR treatments once a week, and whether it was

¹ Speaking of confessing ourselves to be guilty of imperfections, it might be helpful to note that verbal confessions can be extremely healing. In a rare example of cultural awareness, the U.S. Veterans Administration is now paying Medicine Men to provide care for returning Native Americans (some of whom prefer now to be called Indians) who have been emotionally damaged while serving in wars.

As a VA-employed Medicine Man explained it, the sweat lodge can be a deeply healing experience for service members who have hurt their soul by killing or wounding other humans. In ceremonies and in the experience of the sweat lodge, in which hot rocks are doused with water, making steam, the wrongs can sometimes be verbally shared and “you give your troubles to the rock and burn them off. You no longer have to carry those burdens.” (From *NewsYahoo*, “Medicine Men Help Veterans,” Michelle Roberts (Associated Press writer), Nov. 29, 2005.)

The importance of ceremony and verbal sharing of past shames has been extremely healing for others, as well; recovering PDers who are lapsed Catholics may wish to avail themselves of the opportunity to go to confession simply to be able to ceremonially surrender to God any weight of woe that they have been carrying around. People of any other faith who have learned methods by which they can give to God or the Universe their burdens or anxieties are advised to do so.

² My editor pointed out that most young people today have never heard the Tin Pan Alley song “Lady of Spain, I Adore You” and that, therefore, this section-title gag is pointless. However, I like the song, I like the section title, and I’m leaving it in.

the treatments or her own musings on the subject, she couldn't know for sure, but she was different and she knew it. For the record, she had *not* undergone a majestic transcendent experience, nor had she gone through a "dark night of the soul." However, she had become far more easygoing. She shared with me a short story of a recent event that, it seemed to her, had been the turning point. I will paraphrase Roxy's accounting:

"My sophisticated sister from Manhattan made her annual visit to Maine to come visit me and the rest of the family. She hadn't been out of her car five minutes before she riveted her eyes on my khaki slacks and plaid shirt and, with an accusatory, mock-scandalized voice, said, 'My God, Sis, look at you! You look like you're dressed straight out of the LL Bean catalogue!'"

Roxy continued, "And you'll never guess what I did; I just smiled back at her and said, 'Yes.'"

"That's all I said, just a cheerful and sincerely contented 'Yes.'"

"In the past, I would always say something like "Oh, these old clothes...well, I was just going to change," and I would add something about how I was only dressed this way because I was feeding the dogs or some sort of excuse that would have served as an apology for not meeting her expectations. I've always been afraid of people having negative thoughts about me, of not fulfilling the expectations of others. But this time, I just gave her a big smile of loving indifference and said, 'Yes.'"

"My sister was nonplussed. She just stood there, gaping at me, incapable of speech. I stayed right where I was, grinning at her, enjoying the new sensation, and didn't say anything. After a long silence, she restarted the conversation by asking about the upcoming dinner.

"As I realized what I had done – how I had spoken with no fear of her criticism, but with simple acceptance of who I was, or at least of what I was wearing that day – I realized that my whole body felt so *light!* There was a distinct change in the *weight* of my body. I felt – I can hardly describe it – I just felt good, and happy, as if my body had become light and easy to move. It felt wonderful. And I realized that all I needed to do in order to feel this rush of joy was this: tell my truth and not fear what my sister – or anyone else – might think of me."

I have included the above vignette to drive home the point that a person does not need to have a complete sobbing meltdown, rant and rage at the moon or join a monastery in order to retrain the mind to release dopamine. In fact, these superficial actions won't help one bit. In the above case, in order to experience the wonderful sensation of dopamine release, Roxy just let go of her old worry about her sister's clothing judgments. It was that simple.

Roxy had her first flash of truly understanding the nature of her Parkinson's disease-related fear when her uncharacteristic, bold but simple "Yes" was followed by a distinct change in her perception of her own motor function and body awareness. She understood, finally, the power that her constant fear of being judged had had over her entire body and her motor function.

Roxy felt that possibly she might be able to shed a life-long fear of being judged by her sister via simply not caring what her sister thought, by admitting that she was, in fact, dressed from the LL Bean catalogue. Essentially, Roxy had said, "Yep, I'm not a

sophisticated dresser!” In the moment when she performed that confession of “failure,” her brain switched easily and naturally from fear to joy.

Roxy told me that she was going to try to walk a middle course between “terrified of what others might think” and “sappy and happy” (a position she had long scorned).

Sadly, the next time I saw her, six months later, she had created a new fear, possibly to take the place of the old one. She didn’t mention anything about fear of being judged, but she did say that her deepest fear, now that the Qi in her legs was running correctly and she was exhibiting symptoms of recovery, was that the Qi in her legs was going to spontaneously, against all odds and flying in the face of science, going to somehow revert and start going backwards again. The fear was always there.

So, Roxy may have managed to overcome her fear of being judged, but she had evidently replaced that fear with another one. For the person who is determined to have something to worry about, there are an infinite number of potential fears. For the person who is determined to train his mind toward the positive, there are an infinite number of reasons to rejoice.

Fearing the judgment of strangers

While on the subject of fearing judgment, I want to share more examples of similar types of fear. Many PDerers have admitted to me that, when out walking, they have, since childhood, been afraid to stop in midstride and turn around and go the other way when walking, even if they have suddenly realized that they left something behind or realize that they are going the wrong way. Why? Because they are afraid that “some stranger might see me turn around and they will think I must be an idiot.”

For the same reason, they are also afraid to do U-turns while driving. Some even melt into tremors of hot shame when they realize that they forgot to use a turn-signal while driving, even if the nearest car is a quarter mile away. “That driver behind me must think I’m a complete jerk!” is the ego-inflated response of the PDer.

I suggest, in addition to doing this Stop the Judge technique, that PDerers start reminding themselves that people are not judging them nearly as often as they like to think; most people are too busy dealing with their own lives to be wondering about why a pedestrian turns around in midstride or questioning the mental acuity of the driver of a car four hundred yards ahead that has already turned down a side street – whether or not he remembered to use his turn signal.

In case a PDer wonders why he is so dominated by fears of being judged and is always found wanting, he might do well to notice how critical he is of others. In my experience, many PDerers want to blame a hyper-critical parent for their immersion in a life-style of criticism. This attitude will be addressed in chapter xxx.

Instead of looking for someone to blame, let’s consider a more mature approach: sometimes, the very fastest way to turn off the fear of being not good enough is to stop evaluating everyone else according to your own impossible standards. When you stop judging others, you may find that some of your own self-criticism eases up. And when someone does criticize you unreasonably, respond with loving indifference.¹

¹ This may be the more accurate meaning of the scriptural admonition, “Judge not, that ye be not judged.” It is our own judgments on ourselves that cause us to burn in shame. No loving God could possibly judge his children as harshly as most PDerers judge themselves.

USING WORDS TO TEACH ABOUT/CONVEY FEELINGS

As I sat to write these chapters of techniques I was daunted by the inherent difficulty in trying to convey the positive feelings that can be generated by these techniques if the techniques are done correctly and repeatedly. The next chapter's exercises are even more abstruse and may seem even more unlikely to PDers who are looking for a way to shift their heart out of "uh oh" and into "ahh."

Here is the problem: those readers who really need to understand the *essence* and not just the routine of these exercises, probably, will *not* be able to glean that understanding via my words. Why? Because techniques by which joy is eventually accessed make use of our intuition, not the mental commands of our inner, fear-based monologue. Means for contacting the heart's potential joy or the intuition *cannot* easily be conveyed to the skeptical reader by mere words, and yet, the readers who are most keenly looking for help from these chapters tend to be people who interpret their entire existence literally, via words.

The problem is similar to that experienced by people who do not believe that love exists and, since their own heart is closed, would like to have love "proved" to them via some sort of well-worded logic. But, as most of us know, you can't prove the existence of love via words any more than you can describe the taste of an orange or the smell of sage.¹ And you can't get to joy via reading chapters about techniques for removing emotion blockages and habits, or by mechanically performing the techniques.

At some point, while using the techniques as a leaping-off point, there also has to be some listening to the intuition, some reading between the lines. One has to realize that joy can't be found in some external process; joy is already present, waiting shyly in the background – it simply needs to be allowed to step forward. And simply sugar coating a

And for these hyper-critical PDers, it's bad enough that they are making themselves miserable with self-criticism. Why must they add the weight of their critical judgment onto others? Though they think they uplift themselves by criticizing others, in fact, they actually perpetuate their own pain and negativity.

¹ Do not imagine that neural functions such as taste are free from the influence of the heart. Anyone who has received bad news during a meal might have had the "food turn to ashes" in his mouth. In order to truly savor food, one must be in the mood. A person in a hurry who bolts his food cannot enjoy the tastes, and may not even notice them. It is no coincidence that many food lovers prime their palate with alcohol: alcohol temporarily elevates dopamine levels and turns down the sympathetic system. Alcohol thus allows the heart to "open up" a bit. This alteration in the heart's feeling capability then increases the appreciation and even the anticipation of the food.

It can be no coincidence that PDers lose their sense of smell and taste at about the same time that they begin to lose their sense of joy. This concomitant loss can also be understood in terms of the PDer's loss of Qi flow over the face and sinuses and the paired decrease in Qi flow to the Heart channel. The Heart channel decrease necessarily follows from the decrease in Stomach channel Qi making its way into the Spleen channel and hence into the Heart channel. (The numbness on the side of the big toe where a healthy Stomach channel flows into the Spleen channel, SP-3, has been recognized as common in PDers, even by western doctors. In the flow chart of the channels, the decrease in channel Qi set in motion by the foot blockage cannot be compensated for until the flow line comes to the Small Intestine channel (which immediately follows the Heart channel). The Small Intestine channel, like all outer (Yang) arm channels, has a branch that goes to the Du channel. The Du channel is able to provide additional input, when necessary, to bring the system back up to speed.

negative personality with the actions or phrasings of positive behavior is not the same as replacing negative thoughts with positive *awareness*.¹

My message is that, for a deeply negative PDer, a sea change has to occur. He must become willing to shut down, for a moment (and after that, for longer and longer periods), the inner voice of negativity. He must simultaneously open his heart to the simple experience of pure feeling (also known as “awareness”), and then convey the peace of this experience to his relationship with his rigid sense of reason and, even more tricky, to his own feelings about motor function.² When he is able to do this one time, he must then do it again and again. The ego habits of negativity die hard. They must be countered with the building of new habits of awareness.

If there were *words* that could produce in the reader intuitional awareness, brochures would be snapped up at every good bookseller, and antidepressant-drug manufacturers would go out of business. If joy or spiritual perception were available through *words*, academics would be filled with bliss; if joy could be taught in words, every child’s mother would share these words with her lisping toddler and the world would be a heaven; all people would be full of joy. Since, evidently, this is not the case, we might concede that words alone are not adequate to convey awareness or intuitional perception.

Even the greatest teachers throughout history have struggled to convey, through their actions and examples, their parables and poems, the love and joy that is potentially available to every person. Their work is usually misunderstood, at best. At worst, their students imagine themselves helpless victims, and expect the teacher to fix their lives via miracles. They ignore the message that they themselves can become teacher-like by replacing their cleverness with awareness of the ever-present joy within.³

So, if great teachers through the ages have found this a challenge, you can just imagine how daunting this task feels to me, the reluctant researcher. Fortunately, I’ve no

¹ What does it mean to have awareness? It is not something that can be conveyed by words. When Pilate asked Jesus, “What is truth?” Jesus remained silent. And as it says in the Hindu scriptures, “He who knows, knows; none else knows.”

² If the reader is uncertain what I mean by “feeling,” please go back and read the footnote in the previous chapter in which I describe the man whose chest expanded when he heard birds singing. Another common example of pure feeling is the expansion of the heart that one feels from hearing noble music or beholding a magnificent sunrise. The feeling thus engendered cannot be explained in or induced via words. And conversely, the fewer words one has rattling around in his mind, the easier it is for him to feel the heart-opening joy that lurks in all creation, the love that throbs invisibly throughout the universe.

Almost everyone can feel moved when the entire sky is taken over with evening’s purples and golds. It requires more inner serenity and more subtle powers of feeling and intuition to feel the same swelling of joy in the presence of a daisy even though the miracle occurring in the living daisy is even greater than in the miracle of the sunset. PDers, as their illness progresses, often find that they can no longer experience much heart feeling from anything. This ability must be reawakened if they hope to recover.

³ For that matter, most of the great ones never even wrote anything down. In most cases, these teachers taught through the examples of their lives. Their closest disciples were the ones that wrote up the “message,” as they understood it. For example, Socrates was a great soul, but we only know of his teachings through the writing of Plato. The reason for this reticence of writing may be due to the impossibility of actually communicating “the heart message” via words. Still, we all keep trying, don’t we?

shortage of emotion- and heart-touching case studies to help get the point across. Also, while the students of great teachers can indulge themselves in a *laissez-faire* attitude such as “If I don’t discover joy in this lifetime, I can do it in some other life,” I have this advantage: my readers are more motivated. Sadly, many of my readers are scared or in pain. But looking at the bright side, their willingness to “hear with their hearts” might therefore be better. At any rate, though I keenly feel the challenge of writing this set of chapters, I’m giving it a go.

Literalists

I do know that many PDers have subconsciously chosen, *despite changes in symptoms that imply recovery from PD*, to remain partially or intermittently immersed in the disease. They continue to experience paralyzing self-pity, resentment, guilt, and a crushing sense of self-importance or responsibility (adrenaline-dominated behaviors) and a conviction that they are still in the grip of some amount of Parkinson’s disease. These same PDers have also, in my limited experience, tended to be word-based literalists, holding onto an adrenaline-dominated, fear-dominated approach to words.

Now, here’s the problematic part: these people, though they truly do want to recover from their symptoms, may not be able to understand the spirit in which one must enter into the proffered techniques. Instead, they will probably read these chapters’ suggestions and then force themselves to perform the techniques literally, like automatons. The techniques, when performed in such a manner, are pointless.

I have seen repeatedly that those PDers who are most firmly locked into negative thinking are the ones who usually postpone indefinitely initiating practice of the exercises. Then, if they finally do perform them, they resent, if not hate, the process of the exercises and the “wasting” of so much time. They may grudgingly go through the motions, but they will completely miss the point of the techniques. They will have been trying to follow the literal performance of the exercises, but will have completely missed the “feeling” for why they were doing them.

And for those PDers who have dedicated years to counseling and soul-searching introspection and yet have come up empty handed, maybe it’s time to admit that relearning to connect with the emotions can *not* usually be done via words. Words, as noted earlier, are, for most of us, associated with the part of the brain that compares and contrasts. Logical streams of words are activated, for most people, with adrenaline.¹

¹ One might argue that I am accusing speech and even the power of reason itself to be “bad things,” fear-based things. This is not the case. Even though nearly all PDers seem to use words and apply reason from a fear-based stance, words and reason are, in some people, the result of joy-based attitude. The human attribute of *healthy* reason, as opposed to adrenaline-based reason, occurs when reason is conjoined with intuition: mind combined with wisdom and feeling. (Continued on next page.)

The “leap of faith” that most of the great inventors and scientists rely on for their breakthroughs occurs when, in calmness and fearless mental clarity, they allow their reason to resonate with their intuitive faculty: a faculty that is shut out when the ego-directed mental monologue is dominant. Hunches from “the heart” or “the gut” and productive, logical reasoning are positive things. But most PDers, even though they imagine themselves to be highly rational, tend to be predicating most thinking on fear, rather than gentle joy.

Of course, there are singers and poets whose use of words stems from their flow of joy and not their sense of fear. In fact, by adding music and joy to words, very often the mind can switch from fear-based behavior over to joy-based. This is why most people who stutter or stammer cease to stutter when

The ability to read between the lines and resonate with the essence of these chapters' techniques, an intuition-based ability, is often lacking in those people who choose fear as their modus operandi. Not only that, these fear-shields are often the habits of a lifetime. Even if the techniques in these chapters successfully modify the thought processes for a short while, a PDer who is locked into fear-based thinking can quickly return, from sheer force of habit, back into his old thought patterns after practicing these techniques for only a few minutes.

Therefore, the new attitudes may need to be practiced repeatedly. The old thought processes must be defied and the new attitudes must be instituted over and over again. How long, how often must the PDer wage this war? He will need to have as many struggles as the PDer *wants to think* that he needs to have.

The job of changing one's attitude can take lifetimes – or it can take minutes. Ironically, one thing that can determine how long it will take to change an attitude is: attitude. If a person is determined that it will take a long time to change, it will take a long time.

Another thing that determines how long it can take to change from ego-based fear to joy can be a person's ability and willingness to throw away his ego and humbly ask the universe for direction or insight. Attaining this humility can also take lifetimes – or it can be a matter of minutes.¹

they sing. When they switch to the music of the parasympathetic system, they use words via an entirely different neural system – one that doesn't stammer.

And it is possible for people who have learned to control their minds to use words from a joy basis rather than a fear basis. However, most people have many fears associated with speech. The internal dialogue of most people is more likely to be saying "What I should have retorted to that bully is..." rather than pondering which words rhyme with "moon," "June," and "spoon."

¹ How long does it take for a person to learn to accept joy? C.S. Lewis, author of the famous Narnia Chronicles (including *The Lion, The Witch, and The Wardrobe*), was raised a Christian, studied philosophy, and became a staunch atheist for much of his adult life. However, a few days after talking with his good friend, J.R.R. Tolkien, a Catholic, Lewis experienced an instantaneous heart change. He describes the experience in *Surprised by Joy*. The gist of the experience is this: Lewis was in the car on the way to the zoo. When he got in the car, he did not feel a spiritual connection to Jesus, nor did he think that Jesus had a special relationship with God. By the time he arrived at the zoo, he was certain of both.

While many purport to want to feel joy and/or know the truth about the creation of the universe, about spirituality, immortality, and love, most people are reluctant to perform the simple steps of humbly, but with determination and single-minded focus, asking the universe, in the language of their own hearts, to be shown the peace, the joy, and the answers. And yet, if one asks with genuine humility and real desire – and then listens to the answers of the heart – the consciousness can be attuned to long-lost joy within a matter of moments.

Of course, the answers are usually the very things an egoist does not want to hear. Fortunately, the joy of Love and Wisdom is so sweet, so perfect, that the shallow and fleeting joys of the ego instantly dim in comparison. And then comes the battle between ego-based habit and the new desire to follow the dictates of the heart. That battle can go on for lifetimes if one so chooses. But the initial introduction to life-changing joy can sometimes come as quickly as it takes to ask for it.

I read recently an autobiographical sketch in which a man stated that, having been raised in atheist Russia, it was utterly impossible for him to consider any spiritual matters as worthwhile. He wrote that, despite his own atheism, he was good friends with many people who were deeply devout, in various religions; he envied these people because they seemed to have something that he knew he could never have. As I read this, I noted to myself that the only thing preventing him from having this same "something" might be his own certainty that he could never have what they had. It was purely his own attitude that was preventing him from having that "something."

Therefore, a person whose dominant attitude is a negative one may have a self-fulfilling prophesy in place – a prophecy that states “this is going to take a long time, and it probably will not work.” And then the mind and body, working together, will comply with this prediction: changing the attitude will be – as predicted – nearly impossible.

The deepest understanding of these chapters will come about by reading the material, practicing, re practicing, and continuing to practice these possibly hateful exercises until, at some point, you realize that what you are becoming while doing the exercises was the point all along, and never “perfecting” the exercises themselves.

Noticing how your mind waffles and wanders while you do these inner-voice stilling techniques might help you to make the leap to understanding, or at least objectively recognizing, your own behavior. Only when you are able to dispassionately observe your unrestrained mind careening about while you try to stay focused on the exercises might you begin to understand the magnitude of what you are reining in. Then, by recognizing these unrestrained patterns and trying to shift them for a second or two, you will inadvertently begin to change.

Merely reading the exercises and trying to attain their goals by logically understanding the mechanism will not work. You will not change yourself by studying the techniques with a fine-tooth comb as if they are arcane cures: they are not.

In fact, many of the techniques in this group of chapters were invented or modified by PDers during their own battle against habits of negative thinking. I suspect that most readers who truly intend to succeed will also form their own methods – based on the lessons hidden between the lines of the case studies, and *not* strictly, formally, based on the suggested techniques.

Now, from the standpoint of content, this chapter is not finished. My gentle editor suggests, however, that I have too many “techniques for changing the mind” to fit them all gracefully into one chapter. So, for ease of reading, there is a “chapter break” here, even though the material in the next chapter will be simply a continuation of this chapter.



“Awareness cannot be taught, but through discipline and right attitude, it can be attained.”

- Anonymous

APPENDIX III

ANXIETY AND GRATITUDE

This chapter is actually a continuation of the preceding chapter: it contains techniques to retrain the thought stream towards positive, self-controlled thoughts and away from unintentional, habitual negative thinking.

ANXIETY

Anxiety, unlike focused thought, is a pointless whirring of the uneasy mind that keeps the emotions edgy and produces nothing to show for itself.

Anxiety repeats itself and fails to learn the lesson that tracing and retracing the same tired set of thoughts never brings the future any nearer nor changes any outcomes.

Anxiety can worry endlessly about money even though the anxiety will never increase the bank balance. Anxiety can fret about the danger of the highways without taking a step towards improving driving skills or road conditions. It can fret long into the night over which college the young children will someday attend but will not make any improvement in the childrens’ math or spelling skills.

Anxiety is an utter waste of time. It aggravates the blood pressure and prevents the enjoyment of the present. It is one of the most indulged in, most pointless mind games that a person can choose. Many people with Parkinson’s indulge in anxiety to a high degree. The following technique, if practiced, can successfully extinguish specific anxieties, and lead to a state of mental carriage that, eventually, can redirect the mind away from anxiety in general.

Giving away a specific anxiety

A highly effective way for retraining the anxiety-fixated mind is to select an anxiety subject that occurs regularly: one that you want to get rid of. Examples might include repeatedly evaluating how I should handle the retirement plan, what will I do with grandma if she becomes feeble, what car will I get if the Chrysler breaks down, who will be coming for Thanksgiving, what if I don’t get the job/role/political office, what if this hair cut is all wrong for the part.

Next, call to mind someone who can help you with these problems. This someone can be a late aunt or deceased beloved parent.¹ It can be a saint, The Light or Power that runs the universe, God or Pinocchio’s Blue Fairy. This someone must be a powerful

¹ I stress the word “beloved.” I had one patient who decided to ask his late mother, whom he had not liked, to take care of a particular fear. He found that the process was dreadful; every time he thought about his mother, he remembered how much he had feared and hated her, and then he felt worse than before. He had assumed that, since his mother had died, she must have become an angel and, as such, should be the one to whom he could give away his problems.

Don’t give in to this specious thinking. If your mother (or whoever) was unreliable in life, she will be unreliable for you after her death.

entity who is willing and able to deal with these things on your behalf. You must be certain that this someone has the ability to intercede on your behalf with Mother Nature, the Infinite Love, God or Consciousness that governs the universe.¹

Next, *every* time you realize that you are thinking, pointlessly, about the specific anxiety subject that you have decided to be rid of, you must say with great firmness and courage, silently to yourself (or out loud, if no one is around), “I refuse to dwell on this anymore: Saint Teresa (or Blue Fairy, or whoever), I give this problem to You; *You* take care of it.”²

¹ I recall a conversation with a PDer who adamantly insisted that God and religion not be involved in his recovery from Parkinson’s. He said, “I am willing to find joy. I want to find joy; but I refuse to do it via religion. Do you know of any good books I can study on the subject of joy?”

I replied, “Yes, but they are all in the spirituality section of the bookstore.”

“But I want some that aren’t in that section!”

“That may be what you want, but those who have truly found a way to lasting joy have, at some point, realized that what they have found is exactly what all of the great ones from time immemorial have been talking about. They suddenly understand, reading between the lines of all scriptures and “spiritual” instruction, understand that all of the great ones were joy-filled souls who were trying to share with others the truths that have brought them joy.

“Your problem is that you think that spirituality is related to churchianity. In fact, “spiritual” and “joyful” are synonymous. If someone does actually discover a path to joy, it always turns out that the truths and methods that he shares have a spiritual bent. So, by definition, I cannot recommend a book that will lead you to joy that is outside of the spirituality section of the bookstore. Many writers try to disguise their spiritual underpinnings by using words like “the Universal Joy” instead of “God” or by saying “the higher Self” instead of “the Soul,” but these are exercises in semantics. If you are determined to find a way to joy without using the word God, simply exchange the word God with the word Love and select some little-used nouns that work for you to replace words like “soul.” Go ahead: the Love that has set the universe in motion won’t mind a bit.”

² I have heard many times from PDers that they do not wish to give away their problems. Here’s an example: “I love Mother Mary, I don’t want to hurt her by giving her this dreadful problem; it’s better that *I* should suffer than that *she* should feel this pain.” I now consider a martyr-like fear of sharing problems with others to be a not uncommon characteristic of Parkinson’s disease. On behalf of these souls who would rather suffer themselves than let their suffering be shared, I wish to issue a reminder: God does *not* suffer when you turn over to Him your pain-embracing games. God, or the Love in the universe, of which you are a part, suffers for as long as you choose to cling to your pain. By giving your pain over to some aspect of divinity beyond creation, you rid the physical universe of that pain. The Force that set the galaxies in motion can easily absorb your (self-created, if the truth be known) problem and convert that energy back into joy. For those who do not want to “hurt” God, I sometimes suggest they give their pain to Kali.

The ghastly, fearsome image of the Hindu goddess Kali, often misunderstood in the west, represents the awesome power of God that can destroy delusions of material reality, including the delusions of sickness and death, dissolving their energies back into eternal Love from which they sprang. Statues of the red-eyed, black-skinned goddess Kali portray her with garlands of skulls and drops of blood hanging from her lips. She is not a pain-inducing goddess, but rather the destroyer, even “the devourer,” of pain and delusion.

(Although I refer to Kali as a goddess, Christian, Jewish and Islamic monotheists may wish to remember that Hinduism is also a monotheistic religion. The understanding that the infinitude of created things are all God’s handiwork and a manifestation of Him allows the Hindu to refer to the one God by an infinite number of names: to speak of anything is to speak of God. When a Hindu refers to the blooming powers of springtime as one aspect of God and the demolishing forces of winter as another aspect of God, he is not stating that there are two Gods. Although Hindu references to “the God of creation” and “The God of destruction” may create a polytheistic interpretation in the western mind, these inadequate translations into western tongue are, nevertheless, references to *aspects* of the One. The Hindu recognizes that a limited human, with a mental focus on the finite, may nevertheless approach the Infinite through a multitude of

The next time the mind strays back to the unwanted subject, silently, mentally repeat the phrases, “I give this problem to you. I refuse to be haunted by this relentless worry any longer; You take care of it.” Every time that you find yourself wrapping your thoughts pointlessly around this subject, repeat the phrase, “I give this problem to *You*. *You* take care of it.”

Soon, instead of burning ever-deeper habit grooves of negativity and anxiety in your brain cells, you will be building a new circuitry. The new circuitry, when it is finished, will move with lightening speed from the thought of the problem – when the anxious mode about that particular problem arises – straight to the thought of your wonderful someone, your *You*. Eventually, you will have created a new neural shortcut in your brain. The thought process will have formed a direct line from the thought of “Problem X?” to the thought “You, my friend,” or “My beloved!”

You may be astonished to find that when you first try to do this you will need to say “You take care of it!” nearly fifty times an hour or more. When it dawns on you just how much of your conscious thoughts have been taken up with pointless, circular worries, you may start to see why anxiety has been able to reduce you to a shaking hulk.

And, strange to say, this method works. The new habit will start to form; you will be beginning to take charge of your own mind. At some point, the new habit grooves that you are forming in your mind will grow so deep and efficient that, the moment the specific anxiety-producing thought appears, the brain will immediately think of the one to whom you have assigned the problem. Instead of becoming enmeshed in pointless, spiraling worry, your mind will immediately jump to the thought of one whom you admire and trust. A nice trade off!

Be warned, it can sometimes take several weeks before the mind starts to solidify the new neural pathways and break down the old ones.

After this particular anxiety no longer plagues you, select another pointless anxiety – some anxiety about which worrying does you no good – and repeat the Give-It-Away treatment until it, too, is conquered.

finite facets. An anecdote may make the point: during the British occupation of India, a British vicar asked a young Indian lad if he wanted to come to church, in order to “see where God lives.” The Hindu lad replied, “Can you show me a place where He is not?” This speaks to a profoundly monotheistic understanding of God, despite the Hindu references to His various aspects as Gods and Goddesses.)

I often suggest that a PDer who does not want to hurt the gentle mother of Jesus, Mother Nature, or the loving Quan Yin (the Asian Mother Divine) by giving her his dreadful problems can, instead, give his debris away to the hideous Kali and visualize her swallowing it up. This suggestion is nearly always met with approbation and relief.

It is sometimes difficult for our finite mortal minds to understand that the one, unified Force encompasses *all* our needs, including our need for an emotional wastebasket. Putting appropriate faces on the various aspects of the infinite can sometimes be a helpful tool; no one in my experience has minded turning his emotional “junk” over to Kali, the forgiveness aspect of God, that ultimately takes up the thought waves of pain and suffering – once a person has decided to be done with such thoughts – thus removing them from the system. Although Kali looks fearsome, she can also be understood as the ultimate forgiveness.

Great souls, at some point, come to realize that forgiveness is the answer to the problem of the ego. If ego is what some Christians refer to as “original sin,” then forgiveness of the ego is the ever-present, ever-available cure.

As an aside within an aside, some pundits even hypothesize that the “Dark Lady” of Shakespeare’s sonnets is the black-skinned Kali, the loving, forgiving, illusion-destroying aspect of Mother Nature. Try reading Shakespeare’s sonnets with this in mind; their baffling allusions are suddenly made clear.

This method can slowly, over months and years, transform the mind. At first, only the problem in question will be affected. Eventually, however, the mind can begin to recognize all anxiety-related mental habits as such, and will rapidly and, eventually, effortlessly perform the new skill of giving the problem away.

You may also notice that this process of giving the problem away to a specific someone causes the mind to be focused on You, whomever that may be, and away from I-me-mine. As noted in the last chapter, this process of directing the thoughts towards others instead of using them to dote on the ego is the key to balanced mental control.

Unexpected benefits

This technique can sometimes, if practiced with conviction, lead to almost miraculous results; the problem about which one was anxious may even be “taken care of” by an unexpected, impossible-to-explain solution, a remedy that beats all the odds or seems to come out of left field. When such a solution to the problem does occur, the PDer must note whether he responds to this blessing by saying “that was just a random chance – it can’t happen again” or if he responds by stepping up his comfort level with “giving away” his problems. The response may be very telling about the underlying attitude the PDer has about his real desire or lack thereof for recovering from fear-based, negative thinking.

Denial of anxiety

Although most PDers have learned to recognize the relationship between their anxiety levels and the severity of their symptoms, it is not unheard of for a person with anxiety-related symptoms to tell me, with complete confidence, that he has no anxiety whatsoever. The following case study will demonstrate.

Abner had a fairly constant tremor, though it worsened dramatically when he used the computer and stopped altogether when he was relaxed or meditating. He insisted that he maintained a very positive attitude, that he had no anxieties and that his tremor was not related to his mental state. He felt that the reason the tremor worsened during computer use was the steady, inevitable worsening of his Parkinson’s.

One day in my office, his wife, contradicting him, pointed out to him that most of his PD symptoms were improving and that even their friends were impressed with his improved posture and facial expression; her feeling was that his biggest worry was fear of not being able to work. (He used the computer for his work.)

Whenever Abner used the computer, he was increasingly unable to function due to computer-induced increase in tremor. His wife felt that he was getting better in all the arenas for which he had no worries, but activities about which he was worried were ones in which his motor function was steadily declining. She felt there might be something psychosomatic going on, but he was adamant that he had a positive attitude and that negative thinking/anxiety had nothing to do with his computer-related symptoms.

Abner’s little test

Therefore, I spent an hour doing an experiment with him to show the relationship between anxiety and his tremor. I held various spots on his head and neck, pretending to be doing some sort of mild cranial treatment, while saying alternately positive and negative statements.

I started out by saying something positive: “Oh good, your second cervical vertebra has stayed in place since our last session.” His mild tremor completely stopped. I said, “Your tremor stopped!” and Abner said, “Yes.”

I waited one minute and then said an “uh oh” phrase: “Uh oh...it seems to me that the bone might not be really holding the position perfectly. I wonder if it’s going to slide back out of place at some point.” His tremor started up. I said, “Your tremor’s going.” He answered, “Yes.”

I waited a minute and then said, “Wow. This second cervical really does feel great. Real solid. I’m certain it won’t slip out. And the fourth cervical is positioned exactly right!” His tremor immediately slowed, and stopped completely within seconds. I pointed out that it had stopped and he agreed. A minute later, holding lower down his neck, I said, “Uh oh, what’s *this* I’m feeling?” The tremor started up, a little larger than before. We both duly noted the tremor. I waited a minute and said, “Oh, it’s nothing. The energy is moving beautifully through here.” His tremor stopped completely. We both agreed that it had stopped. It stayed stopped until my next statement: one that started with an “Uh oh...”

The reader will want to know that there was nothing in particular about his neck that we had been working on. After about half a dozen starts and stops, his wife started chuckling. She could see that I was turning his tremor on and off every minute, like clockwork, by saying something negative or positive. I tried all sorts of statements: “Uh oh, it’s raining. Sure hope you don’t have car trouble on the way home...” (tremor started up). Ah, you’ve got that great car; you won’t have any trouble at all...” (tremor stopped.)

I did this, alternating positives and negatives every few minutes, for over an hour. The tremor turned on or off in perfect response to my alternating positive and negative statements. His wife, watching and nodding her head in silent agreement with the point that I thought I was making, thought that it was a powerful object lesson. Abner didn’t say anything about what was making the tremor start or stop, but I assumed (incorrectly) that he must have been aware of what I was doing.

I did not, at any point, tell Abner that I was intentionally making alternating positive and negative statements. However, I assumed that this extremely intelligent and savvy PDer would eventually make the connection between my statements and our mutual confirmation of the tremor’s starting and stopping. Even after the session ended, I didn’t say anything about the stopping and starting of the tremor in response to positive or negative thoughts; I assumed that he would go home and give some deep thinking to this start-and-stop experience.

Since he had been so certain that he was not susceptible to anxiety, I looked forward to our next session to learn what he had done with this straightforward lesson in anxiety-related tremor. I was completely stunned at Abner’s next session when, in response to my question, “What did you think about that last treatment,” he replied, “It was the strangest session we’ve ever had. *I couldn’t remember any of it.* When we left your office last week, I turned to my wife and said, ‘I feel as if I’ve been in a trance. I have no idea what Janice did or what she said during the session. All I remember is that the tremor seemed to stop completely and then start up again, quite a few times.’”

His wife, astonished, did not tell him what I had been saying and its relationship with the tremor, because she wanted him to figure it out on his own or else hear it from me.

So, when I then told him what I'd been doing, he told me that he'd had no recall of any of it, and repeated, "I felt kind of dazed, like I didn't know what had happened during the session." He had a very hard time believing me when I told him what we'd done during the session. Even with his wife backing me up, he had a difficult time believing that his tremor had been turned on and off every few minutes for a solid hour simply in response to my positive or negative statements on random subjects, including the subject of his ability to heal.

This recalled to my mind the powerful mental disassociations that so many PDer have about their body. I had to wonder if his mind, subconsciously recognizing what I was doing, disassociated itself from the treatment rather than acknowledge information to which it was emotionally opposed: he didn't want to know that his tremor was anxiety-related or that he was subject to anxiety. Therefore, he possibly had disassociated himself from the experience.

Another possibility that we discussed was that, by shifting his mind so regularly back and forth between fear and safety, he may have become a little bit emotionally "dizzy." The unaccustomed frequency of dipping into positive thinking may have been unsettling to the point that his brain could literally not make sense of the session.

In either case, he was stunned to learn that the starting and stopping of his tremor, which he did remember, had been in response to my spoken words. He protested mildly, with statements such as "I always try to find a bright side to bad things," and "when bad things happen I just say that it's a swing of the pendulum and that good things must inevitably follow."

His wife countered by saying, "All the PD symptoms you never worried about, the ones that don't have an effect on your computer abilities, are getting better. The symptoms that you *are* worried about, the ones that you fear are going to keep you from using your computer, are the only symptoms that you still have – and they show up at their most dramatic when you start using your computer. What's that all about?"

At this point, I shared with him several case studies of attitude-induced parkinsonism.

Abner responded with unusual vigor that he was doing the best he could to always be positive – in light of the way his father had treated him. This led us into an important talk about the power of the mind in deciding whether or not to be a victim of childhood's lessons. (This highly significant subject will be discussed in detail in appendix xxx.)

We left it with him agreeing that he had much to think about. Curiously, at our next session, three weeks later, he announced that he was starting to feel so much better that, for example, he was catching himself dancing in the kitchen for no apparent reason. He wasn't sure what he had done to make this shift, but he also didn't say anything at this session about how his symptoms were getting worse when he used the computer. He was rather marveling at how cheerful he'd become, as if something inside was different.

Changing from specific anxieties to awareness of anxiety in general: progress

One PDer, when I first met him, always shared his current batch of anxieties with me during each weekly treatment session. I made weekly suggestions that he might consider giving away his anxieties, but he vigorously defended his anxiety-ridden thought process; "Better to be prepared for the worst than to have the worst take you by surprise" was his motto.

After many months of gently pointing out that he was worse when he was anxious, he walked in for his weekly session and announced “I asked my wife if I was a negative person. She said ‘No more than lots of people.’ So I think you can stop badgering me on the subject.”) I decided that he and I were making no progress in this area, so I dropped the subject of anxiety.

However, I noticed that, after nearly a year, his behavior around anxieties had imperceptibly shifted. He was no longer sharing specific anxieties; instead, he was rather dismissive of the particulars. Instead, he was now keenly aware of how much time he was spending with his mind trapped in anxious-mode, and asked me frequently what he should do about his pernicious habit.

When we first started working together, his various mini-crises were, to his way of thinking, the Source of the Problems, and he argued strenuously that getting rid of his anxieties would be simply “hiding from the facts.”

But, through the months of slow, steady improvements in his balance, movement, and facial expression, offset with increased tightening in his right leg and right arm whenever he was anxious, he came to realize on his own that, in fact, his propensity towards anxiety was his worst problem. When he was finally able to admit that anxiety in general worsened his PD symptoms at any given time, and was no longer dwelling on the specific problems that he fixated on, he was also able to *consider* doing techniques to teach himself how to change his thinking habits.

However, curiously, his preferred solution to treating the anxiety was not mind-controlling techniques, but a new tendency to blame anyone and everyone in his life – past and present – for having created the various situations which had given him the “anxiety habit.”

This new style of “dealing” with anxiety – blaming others for making him anxious – does not seem to be helping him to have less anxiety. Even so, I consider it progress. At least he does now recognize when his thoughts are looping about in an anxious manner. This is important. And, inasmuch as he now recognizes that the mental pattern, and not the specific issues about which he is anxious, is the problem, I have strong hopes that, at some point, the anxiety and the physiological pains that he has, from habit, “chosen” to manifest during his anxiety bouts will become such an obvious and odious problem that he will take his first tentative steps in regulating his own thought patterns.

What happens when a person successfully “gives away” a particular anxiety? Some people find that simply getting rid of one anxiety makes it easier and easier to get rid of other anxiety patterns as well, and, eventually, the exercise in self-control opens the mind up to incoming streams of positive thinking.

Others, however, find that the mind, if left to its old habits, will simply fill in the space provided with some new anxiety. Therefore, the other part of this technique, if needed, involves finding a new thought-stream to take the place of the anxious one that was given away. If new anxieties stream in to take the place of the old ones, this new replacement stream of thought can be inserted every time the old anxiety is handed over. The name of the new stream is “gratitude.”

GRATITUDE

Filling in the blanks

It is only a very spiritually accomplished person who can constantly hold the mind empty of thoughts and enjoy the blissful experience of pure feeling, pure heart awareness, unmarred by intruding, word-based thoughts. A person who is accustomed to filling his mind with anxious thoughts may need some sort of replacement thoughts.

The beginner to the process of mental self-control should use the mental space that has been vacated during the anti-anxiety exercise above by consciously refilling it with gratitude.

The gratitude exercise

When I have proposed a gratitude exercise to my PD patients, an alarmingly high percent of them, people of high intellect, financial security, stable family life and otherwise good health, have asked me, in all sincerity, what they could possibly have to be grateful for. They seem to think that their trembling or their physical impairment is the final stamp on a life utterly empty of blessings. Bizarre, but there you have it.

Others, happily, have just the opposite approach; throughout their lifetime they have always tried to console themselves with some form of gratitude-based thinking. You will be correct if you've guessed that the ungrateful ones are the ones who tend to get stuck in mental whorls of negativity. Those who have trained themselves to be steadily and truly grateful – despite setbacks and difficulties – have had, in our limited experience, a powerful advantage in recovering from Parkinson's.

One gratitude-oriented person who recovered very quickly from her other PD symptoms once her foot was healed once told me that her insistence on gratitude bordered on the illogical, but that she had always stuck to it. Lynne's policy was to find some way to be grateful for all things, whether or not they appeared, on the surface, to be blessings. One of her many ideas was that, if a "bad" event occurred, she could congratulate herself on having successfully worked past that much negative karma.

Sometimes, when something dreadful or painful occurred to her, she would create a mental story in which, in a past lifetime, she had saved a loved one from having the very same problem by having prayed that the problem be conveyed to herself.

For example, when she was diagnosed with Parkinson's, she immediately wondered if, in some past life, she had prayed that a loved one with Parkinson's might be saved: that the illness be given to her, instead. In that life, the person she'd prayed for had recovered, but she herself had not developed the illness at that time. The appearance of the illness in this lifetime, complete with requisite foot injury, was simply the necessary completion of a prayer that had already been answered in the hidden past.

Her motto was "If life or some person treats you in a way that you cannot understand, assume the kindest possible motive."¹ This may sound completely crazy to a dyed-in-the-wool cynic, but Lynne was deeply satisfied in life and highly successful in the eyes of the world.

¹ Lynne credits her younger sister for this quoted bit of wisdom. In addition to working at gratitude, Lynne is always very quick to attribute all her learning and gifts to the generosity and wisdom of others.

As another example of Lynne's insistence on gratitude, when her California home was destroyed in an earthquake, she was, at first, emotionally devastated, but then she found a positive spin to put on the problem: God had, by destroying her home, reminded her to never put her faith in material things. She was grateful for this reminder. Then, she was grateful that her house, and not her family, had been hurt.

She tried to see each day, not as a birthright, but as grace – an undeserved blessing. When she struggled with the exhaustion of raising her colicky first child, she constantly gave thanks to God that at least she hadn't borne twins. This may seem completely twisted to those who are always analyzing how unfair life is (or those who have twins and are grateful for them), but the point here is that this woman refused to wallow in self-pity, she clutched at any positive thought when she was having troubles, *and* she recovered very easily from Parkinson's disease.

She shared with me that once, years earlier at her workplace, a particularly sour co-worker had demanded of her, "You always act so happy. Why?"

Without even thinking, Lynne replied without missing a beat, "I'm just so grateful that I no longer live at my mother's house. Everything else is so good in comparison." Lynne was slightly embarrassed at having blurted out these words, revealing so much about her past to someone she barely knew, but then she started to think about the truth of her statement. She realized that, for the preceding fifteen years, when anything terrible happened, her automatic, silent internal response had been "It could be worse... I might still be living with my mother."

This "could have been worse" ploy would immediately lift her spirits and inspire her to give thanks to the universe, no matter how difficult or painful the ongoing circumstance.

Shifting the neurotransmitter balance

Some of the things that Lynne invented to be grateful for might seem to the reader to border on the ridiculous, but the truth is that she had stumbled onto a very effective formula for fighting despair and fear. When the brain is engaged in the gratitude sector, it cannot access the fear sector. By forcing herself to invent any possible reason to be grateful, she was shifting the neurotransmitter balance in her brain over to the side of joy.

And we all have reason to be grateful: there is no man or woman alive who has not had some of his or her prayers granted. Remember those, if you can, and be grateful.

The exercise of simple gratitude has been so important to those who have recovered easily that we have suggested, to those not inclined to gratitude, that, just maybe, they should give it a try. For those who have told us that they have nothing to be grateful for, we invented the following exercise:

Lie down and try to relax. Next, imagine something mildly yucky that did *not* happen to you, recently or in the past. Now, think of another unpleasant thing that did *not* happen to you. Keep this up for ten minutes. That's the entire exercise. It can be very powerful.

Because the above may seem obscure to a person who thinks he has very little to be grateful for, I will write up an example of what happened to a PDer with a relentless tremor.

Travis's tremor and the gratitude exercise

Travis's tremor never stopped except when he slept. If he was awake, he tremored. Even though he meditated twice a day, and had done so for over twenty years, his tremor no longer stopped, *even during his "peaceful" meditations*. When I asked Travis to do this gratitude exercise for ten minutes, he told me he couldn't think of anything to be grateful for, at least not anything that would take up ten minutes.

So I asked him if his second grade teacher had ever bitten him. He said no. I said that this was the first thing he should be grateful for. Before Travis had a chance to tell me that he knew of *no one* who had been bitten by their second grade teacher, I asked him whether or not his father owned a grist mill, and did he have to perform the job of donkey in turning the heavy millstone. Travis replied indignantly, "Of course not!"

"Good." I replied in turn. "Then that's the second thing you have to be grateful for."

Next I asked him if he'd ever lost a leg during the war. Since he had never served in a war, his answer was no. I pointed out that this counted as two things: he hadn't served in a war *and* he hadn't lost his leg. Travis protested that the things I was coming up with were completely random and stupid. "'No more random and stupid,'" I replied, "than the negative worries about things that *might happen* with which you fill your mind from sunrise to sunset."

He granted that I might be right about that, and asked if he should be grateful for not being blind? In the past, he had always felt bitter about his need for reading glasses. I thought that being grateful for vision, even poor vision, was a good start, and that he should also be grateful that no one had ever poked his eyes out with a stick or burning coals. And what about eating vomit? Couldn't he be grateful that he had never been forced to eat his own vomit?¹

Then I got silly. "Is Travis a nickname and did your parents actually name you Gengulphus?" I asked.

"No!" he snorted. "Of course not."

"Then be grateful," I commanded with mock severity. "And when it rains during the winter, do you have to sleep outside in the mud?"

"Now you *are* being ridiculous," he protested. I agreed with him, but continued in this vein for several minutes, until I decided that he could branch off from my ideas and think of some of his own.

I told him to think of each one of these things that had not happened to him and then pause after each one and give thanks that he had been spared that particular trouble. After having given thanks, he should choose another unlikely thing to be grateful for, and give thanks for that. He should keep it up for ten minutes. If he thought of any more realistic things, he could of course use those as well as, or instead of, the silly ones.

He then asked me to whom should he express these thanks. I said that it didn't matter; he could give thanks to God, to the universe, to Fate, or to whatever force it was

¹ This example may seem pointlessly vile to the reader who has never studied the methods by which young women in certain cultures are force-fed in order to fatten up so as to obtain good marriage prospects. Sometimes, when these young women throw up the contents of their painfully bloated stomachs, they are "taught a lesson" by being forced to eat their vomit. I am including this information in this footnote in case the reader imagines that he has nothing for which he can be grateful.

that he spoke to when he, like all of us, found himself talking inwardly to “someone who isn’t tangibly there.”

Talking to the universe

All of us have, at one time or another, spoken, silently or aloud, some thought or wish. To whom were we speaking at this time? When you, as a child, silently pledged, “Please, don’t let my mom find out about the tadpole incident, and I promise never to tease little Robin ever again,” you were pleading with *someone*. When your heart implored silently, on your child’s first day of kindergarten: “Please, please protect my baby,” you were talking to Someone, Something.

You can call that “someone” the voice of conscience, the Infinite Love, God, or the higher Self. It doesn’t matter what you call that someone. If you have spiritual resources that allow you to give your thanks to Divine Mother, Jehovah or Allah, that is wonderful – do so. But even if you have no mental image or name of some aspect of divinity, you can offer your gratitude to that unknown someone to whom you have often spoken in the past, even though you didn’t know to whom you were speaking.

The inner monologue of fear or anxiety is usually addressed to the fear-ridden ego. By changing the very direction of the inner voice, moving away from talking to the ego and speaking instead to the love or the divinity that resides both within us and everywhere, we can shift the brain away from the “me” to the “you,” from the fear sector and into the joy sector.¹

Travis stops trembling

I left Travis, whole body trembling violently, as usual, to do the gratitude exercise as he lay on the treatment table while I took care of some paperwork at my desk across the room. I noticed that within less than thirty *seconds* of his starting the gratitude exercise, Travis had stopped trembling. His eyes were closed but he was obviously awake and alert, and I could tell by the look on his face that he was concentrating deeply. His years of meditation had trained him well in the art of concentration, although he had evidently forgotten the component of meditation training that includes gratitude.

Travis’s tremor had never stopped during the time I had known him. He tremored while I held his foot. He tremored when he was relaxed and he tremored when he was

¹ There is growing scientific evidence that these shifts in the brain can be measured. Richard Davidson, the director of the laboratory for affective neuroscience at the University of Wisconsin, has found that, in people who are stressed, anxious or depressed, most of the frontal cortex activity is occurring on the right side of the cortex. People who are calm or happy have more activity in the left side of the frontal cortex. Davidson says, “Each person tends to have a natural ‘set point,’ a base-line frontal cortex activity level that is characteristically tipped left or right and around which daily fluctuations of mood swirl.” In *Time* magazine, July 27, 2003, Richard Davidson explained that people who meditate shift their frontal cortex’s left-right activity ratio over to the left. Also, the more and “better” they meditate, the more the ratio shifts. Since prayer, gratitude and surrendering one’s worries to the higher Self (as in the anxiety technique explained in this chapter) are common components of meditation, as is mind control, it begins to appear that using heart- and mind-calming techniques, and techniques such as gratitude exercises and giving away anxieties – even though some might dismiss them as mere foolishness – in order to retrain the mind towards joy, has a strong scientific basis.

I like to wonder if someday, scientists will find that those dour logicians who insist that they are being “practical” when in fact they are being negative may be found to have more *right* frontal cortex activity – thus proving that their brain imbalance, not their vaunted logic, is the source of their skepticism.

active. His tremor, once a resting tremor, had become a relentless, disabling tremor of high intensity.

During the entire ten minutes that Travis did the gratitude exercise, he did not tremor. Travis took himself, his purpose in life, and life itself very, very seriously. He was very much spiritually inclined. He had devoted his life to self-improvement and the education and spiritual improvement of others. But from the chuckles he emitted during the first few minutes of the gratitude exercise, I suspected that he was following my lead in being grateful for silly things.

Then, his countenance became more thoughtful. He never tremored, though. With his mind full of grateful thoughts, his subconscious mind was not able to access the usual stream of anxiety-inducing thoughts with which he usually filled his day.

When ten tremor-free minutes had passed, I gently shook his shoulder and said that he could stop for a bit.

He opened his eyes and looked around. His tremor started up, but it was extremely small. I asked him if he had noticed that his tremor had stopped while his mind was occupied with gratitude. His reply was disheartening. (Though I paraphrase the following, it is as close to accurate as I can recall.)

“Travis! Your tremor stopped while you were practicing gratitude!”

“So?” (Honestly, this was his response.)

“But it stopped! Wasn’t that great?”

“Yes, I noticed that it stopped for a while, but so what? It came back when I stopped doing that dumb assignment.”

“Tell me, Travis, does your tremor ever stop?”

“No. It never stops.” (The tremor, as if to back him up, suddenly resumed its habitual violence.)

“But it stopped while you were being grateful.”

“So? What’s your point?”

“Don’t you think it’s interesting that, when your mind was occupied with being grateful, instead of with your relentless worries, your tremor stopped?”

“No. I was thinking about stupid things, and it was just a game. It wasn’t real. As soon as I got back to reality, the tremor came right back.”

“Do you think it might be helpful for you to practice being grateful once in a while, maybe a few minutes every day, just to give yourself a break from the tremor?”

“No. It’s not real. I have more important things to do.”

The most important thing that Travis had to do was finish his doctoral dissertation. He had been working on it, a paper on a certain spiritual interpretation of a particular bit of ancient scripture, for years and, to him, it was the most important document in the world. With the hope of shaking him up a bit, I cruelly suggested that whether he finished it or not, the world would probably never take much note. I also said that, if God really wanted a particular wisdom to make its way into print, He could do it, with or without Travis’s help.

Travis was furious. How could I mock him and his self-sacrificing life’s work?

I countered that, from what I could tell, Travis’s anxiety and fear was as much a drag on the world – and therefore on me, a fellow traveler in this world – as his thesis might be a benefit. Since, if all the saints and sages spoke truth, we are indeed all one, and each man is my brother, the suffering of each man is suffering to me. If by learning

to practice gratitude, silly types of gratitude at first, and sincere gratitude eventually, he could overcome his preoccupation with anxiety, he would be doing more good to mankind than a dozen inspirational theses about joy written by a man who was actually full of fear and out of touch with his own heart.

Travis disagreed. He said, in so many words, that he felt that his suffering was his due karma, and that he didn't really mind it. The impression I got was that by martyring himself (not his words) and focusing on his work instead of his troubles, he felt he was performing a virtuous, spiritual sacrifice. I had to ask him why he had even bothered to come to me if he didn't intend to get better.

His reply was that he was willing to see whether or not I could fix him, but if I couldn't, he was willing to accept that.

I pointed out, according to the precepts of his own Vedic-based faith, illness is always the result of one's own wrong thinking.¹ The Vedic scriptures that he studied proclaim that he has an obligation to try and correct his deluded thinking. His body could, if he corrected his erroneous thoughts, manifest the glory of his soul instead of manifesting his paranoias.

He agreed, and said that this was one of the subjects that he might someday write about. When I implored him to consider that he could institute these truths in his life instead of just writing about them, he drew the line. No, he felt the highest good was to be found in writing about these truths for others, not indulging himself in actual manifestation of them.

In other words, when all the fancy words and high-toned ideals were done with, he didn't want to change his habits. However, I shall never forget the instantaneous manner in which he was able to throw the anxieties out of his mind and the tremor out of his body by practicing even the most foolish and playful thoughts of gratitude.

Awareness

For those who want to try something different from gratitude, another, more difficult path is available. Sometimes, in the peace that follows from meditation, prayer, stillness, or gratitude, a person is able to simply appreciate what is happening around him. This use of the mind is called "being in the now," and it is very difficult for most PDers.²

Because of the difficulty most PDers have in living in the "now," I usually don't even recommend that they try practicing stilling the thoughts and focusing the mind on the various incoming neural sensations and their corresponding heart responses. I find that the gratitude exercise, practiced at least once a day for ten minutes, and practiced briefly every time an anxiety is banished, is easier for the beginner. Of course, if one can be perpetually grateful, he will be way ahead of the game, but this mind-shift may take some time, and one should not be discouraged if the change is slow. Be focused on the

¹ The Vedas, as mentioned in an earlier footnote, are the scriptures of Hinduism. The Vedas point out that events that have happened in the distant, unremembered past can be potential forces in the game of cause and effect. Past-life actions can set in motion present-life tendencies – either problems or blessings or both.

² This is similar to the mindset that one has while dreamily forming shapes from shifting clouds, or while creatively daydreaming.

process of changing, and not on the speed of the results, and you will have more fun – and therefore be ultimately more effective at changing.

As you will see in the upcoming chapters on recovery symptoms, many recovering PDers (those who are not locked into intentional negativity) do find themselves spontaneously enjoying themselves in the “now,” as their bodies resume the ability to release dopamine in association with positive thoughts. The experience is usually so novel that they even wonder: “What’s happening to me?! Who am I?” Often, their loved ones are stunned by the sudden, spontaneous change in personality when the PDer starts behaving as if he is at peace. I will share one quick example, one of my favorite recovery stories, before getting back to the business of this chapter.

However, this chapter is written for the many recovering PDers who, despite recovering from injury, choose, consciously or unconsciously, to maintain their habit of anxiety or even become increasingly anxious.

CAUTERIZING BRAIN CELLS OF WRONG HABITS

This is one of the most powerful techniques I have ever encountered. But first let me share with you the way that I “discovered” this technique.

I was trying to find some method of self-change that PDers might use that would allow them to use will power via peaceful thoughts instead of their usual grim, stern, determined version of will power. The problem with most of the exercises in self-change that I was finding in my studies was that they all relied on “will power.”

Well, the minute you say “will power” to a PDer, he immediately slips into the mode of thinking wherein he girds himself with adrenaline and prepares to fight. This is the very mode of behavior that they are trying to put behind them. While it may be true that much of the world needs to learn to buckle down and use some will power once in a while, PDers are past masters of will power. They brush their teeth with adrenaline and will power. They tie their shoes with adrenaline, fear, and will power. The last thing that PDers need is some sort of exercise that tells them to focus on their dreams and “make it happen using Will Power!” The average PDer, in response to this kind of encouragement, charges forward with adrenaline and a Do or Die! attitude. As far as this type of inspirational stuff goes, most PDers could teach the course. They don’t need more lessons of this nature.

So, one evening, after weeks of laboring over this dilemma of how to teach people to use their wills in a *gentle* manner so as to change the mindset to one of hope from one of cynicism, I suddenly realized that I was going to be late for the evening’s midweek inspirational service at the local chapel. I was supposed to be the reader that week, and I had failed to pick out some piece of inspirational literature to read to the group. I dashed upstairs, grabbed a magazine from deep in the tall stack of uplifting reading selections, and tore out the door.

Imagine my feelings when I started to read out loud to the congregation the following technique for overcoming bad mental habits. My prayer, my search for a really

good anti-negativity technique for PDers that could somehow use gentle will power but must not use sternly determined will power, was answered.¹

Here is the technique.

Getting rid of bad habits

Sit or lie down in a comfortable position. Select the troublesome behavior habit or thought habit that you want to get rid of. Then, repeat silently the phrase “I and my Father are one.” You can choose another phrase, if you prefer. I had one patient who refused to do the exercise because she didn’t like her father. If you don’t like this scriptural phrase, a phrase by which Jesus indicated his recognition that his soul was in communion with the whole of creation and the eternal infinite beyond creation, choose something else. You can say that “I and Love are one” or “I and all creation are one.”

Continue repeating this phrase until you feel a peace spreading through your chest. This may take some time. If you are particularly cynical, it may take an hour or so. Many people feel a peace coming over them within a few minutes.

(Now, here comes the next part of the technique, the part that, when I unexpectedly read it out loud during the inspirational service, nearly brought me to tears.)

After you feel peace filling your chest, keep saying your chosen phrase. Continue saying “I and my Father are one” (or whatever you have chosen) while enjoying the feeling of peace. Continue saying it until you sense the peace in your chest being replaced by a feeling of joy.

After you can feel joy spreading through your chest, realize that this joy is a manifestation of the divinity (or whatever sublime word you want to use) within you.

Next, speaking to this joyful aspect of divinity, say, “I command the God within me to cauterize the brain cells of this wrong habit.”²

Bear in mind that this is no vengeful or angry soul aspect that you are commanding. You are firmly commanding the pure joy of your own soul to do your rightful bidding. As you can imagine, this method, rare even for Yogananda, of first contacting the joy within *before* using the will power, and thus using joy, not backbone, as the driver of the will, was recognized by me as the answer to my prayers.

Resistance to this technique

Habits are formed through repetition. The brain makes certain shortcuts and pattern grooves when a certain behavior or thought is used repeatedly. In order to get rid of a habit, one can try to hide the old habit under the cover of new habits. However, it can take a long time for the brain cells associated with the old habit to actually break down or redirect their neural connections. To truly get rid of bad habits, one can selectively destroy the brain cells associated with the bad habits. Please, gentle reader, do not worry about running out of brain cells; the brain, once thought to be incapable of repair and regrowth, is now recognized as capable of enormous plasticity and repair.

¹ “What is Fate?” (excerpted from a lecture given Nov. 16, 1939 by Paramahansa Yogananda) Self-Realization, Fall, 1979, p. 6.

² “Cauterize” is a medical term. Cauterization is the extremely precise burning up of a tiny spot of troublesome tissue. The use of the word “cauterization” in this exercise is therefore a perfect word choice, connoting healing, precision, and careful, unemotional destruction of something bad.

Even so, when I have proposed the preceding exercise to some PDers, I have been shocked at how many have replied that they do not want to destroy any of their precious brain cells, even those built up to service bad habits of wrong behavior or thought patterns. I am always a bit surprised by this. When I ask these people, “If this was a cancerous cell, would you want it to die?” they invariably answer, “Yes! Of course!”

So I now advise that people consider a brain cell that was created and connected for the sole purpose of developing or perpetuating a wrong habit is no more worth keeping than a brain cell that is cancerous. For some reason, this line of thinking makes the following exercise more palatable. Still, I do find it curious just how many people are emotionally attached to their wrong habits. I have even been asked, “Can’t I just tell my wrong thought patterns to be inactive? Maybe put them in a special part of my brain where I can still have them, but they just won’t be so active? I hate to part with them, they are a part of what makes me *me*.”

If you look closely at this type of thinking, you will see the workings of the ego, or worse. What sort of person would say “I don’t want to *hurt* the cancer cells, I just want to move them to a part of the brain where they won’t grow so quickly...”?

I can comfort these people, somewhat, by pointing out that, even if you get rid of noxious brain cells, you will not lose the memory of how you used to be, or how you used to feel. Your memory cells are not being banished. You are only going to get rid of the cells that subconsciously work to compel you to perform unwanted behaviors or thought habits against your conscious will.

Checking it out

Before asking my PD patients to do this exercise, I tried it out on myself. Since experiencing a ruptured eardrum on a plane trip many years earlier, I had been plagued by a vicious, burning itching inside my ears whenever I ate most foods and, more recently, whenever I was feeling hurried or pressed for time. The problem was spreading; for the last two years, the burning and itching had spread so far that the roof of my mouth was engaged in this noxious behavior as well.

I decided that this allergic-type response, like all pain responses, must have at least some input from some signal in my brain. On some subconscious level, obviously, my body thought that I wanted to be having this painful, burning allergic response.

Therefore, I applied this technique. I silently chanted until I felt myself filling with joy and then commanded the joyful presence of divinity within me to cauterize the brain cells associated with this wrong habit.

After having repeated “I and my Father are one” for quite a while, I was, in addition to feeling joyful, in a deeply silent and still frame of mind by the time I got around to issuing this command.

The next day, it seemed to me that the burning pain was less hot. I practiced the cauterization technique again. Over a period of three weeks, during which I daily cauterized the brain cells of this wrong habit, the pain and itching completely disappeared.

Well, the whole thing might have been ready to heal by itself anyway. I selected another habit (my tendency to want to doze off during meditation). I applied this technique and the result came more quickly. My lifelong tendency towards feeling drowsy while meditating ceased after one practice of this technique and never returned.

I shared the technique with a friend. He tried it, and his asthma went away.

The great power of this technique is that it goes right down to the location of the problem and pulls it up by the roots. Replacing a bad habit with a good one can take a long time, and the bad one is still there, biding its time. This mental cauterization process, by which you calmly, joyfully tell your own brain that you have decided to make a change, leaves no room in the subconscious for doubt: you are in charge, and you are making changes.

The severe emotional resistance that I have sometimes encountered when I propose this technique to others is both an indication of its power and of the mixed feelings most people have towards actually making an improving change in themselves.

Therefore, I propose that, if this technique seems too powerful to you, you might want to sit down with your thoughts and carefully examine your reasons for not wanting to cauterize those brain cells that are impeding your conscious determination to make a change in yourself for the better.

PDers often cannot do the above technique

Now that I have explained this technique in great detail, I must add that PDers very often cannot perform this technique. I have taught this technique to my non-PD patients with great success. They can feel the changes taking place in their brain, and they observe with each passing day and repetition of the technique that the negative habit/thought pattern is melting away.

In the years before discovering the heart blockage in PDer, I now and again asked PD patients to do this technique. Invariably, insisted that they got no result. Because my non-PD patients invariably got good results from this method of self-change, I puzzled over this for several years, while intermittently asking PDers to please, give this technique a try.

Not until we discovered the heart blockage aspect of Parkinson's disease did we begin to understand why PDers cannot easily do this technique: by intentional design, they have forbidden themselves to perform the first step of the method: surrender the heart over to feelings of peace and joy.

Now, we suggest that PDers, before attempting this technique, first learn how, using the techniques in the Mind-Heart chapters of this book, to open their hearts. After they have learned to open their hearts to the extent that they feel peace, or at least calm, they will be able to start attempting to do this exercise correctly, and receive the benefits.

PDers should not be discouraged if it difficult to feel, in the beginning, calmness and peace in the breast. After all, they may have been working for decades to attain the exact opposite: feelings of nothing and wariness of mind. But no matter how many years a PDer has spend imposing on himself emptiness and despair, these conditions are reversible. Man's true nature, his soul nature, is joy. Any other emotions or denials are merely superimposed over his true nature.

I have worked with PDers who daily struggled with themselves in the battle of opening the heart for more than *half a year* who, in the end, won through. They were then able to surrender, at will, to the feelings of peace that are always present in the breast. After thus setting themselves up correctly to begin this technique, they can feel the subtle changes occurring in the brain while doing this exercise.

After they are able to perform this technique and get results, they can focus on various negative habits, such as “the physical and mental habits that keep my tremor going.” Other habits that a PDer can work on are the habits of specific dystonias (muscle spasms), habits of negative thinking and dopamine suppression, and habits of self-pity. Some people aren’t sure what habits are the “best” ones to get rid of. They imagine that they need to find some formal format of “wrong habit” in order to get results. This is the exact same mentality that they have cultivated for decades.

Do not worry about whether or not the correct habit is being focused on. Be childlike instead of analytical and judgmental: notice some small thing about yourself that might benefit from change, and focus on that one thing. Do not worry about whether or not you are selecting the “correct” or “most efficient” problem. And do not worry too much about the language in which you couch your bad habit of choice. You know in your heart what the specific problem is that you are trying to get rid of. Just describe that problem to yourself a few times, then refer to the problem as “the habit,” and then do the above technique while saying “I command the divinity within me to cauterize the brain cells of this wrong habit.”

DEPRESSION

One of my patients came up with a novel way for combating depression.

He had suffered from depression for years prior to his diagnosis with Parkinson’s. He mentioned “My Depression” at every session. He lingered over the subject, wanted to discuss it. He was certain that it had become a permanent part of his sensitive personality.

Even after his foot injury resolved and he began experiencing recovery symptoms, he insisted that his long-term depression was just a fact of life; I insisted that, since his Qi was now running correctly, he could physiologically produce dopamine if he could only bring himself to have an expectation of feeling joy. He countered, facetiously, that I made it sound as easy as flipping a switch. I replied that, yes, that’s how easy it could be. The next time I saw him, he proudly shared with me his new invention: the dopamine switch.

The dopamine switch

“I decided to create an image in my brain of a large switch, the kind you would use to pull several electrical breakers. What I do is sit down and close my eyes. Then I picture this big switch inside my head. Then, I get ready, and then I pull the switch. I know this will sound silly, but when I pull the switch, I actually do feel better.

“I’ve been doing it every day; I feel happy after I pull the switch.”

All the pretty colors

The next time I saw him, I asked him how his Happy Switch was working. He brushed that line of talk aside and told me about his new system.

“I think that merely pulling the switch didn’t give my brain enough time to really get poised for dopamine release. I’ve invented a new image.

“Now, when I recognize that I’m getting depressed, I image three spigots in my head. One is for serotonin, the second is norepinephrine, and the third is dopamine. These spigots control the flow from the three tanks of liquid neurotransmitter. Each

neurotransmitter is a different color: one is green, one is pink, and one is blue. They're all sparkly and fizzy.

"First, I get this image fixed clearly in my head. Then, I imagine that I'm opening all three spigots all the way. They gush into a holding tank, and they're fizzing and bubbling and they're just beautiful. Then, when the holding tank is full to the top, I shut the spigots.

"The holding tank is controlled by the switch that I invented last time, the Happy Switch. When the tank is full of neurotransmitters, with the colors all running together, I pull the switch. The result is *so* much better than before. I think that because this new process takes more time, I have more time to develop the expectation of joy. I can really feel the change flooding through my brain, and I feel so great."

Three weeks later, at our next session, I asked him, as usual, how his depression was going. He blinked at me quizzically. "Depression?"

"Um, yes, you used to tell me at every session that you were suffering from depression."

He clucked his tongue as if I'd recommended the Charleston as the newest dance craze. "Depression? Bah. I don't have that anymore."

It has now been four months. His depression has not returned.

So, whether you change your mind using bubbly juice or change your mind by commanding it – joyfully – to change, it does seem that, sometimes, to change the mind, the fastest way to do it is to address the mind directly and make it change.

CHANGE IS POSSIBLE

The reader needs to know that many people *have* succeeded in changing their attitudes. Many ex-PDer who have recovered easily have, prior even to being diagnosed with PD, spent time working on their attitude – forcing it to be positive even when it was difficult to produce the physiological (neurotransmitter) support. Others, sadly, have used their waning ability to access positive-mood neurotransmitters as proof that they are justified in their self-pity, resentment, blame, and other forms of unhappiness.

How long will this take?

A sweetly sincere PDer with whom I work every week is adamant that nothing can truly bring him joy, nothing shall ever be able to touch his hopelessly hardened heart. I loaned him the book *A Christmas Carol*, by Charles Dickens, because this book touches my heart every time I read it, and, surprisingly enough, he had never heard the story.

A Christmas Carol is a story of a miserable, miserly man who, in the course of a magical night, is blessed by observing his own past, present, and future as if seen through the eyes of an outsider. Alarmed and then transformed by what he learns, he emerges into the morning a changed, more compassionate man.

My patient returned the book the next week and said, "This book did nothing for me. It had an unlikely conclusion; I do not believe it is possible for a person to change overnight."

I asked him how long it should take. He replied, “A very long time. A lifetime. I am certain of it.”¹

Since then, primarily through the cutting-off-the-arms-and-torso game, he has experienced a very rapid change in his attitude. He detects a slightly improved ability to experience joy. These changes puzzle him. He distrusts their permanence.

The crying monk

When the previously mentioned Paramahansa Yogananda, a great yogi (1893-1952), was lecturing throughout Europe, he stayed one night at a Catholic monastery. After giving a stirring inspirational speech about finding a personal relationship with God, he was shown to his quarters by a monk. Just before the yogi stepped into his room, the monk asked him, “Please, sir, tell me, how long will it be before I am able to perceive Jesus Christ in vision or in the flesh?”

The great yogi said simply, “You could see Him tonight if you really wanted,” and went into his room.

The monk burst into tears.

For those who are uncertain as to the relevancy of the story, it must be understood that the monk had, of course, been imploring for years and years in the course of his daily prayers that the Lord might appear to him. And the truth of the yogi’s statement was not that this night in question was a special night, but that, on any night, if the monk’s desire had been untainted with doubts, fears, self-pity, self-hate, guilt, and a thousand other inventions of the ego, the ever-present Jesus would have been visible to him right along. But the monk’s preference for his ego-based fascination with himself and his own shortcomings and his own imagined sufferings were always greater than his desire for peace or joy. Therefore, Jesus, though ever-present, had not yet been visible to this monk’s preoccupied vision.

The monk, already knowing this at some deep level, and also realizing that, despite his begging prayers, he did *prefer* his self-fascination, also had to admit that,

¹ In a wonderfully ironic moment, during the very next session, I was putting his socks back on at the end of his FSR foot treatment; I pulled the socks all the way up to the calf.

He protested, “Don’t pull the socks all the way up. I always let them slouch down a little.” He chuckled and continued, “When I was dating my wife, years ago, she told me not to pull my socks up so tight; she said I should let my socks slouch down. She told me, ‘Only nerds pull their socks up tight.’”

“Ever since that day, I have never once pulled my socks all the way up.”

Recalling our past week’s discussion about whether or not a person could change quickly, I fixed him with a glittering eye. “For how many years did you pull your socks up tight? Thirty years? Your parents always pulled their socks up tight, right? Everyone in your family pulled their socks up tight! It was a deeply cultural habit. I have to wonder how many *thousands* of times you had pulled your socks up tight before you were unexpectedly told not to do it.

“And yet, you assert that, from the moment your date told you never to pull your socks up tight, you never did it again? It seems to me that, when you are motivated, you can change, in the merest blink of an eye, the habit of a lifetime. Hah! So tell me, why are you so sure you can’t change certain negative thought patterns? Is it possible that you are so emotionally attached to your negative, ego-caressing thoughts that you don’t really *want* to let go of them? Certainly, you were willing to change your sock-sense in a heartbeat rather than be thought of as a nerd. What would it take for you to decide to change your sense of looming negativity?”

He laughed, but was not convinced that his instant change regarding sock-habits had any message for him about his “unchangeable” negative attitude.

given his precious thought habits, the Lord was not likely to appear to him in the flesh for a long, long time to come. And yet, he realized at the same time, that it was this ego-drenched *certainty* that he could not overcome his doubts that formed his greatest impediment to success. After all, it *was* possible – the great yogi had just told him that it was possible to see him tonight. But it was up to him. Therein was the problem.

The monk's conviction that, despite the yogi's words, he *would* not see Him tonight was, obviously, a problem of his own making. His inability to see the Lord was not based on some universal law, but was based on his own disinterest in overcoming his own negative attitude. And so he cried.

“You could see Him tonight if you really wanted to” might also be expressed as “You could change your negative attitude today if you really wanted to.” This is the answer to “how long will this take?”

The struggle

We frequently get queries from PDers asking something to the effect of “How long will I need to struggle with this before I see signs of improved movement and a decrease in my weakness.” Another popular one is, “I am being *so* diligent; I am working *so* hard, why am I not getting better. The answer is, of course, that “surrendering to goodness,” “surrendering to love” means *stop* struggling. *Stop* working so darned hard. To let your inherent understanding, wisdom, patience, compassion and gentleness shine through, you must relax your grip on your heart.

Do not imagine for a moment that by ceasing to struggle you will therefore become lazy. People who tune out their fears and, instead, listen to their hearts and are led by their hearts are the busiest, most productive people on the planet. Certainly, they are more productive than people who can no longer even move because they are so busy being “analytical and productive” in their ego-driven minds even while their hearts are shut down and their bodies are a burden to themselves and to others. If you are wondering how long you will need to struggle with this self-change work, the answer is, “forever: or until you decide to stop struggling and let your inherent perfection and joy in living manifest themselves.”

Crying

Crying can be pivotal in recovering from Parkinson's. Cry with tears, cry out loud, cry with self-pity for your self, for all the times you've been hurt, disappointed, mistreated or misunderstood. This is not the beginning of selfishness, it is the end of dishonesty. Stifling your feelings is not the same thing as not having them. If you think you can't cry, imitate the sounds and motions of a person who is crying.

Cry. And if you feel rage welling up in you alongside the tears, give expression to your rage (with words directed at the walls of a silent room, or by pounding a pillow). If you are spiritually inclined, you can even let God know how you feel about having been hurt (resentful, angry, bitter). Berate him as a two year old berates his good mother or good father. Don't worry about offending God; He/She/Love will be pleased that you're communicating honestly for a change.

A DIFFERENT TYPE OF MEDITATION

A PDer sent the following meditation to me via email. She referred to it as “Tonglen Meditation” and said that it was a powerful technique.

The first step of this “meditation” is to say to oneself, “Other people feel this.” This stage of courage, admitting that one is not unique in his pain or discomfort or fear, is the starting place. Any time that thoughts of isolation, desperation, or personal burden well up, repeat this phrase until the full meaning of it is felt. “Other people feel this.”

The next stage of courage for this particular meditation is to mentally say, “May we all be free of this (type of pain or suffering).”

The third stage of this meditation is to think to oneself, “Since I’m feeling this anyway, may I be feeling it so all others can be free of it.”

I am a bit ashamed of my immediate response to the PDer. I dashed off a quick email reply to her that said, “I’m a little surprised. I thought that we all learned this concept as young children. Isn’t this how most people get through pain and problems? Seriously, is this concept new to anyone?”

She wrote back to me, “To be honest, I sadly did not learn that as a child. I did learn to reason my way out of problems which I now realize just furthered the disconnect. I didn’t learn to trust.”

I wrote back to thank her for her sincere response, and apologized for my arrogance. If this type of meditation resonates with you, please practice it.

For myself, I prefer the following affirmation:

“The laws of cause and effect permeate all the universe, right down to the perfect balance between the number of protons and the number of electrons. Even our very thought waves can act as a force for good or evil, and according to the laws of equal and opposite reactions, if I perpetrate negative thoughts, the results of those negative thoughts will someday come home to roost in my consciousness.

“All illness, pain or suffering is the result of transgressions against natural law, whether initiated in this life *or in some other*. If I am in pain, it was myself that set the transgression and its subsequent pain in motion. I can learn about myself, and learn how to change myself, by studying carefully the suffering that I am feeling. I should never forget that others have the same suffering; this will help me to remember that my transgressions are not special or unique. Even in my transgressions against natural law, I must humbly say that I have not been especially original. Therefore, let me learn from my mistakes and be grateful, celebrate, because the pain I am now feeling, the price I must pay for my ego-based blunders, is even now being worked off. The sooner I have worked off my errors, the sooner I will be free of them, the sooner I will be closer to Wisdom and Peace.

“And if, in the past, or in past lives, I have ever prayed sincerely that the illness or suffering of another might be transferred to me so that I might share or shoulder the burden, I must consider that my present suffering may be the answer to that long-forgotten prayer. In which case, my pain or suffering is the greatest possible honor – it is the answer to my heartfelt prayer. If this is then proof that my heartfelt prayers are heard and answered, then may I remember to always be sending positive thoughts, for every thought is a form of prayer.”

SLOW AND STEADY WINS THE RACE

Despite the fact that overnight change is possible, for some people, it isn't practical to assume that everyone will change overnight.

Therefore, start off small, but steady, with these techniques for shifting the mind. Do not be worried about your progress; the type of thinking that measures, compares and contrasts resides mostly in the fear part of the mind. Spend a little time each day breathing slowly, closing yourself off to the relentless yammering of the world. Re-tune your heart radio, just in case it is misaligned.

Tune in to a simple "you" based chant; banish the chorus of "I, Me, Mine."

Admit cheerfully that you are imperfect: demonstrate your harmless imperfections and maybe even with fearless, even humorous honesty, point them out to others.

At the same time, stop judging others: if you cannot understand someone's actions or the source of some troubling event, assume the kindest possible motive.

Track your thoughts lovingly and, like a gentle shepherd, correct them when they head towards the quicksand of negativity. Cull the toxic weeds of anxiety which might poison your flock of thoughts and give them away to the universal incinerator.

Spend a little time each day immersed in timeless eternity: let some golden moments slip by without counting the minutes.

Did I mention dancing in the silent kitchen while waiting for the water to boil? Imagine the flowing movements of the dance and let your body follow the thoughts of the dance. Don't force your body to obey mental commands of movement: have some fun.

Joyfully command your mind to be the mind you want it to be: burn up the brain cells that are misbehaving – you can always grow new ones.

Invent some amusing image that directs your brain to the kind of mood you want to manifest.

Cry.

Rage.

Be peaceful.

Be grateful.

Be.



Fido circled his master's grave three times and then lied down, never to move again.

- American folklore

APPENDIX FOUR

DOPAMINE RELEASE: PDERS AND DOGS

As noted many times, many PDers are primarily concerned with how *much* dopamine they have, how *many* dopamine-making cells they have lost. What they should be wondering is “Why can’t I mentally initiate dopamine *release*?” The amount of dopamine on hand and the phenomenon of dopamine release are two very different issues.

Dopamine release, at its core, is based on the underlying sheer joy of being alive and the feeling of unconditional safety that accompanies that joy. That joy flows in spite of whatever outrageous slings and arrows may happen to be flying at any given moment.

Actually, as you have already read, this same thrill-of-being-alive feeling is necessary to trigger the release of adrenaline, as well. However, for our purposes, and because most PDers imagine that dopamine is the only neurotransmitter that has a release problem, this chapter will primarily address this feeling as it relates to dopamine release or inhibition. But to start with, I will use an example that doesn’t even involve dopamine or adrenaline.

FAMILIAR EXAMPLES OF NEUROTRANSMITTER INHIBITION

The nursing mother example

Consider the example of a vigorously healthy woman who finds herself unable to nurse her new-born baby because her milk will not “let down.”

The mother may be physically healthy and all her neurological systems are potentially able to work correctly. However, though her breasts are turgid with milk and the baby is crying lustily, her preoccupied mind will not allow her to surrender to the peaceful feelings necessary for triggering the release of the neurotransmitters involved in letting down milk.

Her milk might not “let down” as long as her brain is preoccupied with thoughts such as “I should never have given the dossier to Williams, of all people, and I’m not so sure I wanted to have a third baby since its turned out to be yet another boy. *Why* couldn’t it have been a girl? And I’m so furious with my husband for not even being here until two hours after the baby was born,” or the more tragic “One of the twins died; how can I ever think of this new child without thinking of the one that died?” and so on, and so on.

As the new mother’s worries, sadness and/or resentment carry her away mentally, the screaming, hungry baby must finally be pacified with a bottle of formula: no breast milk is forthcoming.

The mother is healthy and has all her neurotransmitters and hormones. However, she cannot *access* them because of a mental/emotional blockage, one that is locking her into adrenaline-based thought patterns.

You can't nurse a baby when, from a neurotransmitter perspective, you are running from a rhino. When your mind is racing and the heart is not dominant over the mind, the necessary milk drop-down neurotransmitters will not be released.¹

Adrenaline-dominant attitude inhibits dopamine release

In the same manner, a PDer whose underlying, deepest, most internalized thought patterns are excessively cautious, guarded, vigilant, intellectual and/or cynical who has therefore not had, maybe for decades, maybe for most of his life, any experience with the types of thought patterns that trigger dopamine release, may not be able to have his dopamine "drop down" just because his injuries have healed. Even if the PDer is superficially cheerful and pleasant, if his deepest motivations are increasingly hedged by caution or wariness, he is inhibiting dopamine release.

The PDers who get lost in partial recovery tend to be locked into a highly negative mindset.

Many PDers have these negative types of thought patterns; like the mother whose milk is present but will not drop down, these PDers have dopamine, but they are not able to release it.

The next pair of analogies may help demonstrate this principle.

As an aside, the next analogy was partly selected because many PDers falsely assume that they must *necessarily* have a bad mood, fear, or depression because some limb is imperfect, weak or trembling. To counter this self-serving loop-type thinking, I'm including these very familiar examples to show that animals, including humans can still

¹ The nitpicker may want to argue that, in this case, the problem is the failure of hormone release, and not neurotransmitter release. Historically, when the small instructional and communication molecules (hormones, neurotransmitters, and others) were first being discovered, discussions raged over nomenclature. Researchers wanted to neatly define the distinctions between hormones, neurotransmitters, "messenger molecules," and so on. I went to college during this time and memorized lists of which was which. Those lists quickly become obsolete.

Today, although we bow to custom in referring to some compounds as hormones and others as neurotransmitters, the naming rules are increasingly meaningless. The interactions of all chemistries in the body are so interrelated that the old distinctions, in which messenger chemicals were defined by the anatomical structure that released the chemical, no longer apply. Now we know that chemicals that are emitted from a gland (a hormone, in the old days) may *also* be released directly into the blood from cells, and may even be released from neurons. Not only that, even though certain messenger chemicals work directly on an organ, they may simultaneously work on certain cells, and may also trigger other messenger chemical events in the brain. So the old distinctions between hormones and neurotransmitters are falling apart.

A brilliant, very readable book on this subject is *Molecules of Emotion*, by Candace Pert, PhD.

For a pertinent example, we now know that dopamine is present in the blood, as well as in the brain. But even though dopamine travels in the blood, and not only from one nerve to another, we still adhere to the old custom of referring to dopamine as a neurotransmitter. By the way, blood dopamine does not cross over into the brain, and the amount of dopamine in the blood has no relationship whatsoever to the level of dopamine in the brain.

Levodopa can cross the brain barrier. This is why levodopa, which can cross the brain barrier, is used instead of dopamine in the treatment of Parkinson's disease.

have full use of dopamine or other joy-related neurotransmitters even though not physically healthy in limb as long as deepest, underlying attitude remains positive.

The two dogs analogies

Iris, the three-legged dog

Picture a dog who, due to abuse, has lost one hind leg. For myself, I am thinking here of a neighbor's dog, Iris, a black lab-terrier mix adopted from the local animal rescue shelter.

Iris licks my hand through her fence every morning when I take my walk. Iris, who lives one block from the elementary school, also happily tackles the job of licking the hands of every passing school child, morning and afternoon. When she licks your hand, she fixes you with her glittering eyes and her whole body wags with joy. Iris lost her leg through an intentional act of cruelty on the part of her previous owner.

Iris has no dopamine release problems. She does have a very serious structural problem, a physical problem.

A three-legged dog has, and will have, many structural problems for the rest of its life: the remaining hind leg will always need to be carried closer to center when he walks. This shift will always put a strain on his hips, spine, and neck. His shoulders may also shift a bit to carry more of the body-balance burden. These structural misalignments may create very real problems down the road in the form of arthritis and nerve pinching in some of the body parts that have shifted.

However, even with three legs instead of four, nearly all three-legged dogs can move "perfectly normally." Like Iris, they can usually chase bicycles, catch a Frisbee, and romp with other dogs.¹

The point here is that the loss of a leg does not cause a three-legged dog to be depressed. The loss of a leg will in no way inhibit its ability to initiate movement. The loss of a leg will not inhibit the release of dopamine.²

I wrote this above point in italics because many PDers insist that they must necessarily have a negative attitude because they have a tremor or some slight or large movement problem. This is incorrect. They must necessarily have a negative attitude

¹ I find it highly significant, from a self-image point of view, that Iris scratches her right ear with her missing right leg. When her right ear itches, she curves her body as if she was scratching her ear with her foot. The stump of her leg moves vigorously back and forth in the open air, touching nothing. Although no foot is actually touching her ear, Iris leans into the process as if she was deeply enjoying the sensation of scratching her ear. When the attention to the ear creates the desired sensation of enough scratching, she stops pumping her leg and shakes her head, satisfied. Iris, despite the missing leg, has a complete sense of body. Emotionally and energetically, Iris is not missing her leg.

² One of my colleagues in the PD project has a father who lost his arm early in life. My colleague told me "Until I was eight years old, I never even noticed that there was anything missing. My dad was perfectly normal in every way." But PDers, so attuned to problems and negativity, often point to the slightest bit of gait problem or the slightest bit of arm rigidity as the *reason* that they are depressed. These people are wrong. They are negative to start with, on the lookout for trouble. Any deviation from "perfect" will therefore loom large to them.

Physical immobility of a limb, even the loss of a limb, is not necessarily a justification for being depressed or locked into a mental state that prevents dopamine release.

because they are still dealing with some unaddressed fear: they are still locked into sympathetic (danger) mode.

Fido: loyal unto death

Now, picture a different dog: a dog who is a picture of health. When this dog suddenly loses his beloved master or brother dog he may quickly become despondent.

This unfortunate dog, who is perfectly healthy and chock full of neurotransmitters may, due to his broken heart, quickly become dispirited. He may move more and more slowly, and with increasing difficulty, until every movement is a colossal effort. Within a matter of days he may become listless, cold, and even shaky, tremory. He may lose interest in eating and, if force-fed, may soon lose his swallow reflex and be unable to take food by mouth. This dog may likely go on to die.¹

This dog is, from a structural standpoint and from a physiological standpoint, perfectly healthy. However, his heart is broken and he can no longer initiate movement. Like the healthy mother who, for emotional reasons, cannot let down the neurotransmitter that will allow the release of her milk, the healthy but broken-hearted dog cannot “let down” his plentiful supply of movement and mental neurotransmitters, neither dopamine nor adrenaline.

These two dogs illustrate two types of problems. The first dog has structural problems. He has tangible, measurable health problems. However, he does not have mood or movement initiation problems. His sheer joy in being alive allows him to release the neurotransmitters that activate his body.

The second dog has no structural or neurological problems, but he has an emotional problem that prevents the *release* of neurotransmitters. As a result of this emotional problem, he cannot initiate movement. He may even tremor. This problem will worsen in a spiraling or snowballing manner until, abject and immobile, he dies.

Structural problems in Parkinson’s – compared to those of the dog

A person with Parkinson’s disease has *both* structural and emotional problems. Let’s first consider the structural problems. These problems result from the foot injury. These structural problems cause many PDer to have a similar “look,” even in a still photograph.

When looking at a still photograph of a PDer, the observer cannot know that the PDer has a movement initiation problem or tremor. However, the drooping eyelid, the sagging cheek and corner of the mouth, the forward-jutting head and the bent arm, among other symptoms, may allow the doctor to recognize a person with Parkinson’s disease, even in a still-photo. These body changes are structural rather than emotional. If a PDer has these problems, they will be in place whether the person takes L-dopa medications or

¹ The state of Hawaii used to have an importation policy for dogs: dogs brought to Hawaii from out of state had to spend six months in quarantine. The intent was to insure that dogs did not bring out-of-state diseases to the islands. This policy has been changed. Why? Because the dogs usually died before they finished the six-month quarantine period. These dogs were healthy in every way. After many years of this policy, and the deaths of hundreds of healthy animals, the conclusion was made that these dogs had died of broken hearts. The policy has been disbanded. Dogs must now get various health tests prior to arrival in Hawaii. If all the tests and paperwork are in order, a dog can get through “immigration” in less than an hour.

not. They will be in place even when the person is having a “good” day or a “good” hour during which he can move normally. Like Iris’s missing leg, these problems are not related to neurotransmitter release. In PDer’s, these structural problems are due to the backwards-flowing Qi in the Stomach channel and the sequelae of this Qi pattern.

If the PDer happens to feel fleeting joy

Like the three-legged dog, the PDer will be able to move “perfectly normally” when he is in a phase of unusual, for him, emotional competency – when unexpectedly happy, for example, or when “high” from the antiparkinson’s drugs. As with Iris, the three-legged dog, the PDer’s structural problems will be compensated for by those muscles that remain functional.

To the casual observer, the PDer’s movements at these times will appear almost normal. This does not mean that he is actually moving correctly. However, the PDer’s seemingly effortless movements, like Iris’s, may distract the viewer so much that the casual observer might not even notice the structural problems.

Many neighbors, including me, have known Iris for quite awhile before they suddenly realize that she is missing a leg. She moves so normally.

A PDer, when stoned on dopamine-enhancing drugs or when feeling unusually good, will, like the three-legged dog, appear to move fairly normally despite his structural problems.

Earlier in this book, I mentioned that a few people with Parkinson’s disease have specific activities or day during which they can move with perfect ease: for example, the painter who, within ten minutes of being placed in front of his easel, could stand up and paint with fluid, graceful movements.

These PDer’s, like Iris the dog, still have detectable structural problems. However, the PDer’s can move easily during those special times when their highly compartmentalized minds are engaged in one of their rigidly defined “safe” behaviors.

In this condition, with its rare flow of dopamine, they can move easily. *There is no insufficiency of dopamine.*

Like the PDer who is mentally altered by dopamine-enhancing drugs, a PDer during moments or hours of rare joy has brief periods of normal-appearing movement. During this time, the PDer’s healthy muscles must splint for (take over the work of) non-working muscles. The body is able to do that easily, if the mood is light. The comparison between the PDer when he is drugged or in one of his few specific situations in which he lets down his perpetual guard, releasing dopamine in spite of his injured body, and Iris the three-legged dog is an apt one: due to underlying joy of living, whether natural or drug induced, they all move well *despite* physical imperfection.

But what happens to the PDer when he stops doing his “safe” activity or his drugs wear off? The PDer will, once again, not be able to move well.

Animals don’t get Parkinson’s disease

The question arises, why is a dog able to permanently “override” his structural problem, but the PDer is not?

There is a significant difference between the structural problem in the dog and the structural problem in the PDer: the dog has dealt with his injury. If a dog’s injury was traumatic, he may have dissociated at the time of injury. However, as soon as a dog feels

safe again, he will switch his body back over to the parasympathetic system. He will lick, chew, and then lick some more at any body area that calls for his attention. A pain signal from an injury is a signal declaring “Notice me! Care for me!”

When the dog licks the problem area, when he notices and cares for the problem, the pain signal turns off. This tender attention, in turn, turns off the adrenaline response. Furthermore, the gentle stimulation from his teeth and tongue will have encouraged the flow of energy in the wound site or problem area.

The disrupted electrical channels will soon have formed new electrical loops that flow easily and in the right direction. Even in the case of loss of limb, the energy will still be able to flow in the right direction even though flowing in a modified, somewhat diverted route.

The PDer, on the other hand, dissociated at the time of injury and has held onto that dissociation. The PDer has not decided that he is safe. He has yet to pay adequate attention to the wounded area.

Therefore, the dissociation response, a response that tips the neurotransmitter balance towards low levels of adrenaline and inhibition of dopamine, will continue to dominate the PDer's response to his foot injury. The electrical system in the area of the injury will, without some healing attentions, remain disrupted at the injury site. The site will eventually develop enough electrical resistance in the area that currents will have difficulty moving past the blockage.

This is what is seen in PDer's: the electrical resistance become great enough that, at some point, the current that is supposed to pass through the foot begins to follow the path of least resistance: the electrical energy in the leg begins to flow backwards. The physical changes related to the structural problem begin to appear. The PDer continues to ignore these early symptoms, just as he ignored his foot injury.

The negative, anti-joy mental processes that are supposed to dominate the mind during an emergency become increasingly dominant due to the influence of the sympathetic nervous system. During an emergency, during the time when one's attention must be focused on saving the life, one should not be focused on enjoying the sensations of having a body. Eventually, these mental habits provide reinforcement for the PDer's shift into the sympathetic mode.

The dog pays attention to his injury in a productive manner. Dogs do not develop Parkinson's disease. No animal develops Parkinson's disease.¹

The closed heart factor in Parkinson's

A PDer has created, by virtue of allowing his mind to regulate his heart, a “closed heart” situation. The phrase “mind regulating the heart” will be explained in great detail in a later chapter. The emotions, the ability to register feelings, may only be closed with regard to the foot, or they may be closed to anything and everything.

¹ For research purposes, lab animals are poisoned or genetically manipulated so that their bodies imitate the movement inhibition problems of PD. This lab condition is called parkinsonism. This condition only superficially resembles Parkinson's disease.

When the heart is significantly closed, when the thrill of being alive is absent or inhibited, healthy amounts of dopamine cannot be released. When the heart is too empty, movement inhibition, frailty and, finally, death will occur.

There are other illnesses that cause movement inhibition besides broken or closed off hearts. For example, stroke, brain tumors, or polio can all cause movement inhibition. But in these illnesses, the movement inhibition cannot come and go; it is not mood dependent. In Parkinson's, placebo studies have proved that the movement inhibition of PD is mood and expectation dependent.

Anyone who has lived with a PDer knows all about this: the PDer can only move as well or as poorly as he expects he will. This is what differentiates PD from other movement disorders. This is one reason that PD is so difficult to pin down, diagnostically. This is one reason that the "cause" of PD has remained so elusive.

And even if researchers do suspect a mental component, no one wants to be the first to accuse these people who are, in many cases, mental giants, super-responsible, hard-working, selfless and philanthropic PDers of being in the throes of a self-induced mental/emotional illness.

Illness that includes a mental component is still thought of, in western cultures, as being somehow less "real" and more shameful. In eastern medicine, mind is recognized as the most important factor behind *all* illness.

In the case of the dying dog in this example, he is dying from a "broken," or empty, heart. Anyone who has deeply felt the loss of a loved one knows how it feels when the area of the heart seems to be missing some of the vibrations that previously filled it. The broken heart may feel as if a part of the heart is physically missing. The fullness of one's heartwaves (similar to radio waves) and the accompanying heart-nerve signal to the brain is altered when a loved one is no longer alive.¹

In a broken heart situation, there is an actual decrease in the accustomed pattern of heartwaves. This situation, in which the heartwaves and the accompanying heart-nerve responses are insufficient to trigger the healthy, normal release of thought and action neurotransmitters, can lead to decreased mobility, frailty, poor memory, and even death.²

¹ The subject of heart-nerves will be discussed in a later chapter. Briefly, these are the nerves that make up a large part of the heart tissue. These nerves are *not* a part of the system that regulates the beating of the heart. These heart-nerves communicate with the brain, and instruct the brain in many arenas, including whether or not incoming sensory information or thoughts should be processed using adrenaline or dopamine.

² Probably the single greatest difference between eastern and western medicine is that western medicine believes that the wave processes of the body are the result of chemistry. The eastern system recognizes that, from the moment of conception, an individual's primary wave patterns initiate a chemical dynamic, and the chemistry then regulates the secondary wave patterns, which further influence the chemistry, which then creates more wave patterns, and so on.

The western method has no way to make sense of the personality differences between identical twins, let alone the manner in which joy and fear alter the chemistry of the body. The western method cannot explain the placebo effect, let alone the ability of yogis to sit motionless for weeks without breathing by stilling their wave patterns. Eastern medicine recognizes that almost all of the brain's interpretations of sensory signals are determined by the mindset, which is in turn determined by the degree to which the heart is open (resonant with the Love that pervades the universe, and unoccluded by ego.)

Intentional heart emptiness

In the cases of the PDer and the broken hearted dog, the heart becomes empty enough that it can no longer trigger the release of neurotransmitters, a sort of “I wish I was dead” kind of emptiness. However, the dog’s case and the PDer’s case are significantly different.

In the case of the PDer, the emptiness in the heart is not necessarily caused by a sudden loss of accustomed heartwaves but, in most cases, is caused by an intentional guarding of the heart. The PDer, first consciously and eventually, from habit, subconsciously, is living as if his heart was closed: he PDer has cultivated a mental state that corresponds to the dissociation condition, a condition in which the signals from the heart are sedated. I think of this condition as “closed-off heart causing an empty heart.”

In the case of most PDer's in our experience, the heart is being held shut by the mind, and is therefore empty, deficient in the ability to trigger neurotransmitter release. The PDer’s heart may be bruised, it may be afraid of being wounded. The heart may be stunned into silence. But in the case of the PDer, the actual condition of the heart, whether wounded or healthy, sad or frightened, is unknown: because he has learned to dissociate from physical and emotional pain, the PDer has turned off his access to his heart.

Although the PDer may or may not champion the rights of others and be kind to animals, although he may devote his life to community service and be utterly selfless when assuaging the feelings of others, his heart is not complete: he cannot experience his *own* feeling. The guardedness of his heart, as he protects himself from physical and emotional pain, eventually accumulates to the point that he cannot feel, in his own breast, the physical and emotional joy inherent in life.

Heart emptiness from tragic loss

In the case of the dog, the heart is wide open and empty.

The dog does not have a choice in bringing his beloved back from the grave. Happily, a PDer always has the option of relearning how to open his *intentionally-closed* heart.

You can argue Yin and Yang versus physiology until the cows come home and never prove that one school of medicine is better than the other. They are both powerful and valuable. But in its essence, allopathic (western) medicine, derived originally from the German and French fascination with physics and built upon the principle that humans are a very sophisticated form of clock-type mechanism, can only explain or manipulate how *the components* work. Eastern medicine can actually explain how and *why* humans work.

