RECOVERY FROM PARKINSON’S DISEASE

THE CAUSE AND THE EFFECTIVE TREATMENT OF PARKINSON’S DISEASE ACCORDING TO PRINCIPLES OF TRADITIONAL ASIAN MEDICINE

JANICE WALTON-HADLOCK
RECOVERY FROM PARKINSON’S DISEASE

THE CAUSE AND THE EFFECTIVE TREATMENT OF PARKINSON’S DISEASE ACCORDING TO PRINCIPLES OF TRADITIONAL ASIAN MEDICINE

JANICE WALTON-HADLOCK
To the people with Parkinson’s who have participated in this noble experiment, to my colleagues on the Parkinson’s Treatment Team and the many acupuncturists, doctors, physical therapists, and other health professionals who have contributed, and to my loved ones who have humored my, listened to me, and cared for me, I humbly extend my deepest thanks.

This research would not have been successful without the insights of Chris Ells, Rebecca Weinfeld, Laura Walter, Clay Walton-Hadlock, and Darrol Hall of the Parkinson’s Treatment Team.

As for the help given by my Steve, no words suffice. Steve, you are love. Thank you for your steadiness and your puns.

And when my patients, were buffeted by doubts and fears and I was alternately baffled and tormented by the seeming hopelessness of the quest, I clung to the words of Paramahansa Yogananda: “Through Thy grace the sudden shafts of wisdom will dispel error accumulations of countless centuries.”

To You, all love and gratitude,

- J
# Table of Contents

## Part One: Understanding Parkinson’s Disease

1. Parkinson’s Disease: Uprooting the Cause .......................................................... 1
2. Feeling No Pain: A Few Case Histories ............................................................... 21
3. Channel Theory ..................................................................................................... 45
4. Blocked Qi: Trouble ............................................................................................. 61
5. The Development of Parkinson’s: Changes in Channel Flow ............................. 65
6. The Western Understanding of Parkinson’s Disease .......................................... 85
7. West Meets East .................................................................................................. 105
8. Partial Recovery .................................................................................................. 129
10. Placebos and Parkinson’s Disease ................................................................. 159
11. Dopamine release: PDers and Dogs ............................................................... 173
12. Matters of the Heart ......................................................................................... 183
14. The Case of the Missing Heart ......................................................................... 225
15. Turning Off the Dissociation Response ............................................................ 251
16. Untitled and unfinished ................................................................................... 275
17. Untitled and unfinished ................................................................................... 287

## Part Two: Treatment Techniques

18. Demystifying Tui Na ......................................................................................... 401
20. Applying FSR to Legs and Feet ........................................................................ 439
21. Resting FSR

22. FSR: How Does It Work? A Hypothesis

23. Techniques for Turning Off the Dissociation Response (Unfinished)

24. Unfinished
“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, 1817

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.” 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


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 all three 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-42\(^1\) 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 Qi flow in their feet. One person did not: Tim, age 12. 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.”

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, sometimes for years, long before the external, visible tremor ever appeared.) Tim said no to my

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 sums up people with Parkinson’s 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.
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-PDers, 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.


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. 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 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. Many, even those who had

---


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 PDers 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 PDers 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 PDers’ 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, adrenaline, not dopamine, is used primarily during times of wariness, danger or injury. Dopamine is used in these same muscles during times of calm or joy.

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
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. 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 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.

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 brain dopamine that supports conscious mental and motor function cannot be fully released.

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, we have reason to believe 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.

2 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
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 behave as an integrated unit.

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.\(^1\)

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.

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.

\(^1\) The electrical aberration at the center of the foot can be detected by machine. A visiting acupuncture professor from China, the renowned Dr. JuYi 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 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.
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 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 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.)
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. 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.

Backwards flowing currents can be very, very bad

In the PDer, though, the injury never fully heals because, emotionally, it “never happened.” Not only that, but, in terms of body chemistry, 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 scenario, the person needed to get

---

1 Technically speaking, dopamine is also used as a trigger for the sympathetic, fear-based system. A person without enough sheer joy of 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 area of neurotransmitter overlap and refer to the two systems as the adrenaline and the dopamine 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 dopamine system. For 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.
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 trembling, 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-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

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.
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 feels as if he is not mentally or emotionally associated with his injured body part, or even, eventually, 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. This physiological process 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.¹

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

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.
“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 inhibition of dopamine occurs in any anyone maintaining a mental or emotional habit of dissociation response – not just a person with 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 PDers foot the 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 mix, the ability to initiate movement and control 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.


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 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.

We know from the responses of recovering PDers that these nerves are dormant, and not 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, 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 word “lose” 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.
If the heart’s sympathetic nerve response is diminished due to the emotional shutting down of 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.  

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 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 sustain a non-wary heart.

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.


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 the diminishing sympathetic heart-nerves system and away from the nearly forgotten parasympathetic (vagus nerve) system.

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….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 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’s ongoing dissociation response can cause a rapid increase in frailty-type symptoms after the adrenaline-producing injury begins to heal. Even if dopamine production is once again anatomically possible, a heart oriented towards the wariness mode for most of life’s situations may be unable to register the feelings needed to trigger dopamine release. Frailty can ensue.
“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

Nearly everyone gets a foot injury at some time or other. Most people heal from their injuries. The remarkable thing in my PD patients was that their feet had remained as if injured – and yet relatively painless – for decades. We were to discover eventually that nearly all PDers that we’ve worked with have intentionally dissociated from their foot injuries.

This chapter is mostly made up of case history snippets from my earliest PD patients. At the time I recorded these case histories, I noted the pervasive attitude of “the injury didn’t hurt.” But I didn’t wise up to the full significance of this attitude until years later. To give the reader an advantage that I didn’t have, I will precede this chapter’s case histories with some information about the dissociation response.

Dissociation

Dissociation is a recognized mental/emotional/physiological event that can occur in cases of severe trauma. Dissociation causes a shift in heart rate, blood circulation patterns, consciousness and neurotransmitter release. You don’t need to learn all the chemical changes that occur during a dissociation response to severe trauma: the most important thing to remember is that dissociation is a form of sympathetic nervous system response. A sympathetic response (as opposed to a parasympathetic response) swings the body over to an adrenaline-dominant, dopamine-minimized condition.

When most people think about an adrenaline response, they only think of the “fight or flight” response. The adrenaline response actually can take many forms, including “fight,” “flight,” “freeze,” and, most common of all, “wariness.” These responses to danger or challenge feature an increase in adrenaline and a concomitant increase in heart rate and blood rushing to the muscles. However, while these types of responses are the best known, they are not the only types of adrenaline-dominant/dopamine-inhibited sympathetic nervous system (danger system) responses.

The dissociation response – even though it is a type of sympathetic (danger) system response – can create a relaxed, even sedated feeling, and an almost pleasant sense of being OK.

During a dissociation response, the body switches over to the sympathetic (danger) nervous system. Therefore, it inhibits dopamine release and allows adrenaline release. But in this type of response, adrenaline, the chief neurotransmitter of the sympathetic system, is released at very low levels, not high levels. Therefore, the heart and breathing rate become very slow. During this type of response, blood moves towards the interior of the body, away from the muscles and the periphery. The extremities may even become cold. At the same time, endorphins (opiate-like chemicals) are released into the bloodstream. The endorphins can be released in sufficient quantities that one becomes calm, falls into a peaceful sedation, or even loses consciousness.
**Dissociation in prey animals**

Among prey animals, dissociation usually occurs when a predator’s teeth or claws have actually cut the skin of the 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. The severely slowed heart rate, slow breathing, and the sedated condition of the prey animal can cause it to become cold and limp. It may appear to be 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 more fun elsewhere. Several minutes to a few hours 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 goes limp. 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 may lose interest.\(^1\)

**Dopamine inhibition**

Remember: dopamine release is inhibited during any sympathetic (danger) system response. As you will read in a later chapter, midbrain dopamine release should decrease during the initial healing phase of a significant injury. Dopamine release is also inhibited during a

---

\(^1\) *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, addresses dissociation, especially in young people.

It may be of interest for the study of Parkinson’s that highly intelligent children who are regularly abused can learn how to move easily into a dissociative state when the abuse is about to start. In this condition, the body switches over to the sympathetic (danger) system: dopamine release is suppressed and adrenaline release is activated, but the adrenaline is released at very low levels. Opiate-like chemicals (endorphins) are released and a sense of numbness pervades the body.

In humans, the dissociation response can, in some cases, allow a person to imagine that he is not the one being hurt: he can perceive the injuries he is receiving as if he is watching the event from outside his actual body. In this way, he can observe the pain but he doesn’t feel that the pain is actually happening to him – the injury is being perpetrated on his numb body, while he observes the event from an outsider’s perspective. While not all PDers have this type of outsider’s perspective, many PDers maintain this perspective almost continuously.

Sometimes, during a dissociative response, if the opioids are released at high enough levels, a person can have a response similar to a drug overdose: coma and even stopping of the heart. To rapidly reverse the dangerous level of heart stoppage and coma induced by very extreme dissociative response, doctors can treat a person in an excessively severe dissociative state by administering the anti-opiate drug noxalone.

Some people who have learned, often during abuse, the mind-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 learn to revert frequently to a dissociative response can develop significant brain changes from regularly flooding their brains with opioids. In brain SPECT scans, the brain changes of people who frequently dissociate resemble the brain changes seen in people with drug addiction. It is presumed that these children’s brain changes are due to frequent periods of high levels of opioid-like endorphins released during dissociation responses.

How does this relate to Parkinson’s disease? Addiction is now recognized as occurring in response to excessive levels of dopamine-enhancing drugs, including the dopamine-enhancing opioids. Part of the addiction process is the subsequent *decline in dopamine functionality*, presumably in response to endorphin excess.

It may be that the brain changes seen in drug abusers, in people who consciously activate the dissociative response, and in PDers have similarities: they may all have been set in motion by the brain’s healthy and correct adjustments to external or internal chemical alterations in dopamine functionality for certain brain and nervous system activities.
dissociation response. These two forms of dopamine inhibition are not directly related, except that they demonstrate how inhibition of dopamine release for movement and seeking behaviors can be a naturally occurring event: dopamine release for movement and seeking behaviors does not usually occur during times of injury or times of dire danger.

Dopamine is the primary neurotransmitter of seeking behaviors: curiosity, interest in food, playfulness, uninhibited self-expression. Such behaviors need to be inhibited during times of danger and during times of initial healing from severe injury or trauma.

Dopamine during dissociation

During a dissociative response, the normal, healthy style of multi-purpose, parasympathetic (joyful and/or contented) dopamine release is inhibited. Instead, the sympathetic (danger) system releases location-specific opioid-like endorphins. Endorphins cause localized dopamine release. The endorphins activate dopamine release primarily in very specific, pain-numbing, parts of the body (usually at pre- and post-synaptic nerve junctions). However, at very high levels of endorphin release, the amount of dopamine released is also very high: dopamine can overflow the pain management areas. Dopamine can get into the brain, influencing mood and certain aspects of consciousness. An injured animal can have an endorphin release so large that it can slip into a condition similar to the coma induced by opiate overdose.

This high level of dopamine release is not without risk: as footnoted earlier, brain scans of abused children who use dissociation to deal with frequent assault show that the frequent events of high levels of dopamine have set in motion brain changes similar to the changes seen in the brains of addicts who use dopamine-enhancing drugs. However, as a once-in-a-great-while method from escaping from imminent death, the mechanism has definite advantages.

In times of trauma, a person who has dissociated can have the benefit of localized dopamine-based pain relief and even the ability to appear as if dead, without risking the dangers of 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 functions: pain relief and alteration of consciousness. During trauma, dopamine release and receptor activity for other dopamine-based functions such as movement and seeking are inhibited.¹

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.

The source of this dopamine is local to the nerves. The substantia nigra, the source for parasympathetic dopamine storage, is not involved in making and storing the dopamine that is local to the endorphin receptors.

This dissociation process of dopamine release is natural and occurs when endorphins flood the system. “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 you call it, the sense of well being is actually caused by the flood of dopamine that results from endorphin activity.

Some clever PDers have learned that, by overexerting themselves, they can obtain a moderate level of movement function for a while: but the easy movement only lasts until the endorphin rush wears off. This method of staging a false metabolic emergency in order to stimulate endorphins in order to stimulate a short burst of dopamine is not actually a healthy mechanism. Over the long run, it may reinforce the habit of relying on an emergency
I am emphasizing this point because PDers are often fascinated or obsessed by the idea of their “insufficient” dopamine. I want to drive home the point that dopamine-release inhibition is a normal body function during times of severe injury or times of trauma.

Of course, in the case of PDers, they have forgotten how to turn the dopamine-releasing thoughts back on after consciously inhibiting them. But PDers, despite their measurable decrease in dopamine-producing cells in the substantia nigra, do actually have sufficient levels of dopamine to move perfectly normally, if they can only remember how to release dopamine. The chapters about the placebo effect in PDers will make this embarrassingly clear.

The decrease in substantia nigra dopamine levels is a decrease based on the use-it-or-lose-it principle: PDers, being injured and not yet having arrived at a safe place for recovery, do not use parasympathetic (safe and joyful) system dopamine.1

For PDers, it is not just a matter of remembering how to release dopamine: as long as the mind tells the body that it is in the “running away from danger” mode, it is correct for the body to inhibit dopamine release.

In the case of dopamine-inhibition from severe injury, dopamine release should not occur until after the injury has done sufficient healing. In the case of dopamine-inhibition from a dissociation response, dopamine release should not resume until the traumatized person finds himself in a safe place and is able to address the damage that was done.

There is a time and a place for healthy inhibition of dopamine.

1 In a healthy person, the consciousness determines how dopamine will be used. 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 receptors are named, cleverly, D1, D2, D3, D4, etc. According to teachings of holistic medicine, the brain waves themselves determine which receptors are active at any given moment when dopamine is released. For example, if a person thinks about getting up off the sofa, the motor-function receptors for this action should become open to dopamine hook-ups.

Oppositely, pathologies of receptor activity allow dopamine to hook up to the wrong receptors at the wrong times. Sleep-walking is an example of what happens when motor function dopamine receptors are receptive during sleep – a time when motor receptors are supposed to be off and dopamine release is supposed to be minimal.

Although the idea that brain waves can determine chemical function may seem like science fiction and many neurologists are blissfully unaware of the concept, scientific research over the last decade increasingly supports this idea. Please read Dr. Candace Pert’s Molecules of Emotion to learn more about the way that neurotransmitters and other chemicals throughout the body change shape and change function in response to thoughts.
In PDers, both of these dopamine-inhibiting conditions – injury and trauma-based dissociation – are often present. The unhealed injury is a commonality in all the PDers we have worked with. The other factor, the inability to acknowledge and heal the injury, is most likely related to a dissociation response performed at the time of injury – evidence of which we have seen in a majority of the PDers we’ve worked with.

**The dissociation response in PDers**

Based on statements made by many PDers when their injury starts to heal, we now strongly suspect that a consciously-induced dissociation response had been initiated by many PDers at the time of their foot injuries – if not before. A typical recovery statement is something along the lines of “Oh. I just remembered when I decided to not be able to feel pain.”

The problem with this dissociation method of “dealing” with the foot injury or a lifetime of trauma is that, in people with Parkinson’s, they have apparently forgotten to acknowledge the injury at a later, safer time and turn off the dissociation response. Until that response is turned off, the injury cannot be addressed – it cannot heal.

In animals, the dissociation response seems to be a body-wide event. The extent of the response depends on the severity of the danger.

Humans are different. In humans, the dissociation response can be used selectively. A person of high intellect with a leaning towards word-based memory function (as opposed to a person who tends towards visual-based memory function) can mentally compartmentalize a threat, an injury or a specific body-part. He can then dissociate from that particular threat, injury, or body part as he chooses. In this way, some types of people can choose to dissociate from specific threats (a sympathetic nervous system stance) while remaining open-hearted and emotionally present (the parasympathetic nervous system stance) for other aspects of life.\(^1\)

However, the brain is extremely plastic (changeable) and learns by repetition. Each time a person uses a dissociation response, the more likely he is to use that type of response the next time that some problem arises. After decades of increasingly using a mild, highly compartmentalized dissociation response to deal with any unpleasantness, a person who sincerely wants to be engaged with the world and emotionally present may nevertheless find himself increasingly detached and closed off.

Another problem with using dissociation to deal with fears and dangers is that the fears and dangers can multiply in the brain: because of the way the brain builds associations and links,  

---

1 People who have primarily visual-based memory function may have a harder time compartmentalizing, mentally organizing, and making sense of trauma events. People who have primarily word-based memory function are able to make up stories, change 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 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.

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.”
remembered items that were ever associated with risk or danger will make links to other items that may have been only peripherally associated with the risk. 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.

Because of this fear-linking process in the brain, a person who accumulates fears via dissociation instead of confronting the fears and dealing with them may find, after decades, that nearly anything and everything suggests a potential risk or else has become associated with some type of negative, sympathetic system-based emotion: the person with the aversion to corn dogs may find, ten years later, that he scorns all ocean-side amusement parks. Twenty years later, as the negative attitude continues to make expansive links in his brain, he may feel a virtuous arrogance towards “the type of person who could possibly enjoy going to ‘those kinds’ of places.”

Finally, it seems that people who use a dissociation response and never finish mentally processing the trauma from which they have dissociated might force the seesaw of their emotions increasingly, over decades, towards a wariness mindset and away from a contentment mindset. The greater the degree to which a person remains wary, the greater degree to which he uses adrenaline and not dopamine as the movement and mental neurotransmitter of choice.

Working with PDers

When I first started working with PDers, I was surprised at how many of them had a stoic-like 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 more emotional similarities such as inability to visualize anything with a positive connotation, daydream in a positive manner, or imagine their own bodies, especially the damaged areas, being filled with light. Also, the strange remarks made by 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 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 consciously induced dissociation response to physical and emotional pain.

In some PDers, the response has been limited to the foot injury. These people recover quickly from Parkinson’s disease.

In those PDers who struggle with certain aspects of recovery, the dissociation and inability to release dopamine for movement and seeking behaviors seem to have become, over the years, applicable to many or all situations. In this case, normal neurotransmitter release may occur only intermittently, if at all, even if the foot injury is healed and body-wide muscle tone recovers and olfactory and gustatory sensitivity returns. In these PDers, the dissociation must be intentionally turned off before full recovery is possible.

In addition to dissociating from injury – a condition in which all neurotransmitter activity is diminished – many PDers have lived life in a manner that suggests they have been perpetually in sympathetic mode. When these PDers wanted to think about things or get something done, they tended to think and move with adrenaline, not dopamine. Though they were adrenaline-dominant, their adrenaline levels were nearly always 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 alert.”

They almost never engaged the parasympathetic system; the system in which dopamine is dominant and adrenaline is subordinate.

Some PDers have a few behavioral arenas in which they dip into the parasympathetic (content, dopamine-using) mode. For example, some people with advanced Parkinson’s can move normally while painting, playing a musical instrument, or doing the crossword puzzle. But these oases from efficient, quick and highly alert, adrenaline-driven movement and thought usually have carefully defined boundaries. When he is not doing the “safe” activity, the PDer anticipates, plans, and performs using modest levels of adrenaline – not dopamine.

Again, and I will repeat this over and over and over in this book, not everyone with Parkinson’s disease has a severe ongoing mental/emotional blockage towards acknowledging his own body, a mental blockage that was probably created using the dissociation response. However, a majority of our PD patients have presented evidence of a consciously induced dissociation response – one that continues in all or some environmental situations even after the foot injury is gone. In these cases, the PDer’s physical movements show that he can once again trigger dopamine release in situations that he deems favorable. However, his scope of what he deems favorable may have shrunk to nearly nothing, because of decades spent in the sympathetic-mode mindset. Until the dissociation mindset is turned off, nearly everything, including the body itself, may be viewed through a wariness response: dopamine release, even if physically possible, will not occur during those times that the body is stuck in the sympathetic mode.

Therefore, the PDer who hopes to recover needs to recognize sympathetic system mindsets and learn how to turn them off.

THE CASE STUDIES

When I started the Little Project I did not suspect the degree to which mental attitude played a role in causing Parkinson’s. Nearly a decade passed before I understood that, in addition to recovering from a foot injury, consciously recovering from a mental attitude of “pride in stoicism” may, for some patients, also be necessary for complete recovery from Parkinson’s disease.

Even so, I noticed from the very beginning that there was a strange commonality in the injury histories of my first patients. To demonstrate this commonality, this chapter shares a few short PDer case histories from the first years of the project.

The following case studies were written up long before I recognized the hints that a dissociation disorder might be involved. At the time I wrote these up, I was hoping to demonstrate my observation that my PD patients all had unhealed injuries and to show my meaning of the word “unhealed.” At the time I wrote these up, I knew nothing of the physiological mechanism by which a person can compartmentalize and dissociate from an injured body part – or from his entire body – and by so doing inhibit the release of midbrain dopamine.
**Katya**

My first patient with PD symptoms had a slow shuffling walk and profound rigidity, but what worried her was the numbness, the woodenness in her legs. Katya was 56 years old. She came to see me for gallstone troubles. After terrifying, brutal experiences with obstetric doctors in her native Russia, 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 treated her, successfully, for gallstones.

I hadn’t started this study yet. Because she didn’t have a tremor, I didn’t suspect she had Parkinson’s. Neither did she. 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 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 limited knowledge would have pointed me in this direction only.

During her treatment, I was exploring the bones of the 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 handle an area without the patient noticing it very much. 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 the feet until my hand came to a complete stop at the strange deathlike joints 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’s foot was smashed in the car door. The injury was not lurking in the subconscious; Lynne had never forgotten about the injury. She had assumed that the injury had healed normally. During treatment, it became evident that the foot had not healed.

Lynne recalled the event perfectly well: she 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 completed 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.

There was an electrical storm going on 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 frightening day in her young life. So many strange 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, 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. 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 control her response to pain to the extent that she had no physical reaction to a severely 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. In defiance to his walker, he often insisted on shuffling along without it around the house, 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 whuffled. “Never been hurt.”


“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 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 now suspect 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 one of my first patients. 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 broke loose. A few days after that, the second toe broke loose, and a few days later, the others.

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.

For many reasons, I suspect that his foot injury occurred long before high school. One reason makes so little sense that I am hesitant to include it, but here goes. Moments before his foot began to respond to the FSR, as I was sitting with my eyes closed, holding his feet, I suddenly saw, behind my closed eyes, the face of an infant staring at me intently, as if assessing
my intentions. (I had none.) I was suddenly filled with a sense of severe revulsion, and an image appeared before my horrified closed eyes of someone sexually abusing an infant while gripping the child by the feet. As I shuddered at this unexpected, foul image, Norm’s foot relaxed. When I asked Norm, several months later, if those images meant anything to him, he said that they did not. I had not been looking for anything mysterious or psychic. The mental image intruded on me as I was minding my own business. I have not created any scenario to explain the significance, if any, of the strange mental pictures that floated across the screen of my mind, nor do I intend to.

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 tremoring 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. 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 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 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 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!”

TJ

TJ was one of the most stoic people I have ever met. I had been holding TJ’s foot once a week for an hour, for about three months, when she started having excruciating pain in her tibia, about half-way down the bone (the bone on the front part of the lower leg). The previously numb foot had been starting to have terrible pins and needles in it and the color was changing from dark grey to pink. She started to have feeling in her foot and leg. When 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 and hurrying across a busy thoroughfare, dodging traffic. She suddenly realized 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.

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. The first airplane ride of her life, at age four, took her, unaccompanied, to California to live for many years with her grandmother, whom she had never met. On the plane ride, TJ 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 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. As the foot began to experience terrible pain, eliciting screams of fear and agony from TJ, she also began to experience her tibial fracture.

Horses can be strong, but a PDer is tougher

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 ornary 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.

Even so, the foot had been clumsily reattached in a pair of surgeries that would be considered primitive by today’s standards.

In hopes of giving the foot some flexibility, surgeons had hurriedly 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.

Through the years, the rigidity and displacements in the right foot had caused Chuck’s walk to become distorted. His right leg was an inch and a half shorter than the left; he used an elevated shoe. With the limping and the stiffness, 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 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 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.

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.

**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 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…”

**Gus**

Gus came to the free PD clinic (which ran from 1998 to 2002) two years after he was diagnosed. He was treated by a student, Doreen.

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 called his wife to come get him. He sat for a while in the outer office before he was able to get in the car to go home. He was visibly 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. The day during the war that 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 with anyone. He never thought about it. He had not remembered it since that day when it happened. He’d never discussed it with his wife. He did not want to remember it. He could not bear to remember it. As the memory of it began to intrude in his mind, he thought he would die from the pain and fear.

Over the next two weeks, he discussed his options with Doreen. He never shared details of the events that occurred on that hellish day. He wanted to know what he could do to stop the nightmarish thoughts that were swarming over him and surrounding him.

His fear was not that his Parkinson's disease would worsen, his fear was that he would keep remembering more and more details. He came to a brave decision: “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 talked it through. They decided that he had a better chance at reclosing the door to his war-time memories if he did not seek further treatment for his Parkinson's disease.

We were all happy with his informed decision. Gus did come eventually to a sort of peace around the sealed-off trauma that had been touched on.

**Some Generalities about Other Case Histories**

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 are not injuries from which a person limped away with a cheery determination and a defiant cry of, “Don’t worry, I’m not hurt!”

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 “Don’t worry, it’s nothing.” The difference between him and the usual “don’t worry I’m not hurt” person is that the PDer may have truly believed it. The recipient of a dissociated injury may not even be able to cognize that he was injured. And if he is aware of the injury, he may not be
able to feel the injury: he may perceive the injury as if it happened to someone else – a numb version of himself.

All of the PDers that we have seen have unhealed injuries in their feet. Very often, these injuries have been blocked out of the mind for whatever reason and have never healed. In many cases, the injury never even followed the normal pattern of injury with swelling, discoloration, bleeding or pain. The injury froze in place and remained, ever since, 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 might move 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.

**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 one parent but either hostility towards weakness or praise for “sucking up the pain” from one or both parents.

“Don’t show your weakness” is a common theme in the childhood of 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 really painful (a beating) to cry about.”

Others had a kind and loving guardian, like Katya. 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 have emotions.

**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. 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, a PDer may have no memory of any reason to dissociate from an injury, plus he may have no overt indication of injury. Nevertheless, in our experience, he will have a foot problem that hasn’t healed.

Lila

Sometimes, only rarely, in my experience, the injury is almost imperceptible except to the most skilled touch. The cause is unknown, as is the 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 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 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, 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.

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 evaporating as I kept holding the foot.

Very strange. It was quite subtle. 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 warmth, “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. I 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.

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,

¹ I am often asked if I have heard about the latest new 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 the supportive holding method to the needs of the patient.
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.

**Nails and bicycle spokes**

PDers sometimes think that their unremembered foot injury must have been something shocking, dramatic. This is 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 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.

Another common injury in Parkinson's disease case histories is the foot-in-the-bicycle-spoke 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 bicycle 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.

**A FEW QUESTIONS THAT ARISE**

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

Not everyone can remember the injury that set the Parkinson's disease in motion anywhere from fifteen to sixty years after the injury occurred. Many patients rack their brains trying to remember a significant injury. They ask old friends and family members, but come up blank. Some admit 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 feel bad about not being able to remember. Others are concerned that, if they can’t remember, the Parkinson's disease won’t go away.

Actually, remembering the injury doesn’t seem to matter. If I had to guess at the numbers, I would say that about three fourths of my patients have remembered their injury with chilling detail. Some remembered the injury during treatment. Others have always remembered it. About one fourth never do 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 vibrational pattern, the electrical currents will be able to run correctly again. Even patients who could not recall any instance of unusual injury are walking around today completely relieved of any symptoms of Parkinson's disease.

*Is the injury always visible by X-ray?*

A frequently asked question is, “Will the foot injury show up in an 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.

Other feet have glaring displacements. The most common displacements that are visible
on an X-ray are in the bones around the intermediate (2\textsuperscript{nd}) cuneiform bone: the navicular, the
proximal ends of the 2\textsuperscript{nd} and 1\textsuperscript{st} metatarsal, and the 1\textsuperscript{st} and 3\textsuperscript{rd} cuneiform. Other displacements are
not uncommon, or may exist in combination with the above “most popular” sites for
displacement.

It can be important, if one wants radiological proof of displacement, to look at 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 bones are clearly unable to articulate
correctly. And in rare cases the bones may even articulate correctly, but the foot still feels as if
there is a dead place in the center of the foot.

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 years with the bone in that condition.

\textit{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 \textit{to choose} to perform this type of
dissociation, a mental action that allows the PDer to “not be hurt.” It then requires habit or pride
in this type of mental control to continue using the technique of dissociation as a way to avoid
smaller, less threatening pains.

The PDer may imagine that when he says, “I shall not be hurt!” his body \textit{is} not hurt. By
dissociating from a painful event, the PDer can experience the painful experience as if it is
happening to someone else, or as if he has no feelings by which to perceive the pain.

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 \textit{not having had} an injury, and it is not
the same as mentally \textit{healing} the injury.

The mental compartmentalization and denial of injury that appear to be common
components in Parkinson's disease prevent eventual healing of the \textit{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 \textit{not} a healthy, long-term solution to injury.

In the days when people only lived thirty or forty years, denial and/or dissociation 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 dissociation to remove one’s ability to feel a very real injury can ultimately lead to an illness which is more devastating than the original injury might have been.\(^1\)

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 injuries and, in some cases, dissociating from his body.

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 dissociation, he learns to cut himself off from his ability to feel his own physical and emotional pain. If he practices it enough, he can cleverly cut himself off from his ability to feel his own physical and emotional existence.

If mental/emotional dissociation is applied to an injury, the injury cannot heal until the dissociation is turned off.

This brings us to the development of Parkinson’s disease. Due to the unique configuration of the foot, injury to almost any part of the foot ends up displacing, at least temporarily, the center bone of the foot. Due to the enormous electrical significance of the currents that must pass through this exact part of the foot, an unhealed foot injury can, over time, set in motion a dangerous collection of electrical aberrations.

An injury to the thigh, the torso, or the upper arm may be problematic, but if these areas are injured, the local currents are able to flow around and past these glitches. An injury in these areas will not set in motion the electrical aberrations that cause Parkinson’s disease. An unhealed

\(^1\) 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. Many of my PD patients, and a 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 first appeared, years before the PD was diagnosed.

Irregular electrical patterns of rebellious 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, with no cause. (Absurd! Spontaneous generation?)

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 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.

It appears that, not only is Rebellious Qi in the Stomach channel a problem now that we are living longer, but possibly, Rebellious Qi in the Stomach channel is a cancer-causing killer in its early stages, decades before it is diagnosable as PD. People who have the faster developing forms of PD, the forms that appear before age 50, probably used to die of the other cellular aberrations, long before the PD could manifest. No wonder the word “Rebellious” was used for this treacherous type of Qi.
center-of-the-foot injury, however, might spell long-term trouble. The reasons for this will be detailed in chapter five.

**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 really feel his own foot – and hasn’t really felt it since he decided that he could cleverly deal with his injury by deciding that it never happened. In and of itself, a foot injury does not cause Parkinson’s disease. The inability to address 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 will 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 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 help them along 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.

**CONCLUSION**

The problem in PD is always a foot injury, but in some cases a dissociation response to the injury is also part of the problem. These few case studies show why, even in the early years of our project, we began to suspect that an emotional component might be as significant as the foot problem.

The unhealed injury that triggers the Parkinson’s may involve a broken bone, a twisted bit of fascia, or a bit of unhealed torn tendon. The original fear that inspired the PDer to induce a dissociation response may have been anything from dread of death to fear of humiliation in front of friends.

The physical injury and/or the reason for dissociation may or may not have been long forgotten. But we can now guess that whatever the injury situation was, the response at the time was most likely something along the lines of “It’s OK. It doesn’t hurt.”
“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 17

CHAPTER THREE

CHANNEL THEORY

The next few chapters involve channel theory – a crucial part of Asian medical theory and the key to understanding the anatomical changes of 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. Chapters four and five will explain the channel changes that can occur in response to an ongoing foot injury and how those changes lead to highly specific and predictable anatomical pathologies. The particular set of anatomical pathologies happens to be identical to the changes that occur in Parkinson’s disease.

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 Qi (pronounced chee). Channels are the directions and locations in which Qi is most likely to flow, if it flows. The channels have no tangible existence if no energy is running through them. There is no physical, conduit-like structure through which the channels flow.

A metaphor might be helpful for understanding what channels are: channels in humans 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 Qi flows, it tends to flow through the body’s tissues, especially the fascia tissues, in specific, detectable routes. These routes are called channels.

---

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 will be defined in depth in an appendix.

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.

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...
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 the electrical aberrations that develop in Parkinson’s disease.

However, to appease the acupuncturists who may be unhappy if I say that 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 theory explains that there are five types of electron vibrational patterns. Chinese medical theory acupuncture, no studies have been able to prove that channels need the nerves in order to exist. The Asian theory would say that 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 lines 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, Heckawa 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. Currently, if you want to detect the dynamic flow of Qi, tools are useless; you have to feel it in your hand.
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 electricity which is electron-vibration-pattern specific, as opposed to the thought- and emotion-neutral electrical currents that we generate with batteries or power plants.

Your car battery is only concerned with getting a charge to move from here to there. The type of subtle electron vibratory movement is of no consequence. In bodies, the type of electricity used by each channel helps make up the characteristics of that channel.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). Together, the physical structures of the body and the electromagnetic fields with their equal and simultaneous channels give both stability and dynamism to 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 emotion 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 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.

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.
Ironically, in order to “maintain,” many a PDer has denied himself full awareness of his “self.” Thus, he renders himself less able to electrically resist the pathological changes set in motion by his ignored injuries.

In summary, the flow of energy in the body, while obeying the laws of electricity, is considered to be more than just the flow of electrons. This electricity-like energy is referred to as channel Qi.

So, to satisfy any sticklers who may be reading this book, when I refer to the currents as 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. Currents are influenced by the chemistry and structure of the body. The 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 merely electricity. We say that “channel Qi is the energy that flows in the channels.”

On the other hand, if you are not a stickler, just think of channel Qi as “electric currents.”

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 electrical charges (on molecules) that surround each cell, thus determining DNA expression and other cellular functions.

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 This definition is oversimplified. But for now, in order to understand what happens in Parkinson’s disease, understanding the nature of Qi is not crucial.

2 “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 regulate the chemical activities in the cells, including activities in the cells’ DNA. These electrical charges are manifestations of the very smallest branchings of the channels.

3 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
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, working side by side with the electrical currents that run over and through the entire body, join together to provide respectively some structural stability and the ability to respond and change in an ever-changing internal and external environment.

**Nomenclature of the channel system**

The channel system’s largest channels consist of twelve primary channels and eight “extraordinary” channels. The primary channels are so called because the named acupuncture points (which happen to be areas of decreased electrical resistance) are located along these lines.1 The extraordinary channels are distinct currents of energy, but their major points of increased electrical resistance are at intersections with the primary channels, intersection points that are already named via the primary channel. Therefore, the extraordinary channels do not have named points, and are therefore “extraordinary.”

Wouldn’t it be nice if it were really that easy? It’s not. Refuting the rule, two of the eight extraordinary channels do have their own named points. These two appear to be in the “extraordinary” class because they are not bilaterally symmetrical, but instead run up to the head from the midline of the front of the torso and the midline of the back of the torso. No one really knows for certain why and when some of the large channels were named primary and some named extraordinary. Theories abound. Regardless, this is the historic nomenclature, and we still use it. To keep it easy, I will, in this chapter, refer to both sets as the “main” or “large” channels.

The name of each primary channel reflects the internal organ that is primarily regulated by that channel. For example, one of the smaller branches that comes off of the Stomach channel

---

1 As a point of interest, medically recognized “trigger points,” areas on the body that become sensitive when something is going wrong, are areas of increased electrical resistance.
in the vicinity of the skin over the stomach flows deep into body at that point and regulates the development and function of the stomach.

One more bit of information: the acupoints, the locations on the body that are used most often in acupuncture, are named with a channel 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, but are called by the full name of channel plus number: for example, “Stomach one.”

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: a channel on the left side of the body, 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.

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 diggs a river bed. The river bed then guides the flow of subsequent water – until a flood or geological upheaval occurs.

In Asian theory, this famous principle of energy and chemistry’s mutual relationship in living systems is expressed as “The Qi is the leader of the Blood (chemistry) and the Blood is the Mother of the Qi.”
Hopefully, this is enough general information to be getting on with. Next comes some more specific information about channels, including their pathways. You do not need to memorize the paths of the channels, but simply peruse the information and enjoy it.

**Starting with the Stomach channel**

Before you learn what happens when the channels go awry in a person with Parkinson’s disease, you should have some mild idea of where a few pertinent channels go when they flow correctly. The diagrams on the next few pages show the *correct* paths of the channels that are involved in the pathologies of Parkinson’s disease.
Fig. 3.1 Correct flow of 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.

From the hip, the Stomach channel flows down the anterolateral (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.
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. 3.3  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. 3.4  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. 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. 3.5 The upper portions of the Gallbladder channel, the Stomach channel, and the Large Intestine channel showing the correct flow directions
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. 3.7 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.
This short chapter is just an introduction to channel theory. 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” channel flows back into the first.
“Go through: no pain. No go through: pain.”
- A basic principle of Chinese medicine

CHAPTER FOUR

BLOCKED QI: TROUBLE

Basic channel flow theory

Channels, as described in the previous chapter, are pathways for the flow of electricity-like instructions that guide all body processes. When these 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, things go wrong. That’s illness or pain.

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 jammed due to the presence of some non-conductive tissue, the flow is diverted 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.¹

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 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,

¹ Acupuncture needles can be used to reintroduce electrical flow through scar tissue. When correctly needled, scar tissue reverts back to healthy tissue and the numbness goes away.
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. 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.

If the nearby channel is running the *opposite* direction of the diverted channel, the diverted channel’s current, like that of a streamlet joining a river, plunges into the neighboring channel – and changes its direction into that of the neighbor current. (Actually, the neighbor current is 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 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 rather than running laterally into a nearby channel. This submerged Qi is often able to resurface near the skin farther down the 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, 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 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 blockage 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.\(^1\)

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 system is overthrown. Chaos and death ensue. No good can come of rebellion.

---

\(^1\) 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) into the new pathway, while some portion of the deranged current closer to the blockage site continues to run quickly back and forth.
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 air 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” suggested 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 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 discovered the role of backwards 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.

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.

The next chapter starts with the Qi blockage found in all PDers’ feet. From there, it proposes a set of body-wide repercussions of disrupted and backwards Qi flow. These disruptions and consequences are based on a few channel blockage principles of Asian medicine and on basic, high school level physics (electricity and magnetism).
These disruptions and this backwards flow not only comply with the “rules” of Asian medicine and western physics. They also cause physical changes that correspond to the exact symptoms that are seen in Parkinson’s disease.
“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 FIVE

THE DEVELOPMENT OF PARKINSON’S:
CHANGES IN CHANNEL FLOW

Changes in the foot portion of the Stomach channel

By 1999, all of the of PDers that I had come across had palpable evidence of an unhealed
foot injury in the vicinity of the intermediate cuneiform bone, right at acupoint ST-42. (See Fig.
2.7.) Eight years later, as I write this up, I can say that all of the hundreds of PDers I’ve worked
with have the same indications of unhealed foot injury. Most often, these injuries occurred
during childhood. At some point in time, usually around ten to fifteen years after sustaining a
foot injury, subtle complications began to arise as a result of the injury failing to heal.

Many of the foot injuries we have seen did not originate with impact occurring directly at
ST-42, even though the injury quickly congealed in the vicinity of the intermediate cuneiform
bone. For example, the initial point of impact of some of the injuries was the toes, and for other
injuries the point of impact was the ankle.

However, as the force of an injury moves through the foot, the design of the foot bones
usually directs the force towards the center of the foot, in the vicinity of ST-42. The design of the
foot provides for a maximum of movement at the intermediate cuneiform bone. This wedge-
shaped bone is able to move up and down, forward and back. This movement enables the
intermediate cuneiform bone to act as a shock absorber during the normal impacts that occur to
the foot during walking, running, and jumping. The center of the foot, at and around the
intermediate cuneiform bone, usually bears the brunt of an injurious impact to the toes, foot, or
ankle.

Again, when a foot injury occurs almost anywhere on the foot or low ankle, much of the
force may be relayed to the intermediate cuneiform bone, the shock absorber of the foot. The
cuneiform bone is temporarily displaced. If the displacement is so great that this bone cannot
resume its correct position, and if the mental detachment from the injury is so great that the body
does not institute healing measures, the cuneiform bone, and very often the surrounding bones,
may remain displaced for the rest of a person’s life. In a healthy person, some degree of swelling
and internal bleeding, together with the deep relaxation of sleep, usually provides enough wiggle
room for displaced bones to return to their proper position. People with Parkinson’s, due to their
powerful mind control and dependence on adrenaline, do not allow these healing processes to
occur. Some PDers never relax completely, not even during sleep.

An injury always creates a short-term electrical disturbance in the area of the injury. This
electrical disarray helps to signal the rest of the body that an injury has occurred, that healing is
needed. As healing occurs, the electrical currents begin to flow normally again. In a pre-
Parkinson’s person, the injury remains in place in the foot and the impact of the blow continues
to be retained with various micromuscle tensions and mental determination. (A “pre-Parkinson’s
person” is one who is not yet showing symptoms of PD, but who has a PDer’s underlying
energetic disarray – like Tim, the twelve-year old in chapter one.) The electrical disarray caused by the injury never clears up; the injury never heals.

In Parkinson’s disease, the foot problem, still unhealed decades later because of a mind-body disassociation either specific to the point of injury or, in some cases, body-wide, sets in motion a more permanent problem: When the snowballing electrical disarray in the foot becomes too large or too disrupted, 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. Current in the Stomach channel begins to decrease in the areas of the foot distal to the point of injury; the middle toes and the medial side of the big toe slowly, over decades, become somewhat numb.¹

Fig. 5.1
A common presentation of pathological Qi flow in the Parkinson’s disease foot

Stomach channel Qi, impeded at ST-42, jumps over to Ki-2 and flows up the Kidney channel. Then, because the Kidney channel is already filled with Qi at Ki-2, the Kidney channel Qi that flows under the foot at Ki-1 cannot flow into the channel at Ki-2. Instead, it grounds out to the floor. Note the profound deficiency of Qi in the foot distal to ST-42, especially in the three medial toes and along the medial arch of the foot.

Compare this drawing with Fig. 3.7 (page 55): note the absence of Qi in the middle toes and the medial side of the big toe in the Parkinson’s foot.

We’ve detected many variations in PDers’ foot portion of the Stomach channel: once a system goes wrong, it may go wrong in a near-infinitude of individualized foot patterns.

¹ Distal means “moving in a direction away from the head.” Proximal means “going towards the head.”
Due to the electrical resistance, one event that may occur is that current in the foot portion of the Stomach channel begins to flow over into the foot portion of the Kidney channel. The Stomach channel Qi short-circuits into the Kidney channel at acupoint KI-2 or KI-3. This creates an excess of current in the Kidney channel in the area around the short-circuit. This excess can contribute to the enlargement of blood vessels in the KI-3 (medial ankle) area. Other local pathologies can include varicosities and skin discoloration.

When this happens, Qi/energy in the foot-sole portion of the Kidney channel can no longer flow unimpeded from the bottom of the foot to the medial ankle portion of the now excessive Kidney channel. Instead, when the Stomach channel Qi is short-circuiting into the Kidney channels during times of high Stomach channel use, the normal flow of the Kidney channel from KI-1 to KI-2 encounters resistance.

The Kidney channel then is prone to ground out: current on the underside of the foot at KI-1 may make an electrical connection with the floor. This may contribute to the feeling that the foot-sticking and shuffling of Parkinson’s does not feel like mere absence of foot lift; many PDers say that it feels as if there is an active force that is pulling the foot to the floor, making it stick.

The Stomach channel-Kidney channel short circuit may or may not occur in combination with the reversal of the direction of Qi flow in the Stomach channel. We have seen the reversed Stomach channel problem in all correctly diagnosed PDers. We have not seen the Stomach-Kidney channel short circuit in all PDers. Some PDers have other, equally incorrect short-circuits and disruptions on the foot in the areas distal to ST-42: we have seen PDers with Stomach channel Qi flowing into the foot portion of the Urinary Bladder channel or the Gall bladder channel, and sometimes into the Liver channel. These variations contribute to the variations on toe cramping and toe distortions experienced by many PDers.

**Stomach channel reversal**

When resistance at the terminus of the Stomach channel increases, over time, 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 rebounds upwards, back up the Stomach channel towards the face.

Actually, the disrupted current runs in a sort of rapid back and forth current pattern, flowing down towards the foot, hitting the electrical mess at ST-42, and then flowing back up towards the head. This creates an “alternating current” type of situation. This alternating current prevents the normal flow from the Large Intestine channel into the Stomach channel, and triggers other electrical problems that will be described later. Backwards-flowing current and current that alternates rapidly back and forth rather than flowing in the correct direction is called Rebellious (or Retrograde) Channel Qi in Asian medicine.

Eventually, the overall direction of the current in the Stomach channel may become predominantly backwards even while the back and forth pattern is still occurring to a small extent. The predominant palpable sensation generated by the Rebellious Stomach channel current is that it is running up the leg (towards the face) rather than down (towards the foot).

In PDers, after decades of disarray, Stomach channel Qi may cease to palpably flow in the foot or lower part of the leg. Even when the lower leg portion of the channel ceases to have any detectable Qi flow, Stomach channel Qi can still be detected running rebelliously in the upper leg, torso, and neck portions of the channel.

These currents can be felt easily with a trained hand.
Electrical change in the head portion of the Stomach channel

Although at first, some Stomach channel Qi might short circuit from ST-42 into the Kidney channel at acupoints KI-2 or KI-3 on the ankle, eventually the blockage of Qi and tissues at ST-42 is so great that little or no Qi passes into the region of ST-42. Most of the Qi in the Stomach channel then flows rebelliously. At first, when retrograde Qi begins to flow, the Qi moves erratically back and forth in the Stomach channel between ST-42 and the knee. In the lower leg, some of the Stomach channel’s current may (or may not) flow laterally into the nearby Gallbladder channel at or around acupoint ST-40.

Eventually, over years, the Rebellious Stomach channel Qi pattern affects the entire 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 gets to the point on the jaw known as ST-6, the current is redirected – it can no longer flow backwards along the Stomach channel. A built-in one-way-flow mechanism appears to exist at ST-6, so that ST-6 serves as a Stomach channel safety valve. If backwards-flowing Qi in the Stomach channel gets as far up the body as the jaw, the current of Qi is redirected up towards the forehead towards ST-8 in a special path that is only filled with current when Qi is flowing backwards. The acupoint ST-8 on the forehead appears to act as a sort of reservoir or electrical capacitor for excess Qi; electrical charge can build up at this point.

Short circuit into the Gallbladder channel

Over time, this build up of electrical charge at the overflow point on the forehead causes this backwards-Qi accumulation at ST-8 to “short circuit” into the nearby forehead portion of the Gallbladder channel.

If the Stomach channel short circuits frequently enough into the Gallbladder channel, it can establish a new, semi-permanent electrical configuration. Semi-permanent means, in this case, permanent until the electrical flow in the Stomach channel is restored to its correct pattern.

Again, when the Qi is impeded, resistance and momentum cause it to rebound up the Stomach channel, in a retrograde direction, creating rebellious Qi. Rebellious Stomach channel Qi flows upward (backwards) to the back of the mandible in the region of ST-6. From here, it does not continue to flow backwards up the front of the face to ST-1 but is shunted from ST-6 on the jaw to ST-8 at the upper-side aspect of the forehead.

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

ST-6 at the back of the jaw serves as an "overflow shunt” to prevent 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 midline of the back and neck, and over the head to

---

1 Although current teaching about the Stomach channel dogmatically states that the Stomach channel must flow in the sequence suggested by the acupoint-numbering system, this new teaching can be easily refuted. Feel the Qi flow of the Stomach channel with your hand on the face of any healthy person: the Qi runs down the face, and does not normally flow into that branch that terminates at ST-8. This ST-8 branch is only used when there is some problem in the Stomach channel. As for the numbering system, that is an extremely new addition to the lore – it was added in the latter part of the 20th century during one of the attempts to modernize and westernize the acupoint system. This was around the same time that the existence of channels was disavowed. Defensive statements in Chinese medical books of that period even went so far as to protest that while channel theory and the system of correspondences were old superstitions, they were no worse than the old superstitions and correspondence systems used in Europe during the dark ages. This was some years after the Chinese government had methodically deleted all spiritual references in the medical books, leaving in their wake entire sections that no longer made any sense.
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 flowing Stomach channel 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. 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 and the point of origin of the Stomach channel.

**Backwards Qi in the head portion of the Du channel is dangerous to the life of the human organism.**

As illustrated earlier, a branch of the Du channel runs through the head, from the brain stem area at the top of the neck, through the center of the brain, and emerges at the front of the face at the acupoint Yin Tang. This branch directs activity in the midbrain and brain stem – the seat of consciousness. If the Qi of the Du channel runs backwards through the head, runs backwards through the midbrain and the brain stem, loss of consciousness or even death may ensue.

If the Qi running through the head is impeded in any way, consciousness is affected. If backwards running Qi from the Stomach channel were able to flow backwards into the Du channel and thereby 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 a safety shunt at the back of the jaw, at ST-6, to prevent backwards-flowing Qi from the Stomach channel from ever flowing onto the face. If Stomach channel Qi flows backwards as far as the lower jaw, it is shunted to the side of the forehead, to a point named ST-8.

The activity of rebellious Qi at ST-6, the site of the shunt, causes the intermittent lower jaw/back molar pain reported by some PD patients.
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. This irregular pattern prevents the (c) Large Intestine channel from being able to flow over the Stomach channel and across the face. The blocked Qi from the Large Intestine channel flows up to the chin and then runs back down to the fingers. 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 the blocking of the Large Intestine channel as it tries to cross the path of the rebellious Qi shunt of the Stomach channel at ST-6.

Compare Fig. 5.2 with Fig. 3.6 (page 54). Note the fullness of electrical patterns crossing the face in the correct, healthy flow of Fig 3.6. Notice the absence of any Qi in the cheek, jaw, or mouth area in the face in the Parkinson’s pattern in Fig. 5.2, an absence that corresponds to the PDer’s decline in facial expression, lack of vigor in the lower eyelid, drooling, sinus problems, and decline in the senses of smell and taste.
Points of intersection of the Stomach and Du channels on the face

The origin of the Stomach channel should normally spring from Yin Tang, the point of the Du channel between the eyebrows, the point where the Large Intestine channel finally ends and the Stomach channel begins. (See Fig 3.2, page 51.) A branch of the Stomach channel that skirts the lips also meets the Du channel just above the lips at Du-26. A few other numbered points on the Du channel are shown merely to demonstrate the direction of flow in the Du channel (up the spine, over the top of the head, and from the forehead down to the upper lip).

Fig. 5.3
Face points of the Du channel
The Du channel runs up from the base of the spine, over the top of the head, and down the front of the face to the upper lip. A major point of the Du channel is Yin Tang, on the forehead.

Under ordinary circumstances, the Stomach channel Qi does not flow to ST-8, up by the forehead, but flows down from the forehead, and down from ST-6 on the jaw directly to ST-9 on the neck. From there, it flows down to the feet. In the case of rebellious Qi in the Stomach channel, when the channel path is reversed, the Qi is prevented from flowing backwards from ST-6 over to the front of the face. Instead, it is shunted upwards to ST-8 on the side of the head to prevent a calamitous reverse flow through the critical, consciousness-maintaining Du channel.

More about the Stomach channel/Gallbladder channel short-circuit
As noted earlier, rebellious Qi flow is shunted away from the face points of the Stomach channel and instead is directed up to the forehead at ST-8. Ordinarily, a small accumulation of Qi at ST-8 merely causes a headache, if anything. However, if the Rebellious pattern continues long enough, Qi can eventually over-accumulate at ST-8; if this happens, the Qi arcs into the Gallbladder channel at the forehead, near GB-4. This short-circuit is not something that is
supposed to happen anytime that Qi builds up somewhat at ST-8. This rare and somewhat violent short-circuit only occurs, in healthy people, when a serious injury occurs: an injury that completely blocks the flow of the Stomach channel, an injury that, in order to heal, needs some mental and physical “down time” from the usual activities of daily living.

When, after a serious injury, this short-circuit occurs, the new path from the forehead portion of the Stomach channel into the Gallbladder channel causes the Gallbladder channel to carry a greater load, a higher amperage, of Qi than it ordinarily would. Because of higher amperage in the Gallbladder channel, a partial go-to-sleep signal is initiated in the midbrain. As soon as the injury is sufficiently healed that Stomach channel Qi can again flow somewhat correctly, the Stomach no longer shunts into the Gallbladder channel, and full alertness – a non-emergency, dopamine-based alertness – is possible.

In PD, this overflow into the Gallbladder channel becomes an established, permanent pathway for flow of rebellious Stomach channel Qi. This short-circuit in PDers usually occurs about ten to twenty years after the injury event.

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

Often, the first episode of short-circuiting into the Gall Bladder channel is distinctly memorable. 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 internal spinning sensation stopped.¹

**The Effect of the Gallbladder Channel on the Du Channel**

A crucial contribution to the symptoms of Parkinson’s is the alteration in dopamine levels; dopamine levels decrease when the Gallbladder channel runs at a higher amperage than usual. To understand how excess Qi in the Gallbladder channel sedates dopamine, we must consider the normal energetics of the Gallbladder channel.

The Du (Governor) channel is the most important channel in the body. It runs from the base of the spine, up the center of the spine, over the head, and down the front of the face. As the Du channel flows up the spine and over the head, an internal branch diverges from the main channel near the top of the neck (at the brain stem, acupoint Du-16). This internal channel passes through the brain stem and midbrain. This branch, which goes through the head, directing physiological processes in the brain stem and midbrain, emerges from the head at the center of the forehead at Yin Tang, the point between the eyebrows.

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 he was a good quarter mile farther down the road with no recall of any consciousness during the gap. He felt somehow mentally altered by the experience. He had been afraid to tell anyone about his head-shift experience at the time, as were most PDers in my experience. In his case, he referred to the incident in the privacy of his own mind as “my abduction by aliens.”

PDers may be intense, but they are not without a sense of humor.
Meanwhile, 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 the upper lip. At Yin Tang, the internal current rejoins the external path of the Du channel.

*The internal branch of the Du channel: regulator of the midbrain*

The powerful current of the Du channel’s internal (through the brainstem and midbrain) branch sustains conscious activity and directs midbrain functions (the midbrain is an area in which dopamine is produced). It also drives the frontal lobe of the brain, the area involved with conscious direction of will. This midbrain channel runs in tandem with the external branch of the Du channel and is affected by any variation in the flow of the Du channel’s branches.

![Fig. 5.4](image)

**Fig. 5.4**

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

The line that travels the midline of the top of the head represents one branch of the Du channel. The dashed/solid/dashed line that goes through the middle of the head represents another branch. 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 upper branch of the Du channel at the forehead.¹

¹ 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.

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 role of the Du channel in regulating sleep

In complex organisms, such as man and many of the higher vertebrates, the restorative function of sleep is required so that the brain can take time off for other (subconscious) tasks and the physical body can rest. The brain systems slow down to allow restorative processes. This process is brought on by a decrease in the flow of the Du channel. The mighty Du channel, running up the spine from the coccyx to the front of the brain, drives the currents that power conscious and subconscious activity.

The influence of the Gallbladder channel on the mighty Du channel

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 diminish 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.
When the Gallbladder channel surges, it decreases the Qi flow in the Du channel, the channel that, at higher levels of flow, regulates alert, conscious, awake behavior. 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.

The Gallbladder channel thus serves as a switch to decrease the powerful Du channel currents, thereby allowing the decrease in brain stem and frontal lobe activity, including the decrease in dopamine release, that is needed to set sleep in motion.

**The path of the Gallbladder channel on the head**

The Gallbladder channel traverses the lateral aspect (the side) of the head, ultimately flowing in the opposite direction of the Du channel.

(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:

First, recall that the path of Qi from GB-14 through GB-20 runs parallel to the Du channel but in the opposite direction.

Next, note that the path of Qi from GB-8 to GB-12 also runs in the opposite direction of the Du channel. Relative to the internal (midbrain) branch of the Du channel, Qi flow from GB-8 through GB-12 runs parallel and in the opposite direction.)

The path of Qi from GB-4 through GB-7 (as seen in Fig. 3.6) runs perpendicular to the portion of the Du channel at the vertex. While this bit of current does not oppose the Du channel, electromagnetically speaking, neither does it support the Du channel.

**The Gallbladder channel effect on the Du channel**

The net effect: in the head area, the Gallbladder channel current flows in the opposite direction to that of the Du channel. During the day, a Du-supporting balance is maintained between the various head-located channels so that the Du channel can flow over and through the head at a steady rate.

At 11:00 p.m., when the Gallbladder channel begins to flow with a greater level of Qi, the “anti-Du” effect of the Gallbladder channel is increased, causing an inhibitory effect on the currents in the Du channel and through the midbrain branch. This reduces Qi flow through the areas of the brain that control conscious movement and awareness. Sleep processes, including the various neurological and biochemical changes which occur in the brain during sleep, are triggered by this change in Qi flow.

Although sleep is a complex phenomenon and in humans can be influenced by emotions, diet, and myriad factors, the increase in Qi in the Gallbladder channel may be one of the

---

1 The following question arises: Why don't the currents of the 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? 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) Bladder channel flow 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 offsets the inhibitory effect. A nice balance is thus achieved.
strongest triggers for the sleep process. The Du channel does not resume its normal level of Qi flow until 5:00-7:00 a.m.

At 5:00 a.m., the Du channel is stimulated by a surge in the currents of the bilateral Large Intestine channels. As these Large Intestine channels cross over the face just below the nose, they traverse the midline at Du-26 on the philtrum (the groove above the upper lip). This stimulation to Du-26, combined with the flow of Large Intestine channel Qi into Yin Tang, helps the Du channel resume its “awake” level of intensity.

**Perpetual excess in the Gall bladder channel in PD**

When, in PD, Qi flows constantly from the Stomach channel into the head portion of the Gallbladder channel, it makes the Gallbladder channel constantly excessive. The constant excess current in the Gallbladder channel is similar to the surge in Gallbladder channel Qi at bedtime. Any surge or excess in this channel sends an electrical signal to the brain to “stop dopamine release.”

In healthy people, the Gallbladder channel only produces this “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 PD, 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 begins to be broadcast to the midbrain twenty-four hours a day – on one side of the brain. This causes the partial substantia nigra cellular dormancy that eventually, over decades, puts the brain’s dopamine production system into a partial hibernation-like state.¹

Dopamine is a major neurotransmitter. One of the things it regulates is the arousal of consciousness. In a healthy person, dopamine levels decrease during sleep.² When the Qi in the

¹ If injuries are present in both feet, the decrease-dopamine signal may be stronger. There will be more on this subject later.

² Even as recently at the 1990s, medical students were being taught that dopamine levels increase at night. The incorrect thinking was this: Parkinson’s patients appear to have too much muscle tension. Though this flies in the face of the poverty of movement symptoms of PD, it does fit, superficially, with the rigidity symptoms of PD.

Since then, it has been proven by researchers in the field of psychoactive and addictive drugs that dopamine is a major stimulant; it is present in increased quantities during periods of activity, and it drops off during sleep. However, most introductory medical school texts and most neurology departments continued to teach – almost to the end of the 20th century – the disproved myth that dopamine is present in higher quantities at night. This wrong supposition is finally being beaten to a pulp by the findings of the National Institute on Drug Abuse. Today, your fourteen-year old niece learns about dopamine in her health class in the unit on drug abuse. She learns it is a stimulant, not a relaxant. If a neurologist went to school between 1950 and 1990, he may still think that dopamine is a relaxant and that dopamine levels are higher at night.

On a fun note, a study commissioned by a pharmaceutical company set out to prove that there was no truth to patients’ complaints that round-the-clock L-dopa contributed to restless sleep. The results of the study proved that, in fact, the medication did contribute to restless sleep. Just the same, the published report stated that because of the obvious (and the word “obvious” was used without any supporting details) benefits of having consistent levels of dopamine in the body at all times (?), patients should continue to take dopamine-enhancing drugs around the clock. One hesitates to examine the motive too closely here…
Du channel is decreased (due to the influence of increased flow in the Gallbladder channel), the production of dopamine is also decreased, as it is during sleep.

**Asymmetry in the channels of the head**

In Parkinson's disease, the Qi of the Gallbladder channel is usually excessive on only one side. (We have seen PDers with injuries on both feet. These PDers have excess Gallbladder channel Qi on both sides. Also, they do not have tremor.) This excess in turn somewhat effects the Du channel. However, this excess, because it is only on one side, is not as strong as the normal, sleeptime surge in both Gallbladder channels which is strong enough to diminish the Du channel to the extent that self-awareness ebbs and sleep begins. Instead, the change which this one-sided Gallbladder excess causes in the Du channel is only a half-strength go-to-sleep signal. Meanwhile, 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 50% decrease in dopamine release and dopamine-based consciousness. Thus, the Qi in the excess (i.e., foot injured side) Gallbladder channel is not at a level adequate to induce sleep, although it does force the Du channel to maintain a state of pre-sleep muscle relaxation and diminution in overall dopamine levels.

During wakefulness, dopamine is produced in the brain and stored in vesicles, ready to be released in response to joyful or creative impulses or feelings. During healthy sleep, dopamine release and dopamine production are minimal.

**The Parkinson’s pre-sleep condition**

In PD, 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 and creates a state in which there is a physical/muscular sense of pre-sleep weariness all day long.

Even so, mental alertness can be maintained through exertion of will-power and the intentional creation of a sense of urgency, which triggers the body to produce ever-higher levels of adrenaline as a substitute for the decrease in 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 and 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.”
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 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.

**Brain asymmetry and restlessness: the internal tremor of Parkinson’s**

If the foot injury is only on one side of the body, as it is in the majority of PDers in our experience, the eventual short circuit alteration in Gallbladder channel energy on just one side of the head creates a schism between the two brain halves.\(^1\) This imbalance between the brain sides may contribute to the internal tremoring, a deeply internal sense of restlessness, a slowly (over years), growing sense of something being wrong inside.\(^2\)

If *both* legs and/or feet are injured, and *both* left and right side Stomach channels begin flowing backwards, this internal sense of restlessness and internal tremor will probably not develop even though the other symptoms of PD may slowly appear. Some of our PD patients who had poverty of movement, rigidity and balance problems but *no tremor* had damage in *both* feet.

The schism between the brain sides due to the electrical imbalance may contribute to the internal tremor that is very faint at first, causing merely a low-grade restlessness. Over decades, the internal tremor increases, creating a sense of anxiety which ultimately can prevent sleep or relaxation. This blend of pre-sleep torpor, combined with excessive development of will power/adrenaline and the sub-clinical, internal imbalance and anxiety, may cause, in some patients, what one PDer referred to as the "push-pull" of Parkinson’s disease.

As mentioned, dopamine production for consciousness and motor function is produced primarily in structures within the midbrain, which are stimulated by Qi flow of the Du channel. When the Du channel flow is decreased, as in PD, stimulation of the midbrain to produce dopamine is also decreased. Whereas dopamine is normally produced on an "as needed" basis, the signal of "need" is perpetually reduced when Du channel Qi flow is constantly decreased due to unending Gallbladder channel excess. Following the “use it or lose it” principle of physiological efficiency, this lack of dopamine usage results in gradual reversion to neutrality (undifferentiation) of dopamine-producing cells, and eventually, over decades, reduced capability for dopamine production and a reduction in dopamine receptors, reuptake enzymes, and all parts of the dopamine system chemistries.

---

1 We propose the existence of this left-right schism because, during recovery, and recovery from tremor, in particular, PDers have reported a sensation which they describe by phrases such as “it felt as if the brain sides were realigning, and when the brain sides became completely aligned, the internal tremor just came to a complete stop and never started up again.”

2 Sometimes the restlessness caused by the brain schism, and subsequently the Parkinson’s, can develop more quickly than the ten-to-twenty year norm for short-circuiting to begin at ST-8. A twenty-six year old patient who already had most symptoms of Parkinson’s (although her symptoms were still mild) told me that her internal restlessness developed almost instantaneously. Her grandmother had frequently recounted the story of her baptismal accident at age two months: “The priest was getting ready to drop you in the water when his right hand slipped. His left hand was still holding your foot. You fell headfirst backwards into the holy water, but he was still holding your little left foot. Your foot bent all the way backwards, an impossible bend. After that, you were always restless. You were a contented little baby before that. But after that, you were always restless.”

John Bateson, illustrator of this book, developed Parkinson’s at the somewhat early age of forty-eight, but his internal tremor may have started much earlier. He remembers thinking, at age eight, that “there is something deeply wrong inside of me.” He also remembers telling himself not to worry because “by the time they know what’s wrong with me, they will have found a cure.”
Decline 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 sending incorrect signals to all the tissues in its zone of influence as far up as the back of the jaw, creates cell alteration, muscle rigidity, and organ weakness all along the reversed path of the channel, especially on the side first affected. Nerve contact between the brain and the affected area declines. This decline involves nerves that regulate proprioception, sensitivity, and motor function. The problems may eventually become bilateral due to the mutual influence of paired electrical currents.

Electrical snafu at the neck

The disarray in the neck portion of the Stomach channel prevents the normal crossing over of the Large Intestine channel at the neck and back corner of the jaw and onto the face. The 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 facial terminus of the Large Intestine channel at Yin Tang), serves to create a pocket of current-free, energy-empty flesh in the area of the face.

Electrical flow change in the Large Intestine channel

The Large Intestine channel, blocked at the neck and 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.

Tremor

Atrophy on the Large Intestine channel combined with internal tremor and restlessness

The decrease in brain-to-muscle neural connections in the muscles between the index finger and the thumb (and in other parts of the hand if other channels become involved, due to additional injuries in the arm) leads to lack of energy and poor motor response in the index finger. Meanwhile, the disarray in the Large Intestine channel near the index finger (acupoint LI-4) prevents the normal flow of current from the side of the wrist (near the end of the Lung channel) into LI-4, the source point (major starting point) of the Large Intestine channel. The muscles atrophy between the thumb and the index finger, since they no longer receive any electrical signals from cell-directing channel Qi at this major Qi crossover place. The very small area between the index finger and the thumb, in the absence of growth-directing signals, can be one of the first areas where the muscle atrophy, loss of Qi, and loss of brain-to-muscle connection become apparent. This area, after it becomes somewhat lifeless, becomes susceptible to the relentless rhythm of the internal tremor. The index finger, when not being consciously moved, begins to move in time with the internal tremor. Eventually, as other areas atrophy and lose conscious connections with the brain’s motor area, they too begin to tremor.

Anxiety tremor, a larger variation on the usual tremor

A particular variation on tremor can occur when a person is experiencing a higher than usual level of anxiety or when trying to eat (stimulating the Stomach channel). It seems as if this
larger set of tremor-like movements may actually be caused by adrenaline hitting the larger, no-
longer-in-control muscles. The index finger, as already described, is not under good brain
control. However, the line of tissue that follows the Large Intestine channel farther up the arm is
also, in many parts of the arm, degenerating.

In ordinary circumstances, this muscle farther up the arm is less prone to uncontrolled
tremoring. The weighty muscles on either side of it keep it somewhat under control. However,
when increased anxiety starts sending spurts of adrenaline throughout the body, this adrenaline
may well be affecting the areas alongside the degenerating tissues on either side of the Large
Intestine channel. These tissues alongside may not be completely useless, but they are also not
under good mind-muscle control. When the spurts of adrenaline hit, they may brainlessly fire off
with rapid flexions and extensions moving in time with the internal tremor.

More about tremor

There are two components to tremor: location and intensity. Location of tremor, before it
spreads and becomes body-wide, is related to tissues that have become weakened and have
diminished brain-to-nerve function because of local channel-related problems. Intensity of
tremor is related primarily to mental/emotional conditions. Tremor is highly susceptible to
emotional state, temperature, immune system vigor, social stressors and fear: factors that are
influenced by adrenaline and dopamine levels. Fear of tremor itself can even contribute to an
increase in the intensity of tremor.

Because the predominant force behind tremor intensity can be emotional, and may even
be related to a long-delayed shock response, a further explanation of 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 that are related to channel disturbances.

Loss of Coordination

Left-Right integration

In a healthy person, when the right-side Large Intestine (arm) channel surges slightly, 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 the Stomach channel. These surges of current drive the left-right coordination of arm
swing with leg swing.

When the arm swings, the faint increase in Qi in the channel caused by the conscious or
subconscious act of swinging the arm sets off a coursing of energy that results in an equal surge
in Qi in the opposite leg. This surge supports, even encourages, the anterolateral part of the leg
(the Stomach channel, the part of the leg that is called upon to move when walking forward or
turning slightly to the side) to move its muscles. 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 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 in tandem with 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 cannot complete its circuit, it no longer flows to the opposite side of the face. Instead, in PD, because Large Intestine channel Qi becomes blocked at the neck intersection by the rebellious Qi in the Stomach channel and cannot skirt the lips and cross over the face, the ability to integrate left-right movement gradually deteriorates, especially the integration of arm swinging with the leg stride.

**Balance**

In advanced stage PD, loss of coordination becomes severe and is coupled with loss of balance. The balance centers in the brain are governed by the Du channel. The Du channel is diminished, as we have seen, by the presence of excessive Gallbladder channel Qi. The resultant decrease in Du channel Qi contributes to the eventual decline in balance, as does the extinguishing of the left-right integration of the Large Intestine and Stomach channels. Diminished Qi flow through the head may contribute to the “backwards walking” and falling over backwards that is experienced by some PDers.

The inhibition of motor initiation also contributes to balance problems: the dozens of tiny, almost instantaneous corrections in posture that are necessary for balance are no longer possible. When movement becomes slow and calculated instead of effortless and automatic, correcting movements simply cannot be performed in a timely manner. Balance thus becomes difficult. This inhibition of motor initiation may contribute to festinating gait (the torso moving forward faster than the legs can keep up with) and falling without the ability to use one’s body in any way to break the fall.

**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 section will explain why Parkinson’s symptoms eventually show up on the uninjured side.

When similar 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. It can also be felt in the Qi of humans: 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. This mutual influence works both ways: the Qi that is running correctly on the uninjured side helps maintain some degree of normalcy in the injured-side channel even though the energetic blockage is disrupting the channel flow.

```
A____________________________
B____________________________
C____________________________
B____________________________
A____________________________
```

Fig. 5.7
Parallel currents
The Qi in the Stomach channel of PDers is palpably more disrupted on the side where symptoms first appeared. The other side 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 may cause the Qi to run, briefly, in the correct direction on that side. A similar stimulation of the injured side may cause the Qi of the healthy side to run more vigorously in its wrong 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.

**HYPOTHESES SUMMARY**

In summary, the foot injuries we’ve seen in our PD patients can explain all the anatomical symptoms of Parkinson’s disease. (The “unknown cause of decrease in dopamine cells” theory cannot explain any of the anatomical changes that occur.)

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 inhibit dopamine release.

The injury-based electrical changes in the brain keep the dopamine-releasing part of the brain in a sleep time (very low dopamine) mode. Dopamine production slowly declines because dopamine use is severely inhibited. Some dopamine-producing cells become dormant.

These electrical changes also set in motion the deterioration of muscle and nerve function in those areas of the leg, torso, arm, and face whose growth and function are regulated by those particular, and now aberrant, electrical currents.

Contributing even further to dopamine release inhibition, the presence of an ongoing injury causes the release of adrenaline, a neurotransmitter that is released in response to the perception of danger. The presence of adrenaline can serve to inhibit dopamine release.

Furthermore, as you will learn in later chapters, many PDers have inadvertently or intentionally cultivated certain negative thought patterns similar to those that arise out of physical or emotional pain, or fear. These thoughts reinforce the use of the sympathetic nervous system and further inhibit the release of dopamine.

Because healing only occurs when the mind is in non-emergency mode, these sympathetic system, adrenaline-based, thought patterns serve to inhibit healing of the injury.

The PDer becomes habituated to using comfortable, low levels of adrenaline. His injury contributes to an ongoing fear or pain response (use of the sympathetic nervous system) and the concomitant suppression of dopamine release. Oppositely, the fear or pain response and the suppression of dopamine contribute to the inhibition of healing of the injury.

¹ Acupuncturists often treat 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 treating the same 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; the stimulation will serve to increase energy in an already backwards system.
Eventually, because of the brain’s extreme plasticity (ability to change) and its “use it or lose it” prioritizing of cell 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.
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 SIX

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 James Tetrud, 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.


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 distain 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.


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 limpaness; 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. 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

---

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 potion 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.¹

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.”²

Note: the location of the pill rolling tremor and its extension up the arm corresponds to the path of the Large Intestine channel. The lower limb tremor usually begins near the ankle and most often follows the lines of the Stomach channel and/or Gallbladder channel.

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

¹ Eating stimulates the Stomach channel. If the Stomach channel is in disarray and/or if the body is locked into sympathetic mode (during which eating is automatically stressful), the combined stressors can lead to an increase in anxiety and/or tremor when eating or when thinking about eating.

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 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

¹ Many well-meaning physical therapists teach classes for Parkinson’s people in how to keep 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 whom 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: “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.
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.

A common misconception among the general public is that “anything that tremours 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 antianxiety 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 which can 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.

**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.

---

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 to keep them 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 drugs stopped being used.
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.

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, sometimes even to the barely trained naked eye, 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. As an aside, when we examined these glaringly misdiagnosed people, they clearly did not have a Qi irregularity in the leg.

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. (Continued on next page.)

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.
**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 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.”

---

**The L-dopa test**

Recently, despite the fact that nearly anyone who is moving slowly 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 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 again, so will nearly anyone else. 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

---

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.

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 further 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.
**Drug- or toxin-induced Parkinsonism**

Some patients have come to us with a long history of using antidepressant or antianxiety 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 ticings, they have no classic symptoms of PD. And yet, they were given a diagnosis of Parkinson’s disease by their neurologists. Interestingly, these patients almost never have 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 only 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.\(^1\) Increasing amounts of medication must 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 20\(^{th}\) 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)!\(^2\)

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.

---

1. The direct relationship between the amount of L-dopa dose and a decline in dopamine receptors was proven in the Ellidopa 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.

2. 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.
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 dopamine problem that results from it.

**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 and in the publications of 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

\(^1\) I have 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 it was accompanied by information provided by the DBS 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.
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.¹

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.

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 paradigm. The dominant 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, except that the mental signal to end the “state of emergency” 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 joy.

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
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 PDers have a hard time registering this information after they hear misinformed MDs or 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 PDers 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 remains the dominant 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 deep brain stimulating systems manufacturers. 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 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,
“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, tho’ they come from the ends of the earth!”

- Rudyard Kipling

CHAPTER SEVEN

WEST MEETS EAST

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 have 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.


The other two articles described cases of rheumatoid-like and psuedorheumatoid 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.  

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 that 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

---

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 bizarre enough to include. Both of these people did not think they had numb feet. Yet when I tested their feet with needles, they had almost no response. This information showed me two things: a horrible level of numbness can exist in the feet of many PDers, and they don’t realize the extent of the numbness.  

The truth of this is borne out during recovery. When blood and nerve supply return to their feet, the frostbite-like burning can be agonizing. After recovery, they often say to me with pathetic amazement something like: “Did you know that it’s possible to feel the texture of your socks on your feet? Or feel where your toes are, even when they’re inside of shoes?”
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, 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 anterolateral muscles that didn’t work and medial and posterior muscles that did working. 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 anterolateral (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, ranging from fever to a nail through the foot, which fit under the heading of Excess. Rebellious Qi (backwards flowing Qi) is considered one of the many forms of Excess Qi.

---
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.
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 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 the weight of the head pulls it backwards but the tension in the neck pulls the head from 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

---

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 needed to have written to me. The western reader, possibly scratching his head over this, may be able to relate 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 PDer 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 have a severe aversion to needles, both Asian and western.
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 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

---

1 Many of my PD patients have pointed these unyielding muscles on the anterolateral 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.
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 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 more than thirty 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.

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 that 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.

- 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
  o 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
  Most 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.
  o 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.
  o cogwheeling in the ankles
    The “cog” is at the Stomach channel point of the ankle, worse on the SSFA.
  o foot drop, worse on the SSFA
  o 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
  o poor small motor skills: cutting food, picking up small things, doing buttons
  o 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.
  o cogwheeling at the wrist
• cogwheeling at the wrist most pronounced at the intersection of the wrist with the Large Intestine channel
  o 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 turned inward or outward
  o 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
  o 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). Some of the other body-wide symptoms are not so well recognized by western doctors. The following section will present some of the auxiliary body-wide symptoms that present in many, if not most, of my PD patients.

**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 that became involved if 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.

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.
• 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: he may be at his most dynamic and
high-powered, or it may be at the lowest level: he is utterly unable to get up and get
going.
• fear of cold, fear of getting cold feet, extreme difficulty in warming the hands or feet,
even Reynaud’s syndrome
Nearly 90% of the PDers had problems with cold. The other 10% seemed to have
problems with heat: they got overheated very easily.¹ This problem of temperature
regulation, particularly of cold, is not associated with any channel but is associated with
the Asian medicine element of Water, which is associated with the Kidney and the
adrenal glands.²

The list of body-wide symptoms suggests discord in the Gallbladder and Stomach
channels and an overall problem with the Kidney (adrenaline) function. This supports our
hypothesis that current 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.

Another body-wide symptom, poor temperature regulation, can result from diminished
flow of Kidney channel Qi because of a short circuit on the foot. In this short circuit, illustrated
in chapter five, fig. 5.1, the Kidney channel, which should flow over the medial ankle, shorts out
into the floor because of static at the ankle from erratic, disruptive energy in the nearby Stomach
channel. This grounding out can not only can make the feet feel as if they are glued to the floor;
it can diminish the normal flow of energy into the channel that regulates kidney and adrenal
gland function. This erratic pattern can be felt easily (by a trained hand) in the foot and ankles of
many people with Parkinson’s.

Adrenaline, when in sympathetic mode, and dopamine, when in parasympathetic mode,
play roles in temperature regulation. Thus, incorrect energy in the Kidney channel, which leads
to incorrect energy flow to the adrenal gland, may lead to poor temperature regulation.

¹ We did have one patient who never seemed to notice whether it was warm or cold out. His wife said that,
in their forty years of marriage, he had never remarked on the ambient temperature, even though his feet were
always “slabs of purplish-gray ice” and his skin was often very cold to the touch. When he recovered from
Parkinson’s, he started complaining to his wife for the first time that the house was either cold or hot.

² “The Kidneys,” in Asian medicine, encompass the adrenal gland. The adrenal gland is attached to the
kidney. The word “adrenal” comes from the Latin “renal,” meaning having to do with the kidney; “ad” means “to.”
Attitudinal symptoms

Please note: the following list includes symptoms that we recognized soon after starting this project and symptoms that we discovered over several years, including the dissociation-related symptoms that we stumbled across in the period from 2002 to 2007.

• 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
• 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

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.

• fear of anxiety or stress-inducing social interactions
• strong aversion to highly charged emotional situations, particularly situations involving anger

---

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.

2 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 curiously. 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 had to put up a strong face.
• high aversion to being in the vicinity of people who are being overly assertive, threatening, or who might become angry
  o 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.”
  o 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.)

1 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 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,
Location of the attitudinal symptoms

At first glance, the Parkinson’s personality and other attitudinal symptoms may 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

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, one branch of the Kidney channel travels to the Pericardium and influences 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).

Therefore, although attitude may appear to be body wide, the underlying channel pathologies that contribute to attitude symptoms occur in specific channels and body areas.

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 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.
In the case of Parkinson’s disease, electrical aberration at the end of the Stomach channel can be felt disrupting and diminishing the flow of the Kidney channel. Therefore, though the symptoms of anxiety and the insufficiency of joy or courage are body-wide, a reasonable explanation for these symptoms can be placed on the PD symptom map. The location of this channel-based explanation is located in the confusion that occurs at the end of the Stomach channel, on the foot, where the Kidney channel is affected by the Stomach channel irregularities.

**PD personality past and present: an aside**

Remember, not every person with Parkinson’s disease has all the above symptoms. Some may not have the Parkinson’s personality. I have met people with bona fide Parkinson’s who do not fit the above personality profile. 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 of the symptoms of 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 spasm is a sign 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 suggests 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 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.

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.
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

**ONE NEW IDEA**

The reader must 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 politics forbids 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

Traditionally, channels were the starting point for any study of Asian medicine. Therefore, some of the old channel theories are still taught, even though they are taught as historical relics. As for channels flowing backwards, failing to flow, or flowing into other channels, those ideas were old, established ideas.3

If I accepted the idea that Qi was real and that it followed the rules of electrical movement, 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.

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.

Whether it was in the literature or not, I could feel the Qi flow in my PDers; 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,

---

1 Of course, in advanced PD, the problem may eventually effect 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 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 followed the party line and did not believe in the existence of channels.

3 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.
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.\(^1\)

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 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.

**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 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, after examining hundreds of people with Parkinson’s, I was able to construct a hypothesis that did answer most of the questions about how a Stomach channel problem could cause brain changes. The chapters that follow explain how the rest of my questions, questions

---

\(^1\) 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. The use of two different metals creates a small electrical current. Using one gold needle and one silver needle, I can make a very low powered battery that sends a current through the skin or organs. I made a mistake. I reversed the proper positions of the gold and silver needles. Instead of pulling current through her midbrain, I was pushing it backwards. 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 alertness.

\(^2\) 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.
having to do with personality and an apparent reliance on the sympathetic nervous system, came
to be answered.

**SUMMARY**

By showing the extent to which all the well known and lesser-known symptoms of
Parkinson’s are located on just two channels of the body, I have shown that the location of PD
symptoms corresponds to the location of 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 a pattern of long-maintained Rebellious Qi in the
Stomach channel complete with the shunts and short circuits described in chapter five.

Another point that makes itself clear is that the current paradigm, the system-wide
insufficient dopamine theory, simply does not match the limited locations or the mixed natures
of the symptoms.

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 fear- or consciously-induced dissociation-type
trauma response that was never turned off.

The Big Four symptoms in Parkinson’s disease don’t have a logical connection. The
symptoms of sustained Rebellious Qi in the Stomach channel make sense. 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 on recovery 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. That disorder is curable. The two syndromes are exactly the same.
“...So near, and yet so far.”

- Alfred, Lord Tennyson

CHAPTER EIGHT

PARTIAL RECOVERY

Hurrah! ...sort of

Ta da! By 1999, 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, and supported this idea further in chapter seven, 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-PDers 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-PDers 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.

Fear of the dentist

For example, one PDer was doing so well that he was once again able to cavort with his nephews around the tidepools at the beach. His posture was good, his lifetime constipation was gone, and, according to his new wife, he had recently chased her around the dining room table several times. His improvement had been slow and steady over the course of a year.

However, after months of feeling increasingly vigorous, came the day when he was supposed to go to the dentist to begin a series of dental reconstructions. He started tremoring first thing in the morning. By midmorning, he was curled up on the floor in a fetal position, unable to move, tremoring violently. Of course, he didn’t make it to the dentist. It took him several days before he could move somewhat reasonably. Although, after about a week, he could once again move normally, he was convinced that Parkinson’s had returned.

The “Return of the Parkinson’s,” in turn, convinced him that his good days were only teases. This led to a period of over a year during which he continued to report increasing ease in motor function during the times when he could move, but collapses into worsening immobility when some unexpected fear crossed his path. To me, it didn’t seem as if he still had Parkinson’s. But it did seem as if he no longer had a way to access even minimal amounts of courage.

He was convinced that this was a normal presentation of Parkinson’s. He was increasingly wary about “Return of the Parkinson’s” unexpectedly rearing its ugly head. And his fears were rewarded – as fears usually are.

Almost any time he had to do something that had a small fear factor, such as go to the dentist or meet new people, he would collapse into a state in which he could hardly move. If his life was arranged in such a way that he had no responsibilities, no problems, and nothing that he didn’t like, he could go for weeks with no symptoms whatsoever.

When I’d first met him, his symptoms had been only moderate, even mild. He had very early Parkinson’s. He had never taken any antiparkinson’s medications. Now, just two years
later, he was either able to move perfectly normally or else he was nearly paralyzed. There was not much middle ground. His structural symptoms, (the symptoms that are located along the course of the Stomach channel and Large Intestine channel) were gone. However, movement initiation and/or tremor problems seemed to come and go with his thought patterns.

Soon he found himself in a condition in which movement inhibition could be triggered by merely thinking about movement inhibition. Over the next year, more and more thoughts were able to trigger movement inhibition symptoms. And yet, when he accidentally forgot about Parkinson’s, for example, during a party or over a long holiday week, he moved normally until he remembered that he couldn’t.

He was absolutely certain that he still had Parkinson’s disease. He kept coming to me, wanting me to find the problem in his Qi flow that could explain the anxiety and the increasing number of Parkinson’s-like outbreaks.

But it seemed to the PD Team members and me that some mental or emotional trigger was igniting these events. He clearly did not have a neurotransmitter deficiency; when he was in a good mood, he could tear around like a young mountain goat.

He would have none of that. He assured us that he had no emotional problems; he still had Parkinson’s, a neurotransmitter deficiency, and I had failed to cure him. He just knew he was only going to get worse. And he did.

And he wasn’t the only one.

Six partial recoveries

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 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.\(^1\)

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: they had return of facial expression, return of arm swing, return of the atrophied muscle between the thumb and index

\(^1\) 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, high as they were on the drugs, they weren’t particularly susceptible to anxiety and motor inhibition.

Since we stopped working with medicated PDers, we have found that a majority of unmedicated PDers is at risk for partial recovery. A later chapter will explain in detail the types of attitudes and emotional postures we see in those who have an easy recovery and the characteristics of people who need to do a major mental adjustment in order to fully recover.
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, such as when walking in the woods, when doing the laundry, or when 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.

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.

---

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.

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.
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). Clearly, 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.”

Another partially recovered person could once again play soccer with his teenage sons and could move easily during most activities. He was so completely recovered that, when he translated for me at a class in Germany and mentioned, on the second day of the class, that he’d had Parkinson’s disease, none of the class participants believed him.

Nevertheless, whenever he got into his car, he could barely move his right arm through the motions necessary to operate the gear shifter. This one problem was steadily worsening. If he was outside of the car, he could, with grace and vigor, move his arm through all the positions necessary to shift gears. However, once he got in the car, he could not perform these motions. At the time, we merely felt that this was ironic, considering that his greatest fear was losing his ability to drive. We have since come to understand that this situational immobility was caused by his deep fear of not being able to drive.

Yet another partially recovered PDer was doing well except for intermittent foot cramping accompanied by tremor. The cramping was getting more frequent and more painful. She realized that the foot cramping was caused by wearing shoes. She started wearing sandals
and the feet stopped cramping. But within a few months she realized that thinking about wearing shoes caused the same excruciating foot cramps and tremor. And her mind seemed to wander increasingly into the subject of shoes.

We later learned that her mother owned hundreds of pairs of shoes, but begrudged the money spent on her children’s food. When this partially recovered PDer was a child, she had often said to herself, “A person should love their children more than their shoes.” The negative connection with shoes had slowly grown in her brain to the point that, when she put on shoes, her foot went into dissociation mode, which can include foot and toe spasms. The shoes-are-bad association continued to grow in her mind: eventually, just thinking about shoes caused her to revert into a partial dissociation-type response.

(To better understand the foot spasm relationship with dissociation, picture a mouse that has been attacked by a cat and then dropped. The mouse, if dissociated, will lie motionless, but not limp: his toes and limbs will be pulled in hard, as if he is experiencing rigor mortis. This reaction will be discussed later in detail.)

Not yet understanding the underlying problem, we wanted to convince ourselves 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!

But somehow, the solution evaded us. And our research continued.

The next few chapters briefly describe the continuing path of our research. In particular, they explain how we stumbled across a peculiar habit of most PDer’s: the mental habit of staying in sympathetic mode. Then, for a few years, we tried to help them get their minds out of sympathetic mode but to no avail. Finally, in 2007, we realized what mechanism that they were using to be simultaneously in a state of seeming self-control while having uncontrolled negative thought patterns characteristic of the sympathetic mode: the mechanism of an ever-spreading, ever-linking to new brain compartments, ongoing condition of compartmentalized dissociation.

A dissociation response is supposed to end automatically, as soon as the surge of endorphins is neutralized and the almost-killed animal begins to feel his body once again. The “feel his body” component is important; when endorphins wear off, the body is almost oversensitive to touch, light and sound. For example, when a heroin user’s dose wears off, his body is in a state of heightened sensitivity for a while. This same heightened sensitivity should also occur when endorphins wear off. This increase in sensitivity is what restarts the heart’s basic chain of command, the heart’s ability to initiate vitality, accompanied by either adrenaline or a flood of dopamine. Healthy people who have had a harrowing, near-death injury and “come back” are often in a condition of over-the-top sensitivity for a short while. Colors seem brighter, sounds are more beautiful and intense. This heightened sensitivity “turns the heart back on.”

PDer’s are in a dissociation response without end. What we didn’t realize is that PDer’s had started this whole process by commanding themselves to not feel pain: creating a dissociation-like response. This self-instruction, can, in some people, allow the dissociation response to start – but if maintained, it can also prevent the response from turning off. A mental
instruction to not register sensory input or emotional pain prevents the dissociation response from ending automatically, as it is supposed to do when the crisis is over.

A heightened sensitivity to the body and to the environment is the event that turns off the dissociation response and turns back on the full functionality of the heart nerves. But partially recovered PDers, through denial-of-feeling mental habits, keep themselves in a slowly expanding mind-set that specifically prevents turning off the dissociation response.

We also came to learn that most PDers, after their diagnosis, are deeply worried about dopamine levels, not adrenaline levels. But their long-term mental habits of using, primarily, adrenaline-releasing thoughts can create movement-inhibition monsters of its own—if adrenaline levels begin to decline. When, due to effective treatment, a PDer’s foot injury begins to heal, his adrenaline-release levels also decline. But if his mental habits keep him locked in any type of sympathetic mode, including a dissociation mode, he will still need adrenaline, not dopamine, to initiate movement. With the foot injury gone, adrenaline levels can become too low to support movement or to support courage when confronting anxiety. We began to suspect that this decrease in adrenaline after recovering from the foot injury could cause the rapid collapse into panic and immobility that occurs in some partially recovered PDers.

But it took us several years to figure this out.

Meanwhile, we plugged along, slowly puzzling out the neurotransmitter and thought pattern commonalities in people who became stuck in partial recovery.

And 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 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 earlier might have been to someday publish my research. Her words gave me hope for the Little Project and added to my sense that this project was a part of something far bigger than myself. 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.” (Continued.)
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 2007, and also our new hypotheses about mental/emotional habits and chronic reliance on the sympathetic system.

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 has helped with the proof-reading, likes the time-line format. He says it adds a touch of “you are there.”

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! She had also, she confessed, spoken to a physics professor to have my statements about parallel electrical circuits confirmed.

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 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.

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.¹

¹ I wish to credit Dr. Fred Jones, PhD, who advised me during this period. Dr. Jones had once taught the medical research course at the medical school in Rochester, NY. I was very uncertain what to do with my finding that Parkinson’s disease was curable. He told me, “You have two choices. The first is, years from now, people will say ‘There used to be a woman who could cure Parkinson’s, but now she’s dead.’ The second is that you can figure out exactly what’s going on and publish your results.”

He also gave me a few other pieces of sage advice: “Don’t waste anyone’s time with nit-picking statistics; show me evidence that I can see from across the room!” and “In doing research on humans, the single-case study is increasingly accepted as valid evidence.”

The latter statement was with regard to the modern idea that hundreds of people, both test subjects and control subjects, must be tested in a “double-blind” study in order for research to be valid.

Some types of research do demand this type of test format. For example, drug testing, which needs to determine what percentage of people can use a drug without dying or suffering unacceptable adverse effects, must be done in a double blind manner on hundreds of people. However, when working with most illness scenarios, no study, however large, can accurately predict what will occur for any one person. Although the general public still finds safety in numbers, researchers in medicine are increasingly agreeing that, especially if mental or emotional issues are involved, what happens to any given person may have almost no relevance to (Continued on next page.)
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. 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 dentist phobia in the previous chapter, 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 dive 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

what will happen to any other given person. And researchers are increasingly finding that, in nearly every case involving a human, mental and/or emotional issues are involved.

More than a year later, I shared with Dr. Jones my finding that 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 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.”
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. It came out that some PDers – the ones who recovered most quickly and completely – had long ago used a consciously constructed mindset of pretending to have no physical feelings only for a short while – maybe a few minutes or days– at the time of foot injury. Forever after, the emotional denial only applied to the foot injury. The foot injury had been, at the time, highly dangerous, potentially traumatic to self or parents, or even life-threatening – from the child’s or young adult’s point of view: hence, the need to pretend it hadn’t happened.

The case study of Gus in chapter twelve is an example of very limited dissociation. Gus is the PDer whose foot had been smashed by an ammunition box during WWII on the same day that every person in his platoon died – except for him. His foot injury, when recalled at age 84, sent him into shock, complete with nausea, and violent shaking. He was an easy-going guy with no excessive level of drive or intensity of purpose. His foot injury had occurred after most of his personality had already formed. His mental block was centered around the foot injury and the horrible Day of Death in Japan. He had never remembered that black day, had never mentioned it to his wife. He had completely blotted it out until we awakened his memories by treating his foot.

But the important point here is that, aside from mentally blocking out one day, the rest of his personality was not involved in a posture of mental and emotional wariness.

At the other extreme, some PDers had consciously cultivated, over decades, powerful denial and repression of body awareness and their own emotions. Some had even proudly integrated the denial of personal-injury feelings into their adult personality. Some PDers had expanded the number of mental arenas in which they pretended to feel no pain to include the arenas of social disappointments and other, not life-threatening, physical injuries. Some had such strong emotional suppression that they were not capable of crying or feeling emotional pain. Some confessed that they had never cried or outwardly lost their temper since early childhood.

---

1 Some PDers are not only proud of their capacity for denial and repression, they feel that their rock steady emotions (or lack of emotions) are proof of some rare purity of soul. These people have confused the self-deceits of denial and repression with the wisdom and compassion that leads to equanimity of mind and mood and the correct (unfearful) interpretation of pain signals from physical or egoic injury.
even though, inwardly, they felt an almost constant emotional strife, self-pity, and/or emptiness, maintained through a continual inner monologue of concern, judgment and criticism.

Some of these adults had learned, as adults, to show emotion, but often the predominant display of emotion consisted of tears of self-pity.

Interestingly, most of the people who were most deeply mired in partial recovery also had a powerful aversion to the word “surrender.” I don’t know how many times I’ve heard an immobile, violently trembling PDer tell me with defiant, if halting and whispered, words and with no trace of irony, “It’s my refusal to surrender that’s made me what I am today!”

Yet others had employed a broad and deep level of the body-denial and feel-no-pain emotional stance for most of their childhood and early adulthood but had, at some stage in their adulthood, worked hard to overcome the consciously-created habits of living in wariness, of feeling little or none of their own emotional and/or physical pain. They had taught themselves to cry or taught themselves to experience feelings, however painful. They had been able to make this daring change because they had decided to work on a change of heart.

Curiously, this change of heart was often described as a decision to surrender to some Goodness in the universe, and/or grateful acceptance of their difficult past experiences as a part of some great mystery that has, at its core, beauty and Love. These people recovered easily; when we fixed the purely physical problems of foot injury, the electrical disarray set in motion by the foot injury straightened itself out. After that, they were symptom-free, in a relatively short time (ranging from five weeks to about a year).

Also, musicians recovered easily. We had five professional musicians with Parkinson’s, including a composer and a conductor. They recovered very quickly, with no emotional glitches.

The two latter groups, the musicians and the ones that, prior to diagnosis, had taught themselves to surrender to Good, surrender to heart feelings, were able to recover most easily from Parkinson’s disease after the foot injury was removed.

We began to suspect that even the rate at which PDers’ foot injuries responded to treatment and also the subsequent blooming or withering of their capacity for joy and playful imagination might be related to the extent to which the “stay wary and feel no pain” mentality had come to dominate their consciousness.

**Stymied**

This was frustrating. The partially recovered PDers seemed to have some sort of mental/emotional blockage. 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

---

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 copier. 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.
The “Other” side, the injured side of the body

Numbness and poor proprioception

Many PDers 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, many PDers had a high level of numbness in certain areas on the injured foot, though PDers rarely realized it. I tested for this numbness in the following way: I plunged an acupuncture needle deep into an acupoint – a point that is supposed to be particularly painful – on the side of the injured foot, proximal to the big toe. PDers would assure me that they could feel my fingers on their feet, and that they could feel the prick of the needle. Therefore, they concluded, their feet weren’t numb.

But I happen to know that this particular point, SP-3, should send sensory shock waves jangling through the whole body. When PDers were merely able to notice the needle, and didn’t sit bolt upright in pain, I knew that they had serious lack of sensitivity in this area.

This was confirmed during recovery. When full blood flow, warmth, and full sensory awareness returns to the feet, this acupoint suddenly becomes highly sensitive. At this stage of recovery PDers often say things such as “Did you know that it’s possible to feel your own feet even when you have shoes on?” or “Did you know that you can actually feel socks against your skin even when you’re wearing shoes?” or even, “I can feel my feet!”

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.
Getting back to the point, needling the injured foot usually got a poor response, but needling the healthy foot at this point very often got a lively response. Most PDers insisted that I was doing something different on the PD side to make the point less sensitive. I wasn’t.

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.

What was surprising was 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**

Within a few years of starting the project we had noticed something curious. 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. However, the injured side was 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 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.

“What ankle?”

Similarly, if a PDer says that, in the past, he frequently twisted his ankles, I will ask him, “Which ankle, the left one or the right one, did you usually twist?” It is 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 is 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 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 is quite visible via his body language.

Every once in a great while, the patient will 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 will lower his incorrect arm and put the correct arm in the upright position.

Then, 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 will first ask a PDer to raise the arm on his healthy side, instead of the PD side. The PDer doesn’t pause, doesn’t look at the opposite side. He simply raises the correct arm. Of course, if I then want to also check his PD-side response to this test, I must 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 new, 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 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 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, 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 either that “I’ve never been able to do visualizations and I don’t want to,” or “I can’t make light go into my foot and that’s that,” or “Visualization is a sin,” or “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.”

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 are 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

---

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 sometimes cease completely as soon as the mind fully and permanently accepts that that particular body part is back.
All excited over a dead-end

When we realized the extent of PDers’ inabilities 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 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 “blackened 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.¹

Most PDers spent hours struggling to do these techniques. Most of them hated the processes or were even repulsed by them and understandably refused to do them. Others really tried their best at it but their hearts weren’t in it. 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.²

¹ 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 using various practitioners. The results ranged from fleeting to zero. And the simple heart-calming techniques of the Heartmath Institute are not effective on PDers.

² 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 trembling 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 the severe mind-body disassociation.
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
more important function of the Team is that we include, with the treatments, free training for the
patients’ accompanying health practitioners.  

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

---

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.”

---

1 For more information about the PD Team, please visit the website at www.pdtreatment.com
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.¹

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 1999 to 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.

¹ 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.

Dr. Jones said 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, especially if it brings money into the pockets of the “authorities.”

But there is no product of profit to be made for anyone in our findings: the 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, PDers 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 PDers 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 PDers 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.
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 PDers 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 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.³

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 uptaked 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. 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 PDers.


³ 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 agones – 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
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.\(^1\)

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 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.

\(^1\) 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.
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.

We were 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

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.

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 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.
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 that 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 probably 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.1

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 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) mode. 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 compartmentalized 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,

---

1 One patient with classic PD symptoms was very angry with me when her doctor told her that she had never had Parkinson’s. The doctor said that her foot dragging, lack of arm swing, slowed movement, hunched posture, loss of small motor function, decline in voice production, etc, must have been caused by a pinched nerve in her neck. The ex-PDer was so angry with me. She declared, “I wasted all this time coming to your free clinic, and all I ever had was a pinched nerve!”
nevertheless, if something in the mind or emotions might hold a clue to the mystery of the partially recovered PDers.
“The mind can make a heaven out of hell or a hell out of heaven.”

- John Milton

CHAPTER TEN

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.3

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 problems. 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.

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

---

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 some chemical or 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 Most of the placebo tests on PDers have been done on people who are accustomed to taking antiparkinson’s medication. I have to wonder if a PDer learns, via the drugs, how to enjoy the mental state that comes from enhanced dopamine levels and can then replicate that mental state when he takes a placebo. It would be interesting to see if PDers who have never used mood-altering drugs, such as the antiparkinson’s dopamine-enhancing medications, are equally able to respond to placebos or positive suggestion.

Many of my (unmedicated) PD patients are highly wary and skeptical of positive suggestions about their own physiology. It would be interesting to see if these guarded people are able to respond to placebos as well as medicated PDers, or if the “mental retraining” in positive attitude that is provided by mind-altering dopamine-enhancing drugs is necessary in order for the placebo to be effective.
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.¹

**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.² Due to familiarity with drug effects, these people already knew how the drugs would make them feel: happy enough to move.³ Thus, they had developed a specific expectation for drug-enhanced movement in response to their usual dose.

---

¹ If the symptoms of fear or stress are severe enough to produce symptoms that actually resemble Parkinson’s disease, the condition is referred to as “psychogenic parkinsonism.” This newly recognized syndrome is discussed later in this chapter. Because some people do not believe that a person can rapidly, sometimes in minutes, develop symptoms that resemble Parkinson’s disease, I’ll give a few familiar examples in this footnote.

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, 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. These examples may also help to make more understandable the purely psychological and relatively rapid manifestations of psychogenic parkinsonism that will be described in detail later in this chapter.

² 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.

³ 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.
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 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

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.
several years before surgery. After surgery, she was able to hike and ice skate. She eventually learned that she’d had sham surgery.\(^1\)

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.

Maybe the adrenaline response to having a hold drilled in the head, combined with the dopamine-releasing positive-expection (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.\(^2\) 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 subsides as the wire-and-battery induced stimulation 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.\(^3\)

---


3 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 sedating the electrical disarray in the brain, but the surgery itself may also provide an expectation of feeling
Getting back to the implant/placebo study: one group of 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, some of the PDers 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 tremors would stop almost immediately. These PDers rapidly began to experience normal movement initiation. Their tremors 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 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 better. Thus, in some PDers, some amount of dopamine release may be activated, especially immediately following the surgery. For the long run, the implants elevate adrenaline levels – not dopamine.

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.
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 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

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.
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.

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, PDers’ 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 were 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 PDers 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 PDers 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 PDers, 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 PDers 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.]¹

“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,¹

¹ This edition of the book was being posted, one chapter at a time, on the website of the Parkinson’s Recovery Project, pdrecovery.org. Chapters were posted as quickly as they were being written. At the time this reader wrote to me, in 2005, chapter 21 had just been posted.
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

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.
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 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 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 discusses the new western medicine realization that some people do develop Parkinson’s-like symptoms very quickly in response to stress. Because these people can recover quickly if the stress is healed, these people are not considered to have idiopathic Parkinson’s disease. The new name for this manifestation of symptoms is “psychogenic parkinsonism.” However, we have seen that, if these people are incorrectly told by their neurologists that, on the basis of their rapidly developing symptoms, they have incurable idiopathic Parkinson’s disease, they quickly go into a downhill

---

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 is has for the subject of Parkinson’s disease.
slide. If they are told that they have a passing condition, psychogenic parkinsonism, and that the condition responds to counseling or stress relief, they can sometimes recover easily.

**Psychogenic parkinsonism**

Psychogenic (caused by the mind) parkinsonism is the name given to a movement disorder that in many ways resembles idiopathic Parkinson’s but which is *not* accompanied by dopamine receptor deficiency as determined by PET and SPECT scans.

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. Tests of dopamine transporters (PET, SPECT scans) are negative [do not suggest Parkinson’s] in these cases and if caught early they can be treated to complete resolution in many cases. They are not as rare as one may think!”

I know that these cases are not rare. I have seen some. Here is the story of Roma, who rapidly developed psychogenic parkinsonism.

**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 while *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 fascinating answers.
Her tremor was the worst when she was eating. While this by itself is not unusual for a person with Parkinsons, note this: she never tremored when she was eating desserts.

I had her do several experiments. She had meals with no dessert; she had dessert-only meals. She had dessert first and dinner after, she had dessert in the middle of the meal. She had extra sandwiches in the afternoon. She had dessert-only in the afternoon. None of these variations made any difference: she tremored with meals, she didn’t tremor with dessert.

I was baffled and asked her what she thought it was all about. She laughed and said, “Dessert doesn’t matter.”

This intrigued me. I asked her why non-dessert meals “mattered.”

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 tremoring 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 that 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 tremoring. 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.

Roma’s problem was beginning to make sense.

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 falling. 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 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.

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: fully fledged psychogenic parkinsonism.

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 mastered it, she was once again, in her words, “flying high.”

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.

Wrapping up this chapter on placebos, I wish to emphasize one point. Placebos do not allow PDers to move simply because PDers imagine that they are able to move. The placebo studies that measure actual brain neurochemistry have found that PDers have actual shifts in brain chemistry – an increase in dopamine release – in response to placebos. In response to positive placebos, PDers release dopamine. The dopamine change is measurable.

In other words, PDers do have sufficient dopamine to operate their mental and motor systems. What PDers 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.¹

The next chapter will expand on this concept of inhibited dopamine release. Dopamine release is utterly, completely dependent on expectation and feelings. 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

¹ Early in my research, I stumbled across a study that said that the substantia nigra cells in Parkinson’s disease did, in fact, resemble embryonic, undifferentiated, cells. I could kick myself for losing the citations for that study. I am not a particularly keen scholar; I have not carefully filed away all of the hundreds, maybe thousands, of articles that have crossed my desk since I started this study. I am more interested in finding an effective treatment for Parkinson’s disease than I am in giving those footnotes that are beloved by academia. Still, in this particular case, I wish I had made a note of the source of the undifferentiated-cell study.
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 interpret the sensory experience of having a body as joyful 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 ask his heart to call for dopamine release in the substantia nigra area. Though he once again has the capability to release dopamine, he will remain locked in dopamine deficiency and dopamine deficient behaviors. Although his body may be straighter, his face more symmetrical, and his other Stomach channel symptoms completely gone, he may still have difficulty with movement initiation, slowness of movement, and tremor.
“Fido circled his master’s grave three times and then lay down, never to move again.”

- American folklore

CHAPTER ELEVEN

DOPAMINE RELEASE: PDERS AND DOGS

This chapter will continue with the issue of expectation-dependant dopamine release.

Due to misinformation, 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, a pervasive joy that flows in spite of whatever outrageous slings and arrows may happen to be flying at any given moment.

Joie de vivre

The electrical signals that initiate dopamine release are generated in response to a positive sense of self-awareness, positive attitude and feeling – what the French call joie de vivre, the joy of being alive. This thrill of self-awareness – a glad-to-be-alive thrill that courses through the heart whether a person is sad or happy, calm or excited, young and vigorous or hoping to see one more sunrise while lying on the deathbed – is the trigger that releases dopamine. Dopamine release then sets off the chain reaction of dopamine-based motor and mental functions.

Unless this “sheer joy of being” is operating in the emotional background, there is no way to access dopamine, no matter how much dopamine is in storage.

Actually, as you will read later, 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 only address this feeling as it relates to dopamine release.

FAMILIAR EXAMPLES OF NEUROTRANSMITTER INHIBITION

To start with, I will use an example that doesn’t even involve dopamine.

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.

---

1 I have asked around amongst my fellow English speakers, and so far as we can figure, there is no word or phrase in the English language that conveys the sense of “joie de vivre.” I also seem to recall that Parkinson’s disease is far more common in England than in France.
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 it’s 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.

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 became 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.
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 tremoring. To counter this self-serving loop-type thinking, I’m including these very familiar examples to show that animals, including humans can still 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.\footnote{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.}

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 not inhibit its ability to initiate movement. The loss of a leg will not inhibit the release of dopamine.\footnote{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}
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 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 PDers 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 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.

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.
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 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 PDers, 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 PDers, like Iris the dog, still have detectable structural problems. However, the PDers 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 PDers 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 PDers: 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.

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 his 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 PDeors 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 deceased 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 unocccluded by ego.)

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
**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 PDers 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.

**Review**

As mentioned previously, the presence of an unhealed foot injury is the one commonality in people with Parkinson’s disease. But even more important, in terms of dopamine release, are the emotional reasons behind the failure to heal: life-threatening fear; shame; or guilt. Sometimes these worries are long forgotten, very often childish and seemingly insignificant when seen in the light of “adult” worries. However, at the time of injury, these worries loomed large.

While the unhealed foot appears to be a commonality, the presence of other mental blockages and mind-body disassociations, and the intensity of these blockages and disassociations, is a highly variable factor. Some PDers have almost no mental resistance to healing even though healing requires exposure to the pain of the injury and the internal admission that the injury did, in fact, occur and was painful. Other PDers lead lives that have been almost entirely shaped by a fear/denial mindset. Most PDers are somewhere in between.
The emotional-cause portion of my overall cause-of-Parkinson’s hypothesis is based in part on thousands of hours of interviews with PDers, life stories of PDers, and the unexpected floods of emotions that gush through the bosoms of PDers during recovery – emotions which remain accessible after recovery is complete.

The emotional portion of the hypothesis also helps to explain the mechanism by which the feet were unable to heal from their injuries.

The emotional portion also explains the variable difficulties in any given PDer’s movement initiation. The weakness and movement initiation problems of Parkinson’s, problems that come and go in response to mood, weather, and social stress, can only be explained by a solid understanding of the manner in which dopamine release is “authorized” by the feelings. This subject will be addressed in enormous detail in later chapters. I will merely mention here that the difficulty in movement initiation that is seen in Parkinson’s, a problem that varies from hour to hour and day to day, a difficulty which is lifted quickly, though usually only temporarily, in response to almost any convincing placebo treatment, seems to correspond exactly to the guarded, but always slightly variable, heart-stance of the PDer.

With regard to the structural body problems that develop, over the decades, in the wake of the foot injury, those problems do not come and go in response to mood or emotion. However, with regard to the weakness, depression, and movement initiation portion of the illness, the PDer nearly always moves exactly as well or as poorly as he expects he will.

As for the tremor, the location of tremor (until it becomes nearly body-wide) is related to structural problems. The fluctuations (when, for how long, and with what amplitude) in tremor are usually related to self-consciousness (fear) and expectations.

A person who is an unwitting perpetuator of a dissociation response may be locked into a mental posture of underlying suspicion or negativity. We have seen that PDers who have, despite this inherent posture, worked to cultivate an underlying optimism, a faith in the ultimate integrity of the universal fairness, the laws of cause and effect – even as they relate to his own difficulties in life – will have a much easier time recovering from Parkinson’s disease.

Oppositely, we have seen that those people who take active pride in their wariness and negativity have a difficult time with recovery. Not only because it is difficult to heal their injuries, but because they are not able to believe that it is actually possible that they might be doing better. After partially recovery, they are able to manifest symptoms of full-blown Parkinson’s disease any time their negativity gets the better of them.

If a PDer is willing to examine his own mental outlook and work to consciously override any underlying negativity, he may be able to greatly enhance his speed of recovery and his own ability to maintain dopamine release. This concept is so crucial to recovery that the next chapter will be devoted to still more examples and explanations.

These chapters on the expectation-dependency of dopamine release have interrupted the chronology of the research project. I will be getting back to the chronology soon. But first, I will
share some pertinent information about the heart that was becoming available from western researchers during this time, around the beginning of the 21st century.
“Pay no more attention to the mind than you would to the ravings of an idiot”

- St. Teresa of Avila

CHAPTER TWELVE

MATTERS OF THE HEART

As part of our ongoing research, we had been keeping up with the new information in the field of neurocardiology. This new research had direct implications for our research on PDers. 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

As you may have noticed, I’ve been using the word “heart” quite a bit, and in a manner that suggests something other than the heart’s role in pumping the blood around.

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

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.
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 brain-based thought patterns. The heart feelings are formed by the heart’s electromagnetic resonance with outer and inner experiences. These heart feelings are then communicated to various parts of the brain via the heart-nerves.

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.

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 refuse to 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: brain instructions are subordinate and can be overridden by the heart. In PDers, we find that this excellent chain of command system has been over-rulled… by the brain. It is this brain, the brain that has usurped the leadership role of the heart, 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 (atriol neuratie 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 Bihr, “Un air de déjà-entendu…” National Geographic France, août 2003, p. 19.
The processes involved here begin with the original heart experience, the heart feelings, at the time the music 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. 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

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 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.¹

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).²

**HEART BRAIN ENTRAINMENT**

When a person feels content or calm, his brain-wave patterns are entrained with his heart-rate-variability patterns.³ 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.

¹ A recovered PDer told me that, as a young woman, she often went to music concerts. Her best friend once asked her, “Why do you come to the concert and then spend all your time looking around at what everyone is doing? Why don’t you just sit back and enjoy the music once in a while?” The ex-PDer told me, “I had no idea what she was talking about.”

² “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.

³ 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.)
When the fear is over, the brain’s wave patterns can again become entrained with the heart’s wave patterns.¹

**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, to some extent, guided by the heart.²

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.³

¹ *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 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.”

² 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-nerves continue to send information about the degree of problem, and the extent to which thoughts become adrenaline-dominant.

³ Looking ahead for a moment, note that, when people consciously shut off their hearts to prevent awareness of a negative experience, they inadvertently also shut themselves off to positive experiences.
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.

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. A person resumes, via dopamine, the ability to playfully imagine and visualize, and to anticipate purely happy outcomes. The ability to feel physical and emotional input regarding one’s own sensory experiences, either negative or positive, a heart-based ability, returns.

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-nerves’ 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.


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, repeatedly, that their hearts are blissfully incapable of any sensory feeling, and then pretend, momentarily, that the heart can feel. When they imagine that they have a feeling heart, one that is connected to their brain, they often feel, sometimes instantaneously, the joy and contentment that has been long absent from their lives.

Therefore, we suspect that decisions made by the brain, and not merely a condition of calmness in the heart, 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 dissociation response 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.

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 will to live decreases, the capacity for life also declines: the levels of both courage and joy diminish. When the amount of heart nerve 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. In a fearless person, one who has utterly surrendered his life over to the Goodness 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 allow their mind-led brains 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.

According to Asian medical theory, the Heart is the source of joy. 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.

Keep in mind that there are two problematic facets of the mental/emotional blockage of Parkinson’s. The first, the inability of the heart and brain to become entrained, 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. 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
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. Anxiety is the name of this condition, in which fear dominates the mind but the body is not able to mount an active, dynamic 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 neurotransmitter or 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.

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.
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 dopamine triggers more dopamine and a tilt towards the parasympathetic nervous system’s connection to the brain.¹

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, 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 heart resonance are very nearly the same. The former is more purely energetic, the other is the more physical manifestation of the joy energy.

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. The resonating heart is the 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 nigra and other parts of the brain, are merely satellite supplies of dopamine. They are activated, dopamine is released into various parts of the brain, when the heart instructs the brain to respond to sensory events with conscious 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 dopamine is only diminished when a person ceases to feel the sheer joy of being alive. 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

¹ 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.
for death. In PDers, heart SPECT scans show that dopamine receptor activity is significantly diminished. PDers have trouble feeling. We might say that PDers are getting ready to die.\footnote{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, some 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? No one is 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.}

DEFINING “FEELING”

To further explain Asian medical theory, Parkinson’s disease, and the heart-mind relationship, I must first define “feeling.” Unfortunately, in English language this word has a wide array of meanings. To prevent confusion, I need to use a narrow definition of the word “feeling.”

Herein, “feeling” refers to sensory and heart-wave input to the brain. For example, input from the nerves of smell, taste, vision, hearing, or touch brings information to the brain. This input is felt. The five types of sensory nerves impart feelings to the brain.

The sixth type of feeling 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 sense of touch can recognize the feel of many types of surfaces, such as rough, smooth, and silky. The “sense” of emotion can recognize the feel of compassion, gratitude, contentment, joy and intuitive knowledge and “hunches.”

When I speak of feeling, I am referring to these 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.

\footnote{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, some 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? No one is 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.}
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

I am proposing, based on Asian medicine principles, Vedic teachings, and our experiences with PDers during recovery, the following: in times of safety, the heart-nerve information is the primary determinant of whether the incoming information is perceived as good (resonant) or not, and to what degree. If the brain is in sync with the heart, the vagus nerve signals from the heart influence the way that the brain interprets and acts on all other incoming sensory information at that moment. 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 summary, in times of safety, the heart-nerves tell the brain how to interpret feelings.

Changes in “feeling” capability when heart and brain waves disconnect

If, due to the perception of danger, the brain disrupts the resonance of heart and brain wave patterns and sends signals to and from the heart using, predominantly, the spinal nerves (instead of the vagus nerves), the brain then interprets sensory information using adrenaline-based thinking. This type of thinking is extremely fast. It is very efficient. Of more concern to the person with Parkinson’s, adrenaline-based thinking does not register or involve the full spectrum of feeling or emotion, nor does it allow for the full spectrum of imagination or visualization.

As demonstrated in the ravening lion example, feelings of physical or emotional pain get in the way when a person is in danger. These distracting types of sensory perception are not accessible when adrenaline-based thinking is dominant: when the heart and brain waves patterns are not connected. As noted already, the areas of the brain that allow for playful imagination, visualization, and creating positive outcome scenarios are not accessible when adrenaline-based thinking is ongoing. In times of high danger, a person thinks. He doesn’t feel.

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).

---

1 Post Traumatic Stress Disorder is related to this phenomenon, in which a terrible fear links up with more and more brain regions until the entire brain is linked to fear.
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. When the sea lions surrendered their will and 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 had projected to her as they basked in the sun, a swelling in the chest from the sheer joy-of-being that she shared with them as they slapped fins and roared at each other.

“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 feelings.

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.

Being redundant yet again, let me say that, 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 heart response, 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

---

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.
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, after living decades, maybe most of his life, without having been able to behold these imaginings.

After recovery, a PDer responds to sensory experiences as if his brain wave patterns were entrained with his heart wave pattern. Therefore, we conclude that, while having Parkinson’s (and for decades prior), his brain wave patterns were not entrained with his heart wave patterns.

Emotionalism

Continuing the definition of “feeling,” I am not using the word “feeling” to refer to emotionalism. Emotionalism begins in the mind – it does not originate directly from sensory input. 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. All of these conditions begin with the ego, and combine with thoughts of satisfaction or dissatisfaction of one sort or another. These are not feelings, per se. They are not input from the six senses, they are ego-based interpretations of the brain’s information: the results of mental manipulations.

Emotionalism can be related to 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 feeling. Emotionalism is ego and thought habits responding to feeling, responding to sensory information or to a train of thought.

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 states, or feelings, effect 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. He rarely, if ever, shuts down his restless or anxious inner monologue.
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.

## 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.
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 an 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.

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.

Playing the violin is an activity that uses habits of highly complex, highly integrated movements. The following example suggests that the violinist 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.

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 useta 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
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 sensations, 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 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 feeling – this is the 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 motions of the body and with the brain’s incoming sensations from the body.

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?!”

_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 subordinate to it. Attunement with Divine mind can conquer any material problem because all of the universe, all of creation, is a construct of that Mind._

_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 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._
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 or Tai Chi Chuan. This is because they were mentally focused on the positions and competency of their 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 resonance with the wave patterns in the brain that are created from the sensory input, from the feelings of one’s own body moving.

If the brain waves are in sync with the heart waves, the heart feeling amplifies as it resonates with the brain as the brain enjoys 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; they move in whatever manner 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 PDers when the time comes to re-open the heart.

Some PDers are stunned by, if not 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 ease into this thought, I want to share some ancient Vedic theories.²

---

¹ Looking ahead, when we get these PDers to stop pretending that their heart is small, dark, or whatever dismal construct they have invented, and pretend instead that their heart is normal, 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 Chi, or other “joy-inducing” modalities.

² I am often asked how we came up with our extremely simple treatment for turning off the sympathetic system. The treatment was 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 holds one of the keys to the very simple treatment that we eventually developed.
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 the area from which the vagus nerve emerges). Next, when energy flows from the midbrain 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 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 (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,

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 predominantly 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.
because he can still feel, perceive, the vibrations of color and sound. Even on the causal plane, the realm of pure thought, an individual has self-awareness because he still feels a response to different thoughts (expansive feelings if the thoughts are attuned with Right, painful constrictive 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 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, the author declared synonymity between Chit (Universal self-awareness and feeling, The Universal Heart, and Universal Love. Feeling, 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, as you will read in a later footnote, India has the lowest rate of Parkinson’s per population in the world. England – which uses 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.
**Dissociation inhibits a person’s ability to feel**

Problems with partial recovery occur when a PDer’s unhealed injury begins to heal but he 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 crisis portion of a life-threatening event is over and done with, a person can remain locked into the dissociative mode.

A fleeting dissociation response is supposed to last for about ten minutes in humans. 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 has set in motion a retained dissociation response, his body will behave as if it is trying 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 Eastern medicine, the heart is considered the most important organ. The heart is referred to as the King or as the home of Joy, Divine Mother, or Love. The brain is merely one facet of 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. These people have been misled. In order to not be jerked around by one’s feelings, one 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. Suppressed feelings 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, one must conquer (not be dominated by) the illusion of feelings. To do this, one must recognize feelings 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

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 Paramahansa Yoganada, in his Autobiography of a Yogi, said it more succinctly: “Truths suppressed lead disconcertingly to a host of errors.”
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 heart-brain wave entrainment and the corresponding relationship between adrenaline-based motor function and dopamine-based motor function.

---

¹ I will share an example of dealing with feelings of foot injury that demonstrates the correct way to deal with physical pain.

Paramahansa Yogananda was outdoors, working with yoga students on a construction project, when, accidentally, a large cement cylinder was 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 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.
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. For those readers familiar with Chinese medical theory, this problem is a variant on the ancient syndrome of Heart and Kidney (mind) Not Communicating.

However, brief though it is, this introduction gives enough information to explain the thinking behind the experiment presented in the next chapter.

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 PDers are maintaining a severe dissociation response that prevents access to their feelings. Some PDers 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.
"How long, how long, in Infinite Pursuit
Of This and That endeavour and dispute? “

The Rubaiyat of Omar Khayyam

CHAPTER THIRTEEN

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 a monthly visit. He
was once again working on his juggling, exercising several hours a day, and very much enjoying
his retirement. 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.

*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 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.

Joy, a feeling of joie de vivre, the sheer joy of being alive, triggers the release of dopamine. Dopamine does not make people joyful. Being joyful allows the release of 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 is released in response to joy, or the imminent expectation of joy-based feelings.¹

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

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 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 one’s underlying joy.
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 a recent autopsy-based study of PDers. 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

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
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.)

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” 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.
exam. As the footnote below will explain in greater detail, this is only a sort of “negative proof,” but still, it was encouraging.¹

Even if the partially recovered PDer did not completely recover, we could find comfort in this thought. We began to wonder if, possibly, our findings might provide doctors with a more accurate way to correctly diagnose idiopathic Parkinson’s disease.

**The Parkinson’s Personality**

In 2003 we were still unable to get to the underlying source of the partially recovered PDers’ inability to visualize or perceive large portions of their own bodies, and particularly their feet. Western doctors refer to this type of mental problem as mind-body disassociation. In our PD patients, this foot disassociation turned out to be a mere subset of a much larger problem of mental/emotional blockage.

We weren’t sure what we would find at the core of what we were calling the “mental/emotional blockage,” but we suspected that the famous “Parkinson’s Personality” was related to this underlying problem.

We suspected a mental/emotional component from the early days of our work. However, it was not until 2003, after we stopped working with medicated patients – who often had mental problems because of the drugs – that we could concentrate more closely on those patients who seemed to be stuck in a condition of “partly recovered.” Only then, after working solely with unmedicated PDers, did we begin to fully understand the significance of the mental/emotional stance of so many of our PD patients.

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
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. We did know, at least, that the personality was not related to dopamine levels, per se.

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 was 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 own research suggests that the personality is based on a rare combination of adrenaline driven behaviors and compartmentalized denial of some physical and emotional feeling. Use of brain

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.
altering drugs such as L-dopa does not necessarily turn off adrenaline-based mental habits, nor does it restore feeling if the dissociation is still in place.

**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 PDers: 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 PDers, it appears (based on recovery symptoms) that the feel-no-pain attitude was initiated to deal with the foot injury. Other PDers 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 PDers, 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

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 situations. For example, consider the case of the unmedicated PDer who is absolutely paralyzed except when an easel and paints are set up in front of him. (Oliver Sachs, MD, *World Parkinson Congress Journal: Creativity and Parkinson’s*, 2006, p. 1.) 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 PDers 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 PDers, remained free from the growing emotional guardedness. Therefore, in these rare PDers who have 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 PDers 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.
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.\footnote{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.}

This astonishing mental achievement requires an enormous amount of emotional and mental self-control. PDers are notoriously highly intelligent. They are also notoriously unable to be flighty, irresponsible, or flamboyant with their deeper emotions.

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 PDers 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 PDers 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.\footnote{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.}

But then, after spending more than a year pondering PDers’ difficulty in imagining light in inured 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 PDers about it.

An imaginary separation of physical body and energy

Not only was it hard for most of our PDers 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 PDers, 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 \textit{much} more of a threat than the mere inanimate light that we’d been asking them to imagine.

Many of our PDers 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.

**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 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.

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.

**Not quite connected**

While most of my PDers’ minds do not go to quite the extreme of inserting their self-images in cages when imagining their bodies being filled with light, it turns out that most of them do find it easier for them to imagine bodies made of light that are not connected or are not fully connected to their physical bodies. Most often, the image that they conjure up is floating nearby. Sometimes it is floating just a few inches above the physical body, other times it is behind, in front, or somewhere in the vicinity of the physical form. Sometimes, part of the light-body is inside the physical body but one or more “light-limbs” are sticking out from the physical form.

For example, one PDer could imagine a version of his body filled with light, and this vision matched up with his physical body – except for his arms. The arms on his body-of-light were sticking straight up above his head. And he couldn’t even dare to imagine bringing them down into his physical arms. He was also, despite many indications of recovery in his legs, rapidly losing the use of his arms.

**Astral bodies**

Before I go any further, I need to make one thing perfectly clear. These patients are not having visions of their astral bodies. The astral (light-wave, electrical and electromagnetic power sources that hold the atoms together and drive the life forces) body of a person is intrinsically
integrated with and is the cause of the physical body. Some people imagine that their astral body is some sort of ghost that can come and go from their body whenever they sleep or relax deeply. This is bad science fiction/superstition.

The astral body is the energetic backdrop of the tangible body. The various electrical forces, electromagnetic waves, lights condensing into atoms, thoughts condensing into brain waves and brain waves changing into thoughts, are all energetic (and usually invisible) parts of the body.\(^1\)

The physical body is a tangible, solidified manifestation of a portion of the energy that forms our atoms and our chemical connections. The non-chemical energy in the body, if perceived as pure light, sound, and electromagnetic forces, is known as the astral, or energetic, body. The living physical body is a material manifestation held together by the astral body. The astral body does not leave the physical body until death.\(^2\) At death, the atoms stay behind and eventually break apart. The electromagnetic properties and light that make up the non-material parts of a person, the thought processes and the energy behind the scenes, do not break up.

When PDers tell me that, in order to imagine themselves with a body full of light, that light-body or some part of that light-body must be located outside of the actual physical body, this is not a statement of astral detachment. This is a statement that the mind of the PDer refuses to think of his own body as capable of being full of light, and more importantly, refuses to think of light or energy in his physical body.

The problem lies in the mind of the PDer; this is not a problem in which the soul is disconnected from the body. It is, however, a problem in which the emotions and self-awareness have been disconnected from the body or some portion of the body.

This blockage is, again, a mental construct. Despite the strong insistence from a few PDers 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.

---

\(^1\) “If our senses conveyed the whole truth to us, we would see the earth as rivers and glaciers of electrons, each speck of dust as a rolling mass of light.” Paramahansa Yogananda, from a desk calendar titled Reflections, 2001.

\(^2\) As always, there are exceptions. Many great saints and highly advanced spiritual adepts are able to separate their consciousness and underlying energetic form (their astral bodies) from the physical form. In cases like this, when the energetics of the body separate from the physical form, the physical body becomes motionless, and stays in a state of breathless “suspended animation” until the astral form and the physical form are reunited. As an exception even to the exceptional, great avatars such as Jesus, the Buddha, Krishna, and others are able to make their physical body go through the motions of breathing and moving even while their energetic body – the energy that runs the cosmos – is engaged in multiple activities in multiple locations.
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

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?

Same with treating 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 soppy 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.
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.

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

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.
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, I suppose. 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 get sullen or resentful when they
realize that their mental self-image and their physical self are 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.

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, and I
want them to know that these mental blocks could, in some cases, be 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.

We did eventually discover an effective technique for healing these disassociations. But
not until we realized how PDers had convinced themselves that having a body filled with light
would put their lives on the line.

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 tremoring 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 mind-body disassociation. I know I’m being 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 evidently formed complete mental disassociations from their physical bodies, to the point of having an active alter-ego. 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.

Honoria 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 Honoria 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 Honoria felt her chin start to quiver, her mother said, “I do hope you are not going to make a scene about this.”

Honoria 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. Honoria had been a vibrant, champion tennis player and lover of opera, a mother of three and beloved by all. Honoria died in her late 80s. At Honoria’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.”
Sharing my amazement

When I first heard about Honoria’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 PDers. 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 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 PDers 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.

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.
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 PDers’ mind-body disassociations, we still had, as yet, no consistent method to help PDers 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.

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 the 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 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 and the inability to imagine one’s body being full of light 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.

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...”
CHAPTER FOURTEEN

THE CASE OF THE MISSING HEART

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 PDers to visualize any perceived problem as being exaggerated and far worse than it actually is, developed out of our discovery that PDers, 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 PDers 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.

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 turns out that, in the preliminary skirmish with this type of mental-disassociation problem, it doesn’t really matter whether one imagines the problem getting worse or getting better. The great thing is, in the beginning, to be able to turn the previously reluctant, even disconnected, 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

---

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 PDers can easily, cheerfully, gain access to forbidden body parts by pretending to mutilate them. So we often started PDers down the road to imagination by letting them create horrors on themselves. Spouses and practitioners were appalled, but the PDers 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. Many PDers 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 PDers make the first step in turning their hearts back on.
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. 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 PDer's 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 PDer's 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 differed 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 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

---

1 Losing interest in the exercise usually signifies that the problem has become, to the imagination, less problematic.
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.

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.

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, the doctors didn’t know about that fact until it made the national headlines.

223
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 overactive 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.

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 tell a person exactly what to do with the problem. I always use the same words that the PDer uses and 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 obvious 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 imaging 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 some 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 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 a person who is in
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.

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 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.
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, 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.


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 this person, twenty minutes earlier, said 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? 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 the “wall” had been the impediment 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. 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 good, 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.
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

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 more calm, and she could see how her recovery was accelerating. I left her on what I thought was a high note.

¹ This technique and other helpful attitude-changing techniques are included in the appendices.

² I used to be astounded at most PDers’ disinterest in doing the techniques that I share in this book and in my classes – even those PDers who are certain that this program is the answer to their problems. Sometimes PDers come to our program from a long way away, maybe even from across the sea. 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.
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 shrunk 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 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 PDers, 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. (Continued on next page.)
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 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 poking 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

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, many similar incidents had occurred, 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 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.
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 lightening-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. A consciously-induced dissociation response 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. The dissociation response remains in place. Over time, it incorporates more and more brain arenas into its heart-numbing mode.

**Overseas corroboration**

Thinking that I’d discovered The Answer, 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 PDers 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 PDers. Their responses were, for the most part, extremely gratifying. Chris also invented variations on the technique to fit the various PDers.

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, “unsprung 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 PDers. 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 PDers.

**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.

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.

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.
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 brian 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 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. 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.

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. Professional musicians, 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 even if they dissociated from their foot injuries. This confirmed out 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, that was the biggest problem of all: 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?”

---

1 For example, one PDer complained that he couldn’t get his mind off his anxieties. I asked if he enjoyed listening to music; did music calm him down? He replied that, years ago, he had loved to listen to music. Now he found that background music was annoying. I asked him if background music was a problem because it distracted him, preventing him from being able to focus on his anxieties. He replied, “That’s it exactly. I can’t stay focused on my anxieties if there’s noise in the background.” Although his reply makes it seems as if he dislikes music because he is choosing to cultivate anxiety, this is not the case; his automatic interpretation of music and sound as being a distraction from the very important job of listening for danger or listening to worried thoughts is actually a normal part of the sympathetic nervous system response to potential danger. More on this later.
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.

In the greatest sense, the body cannot truly heal, cannot work as an integrated whole, if a person cannot feel his own body. (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.)

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 extended variations on the
dissociation response, understood 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 them.

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.

Another year passed before we came up with a technique that goes right to the core of the heart-mind disconnect. We actually found a simple method for turning off the dissociation response. We were helped along the way by studying the latest western findings on the heart-brain relationship. The next chapter shares our discovery of a most crucial recovery technique.
“Here’s a 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?”
“(The answer: Stop pretending!)”
- as told by a friend’s nine year-old baby sitter

CHAPTER FIFTEEN

TURNING OFF THE DISSOCIATION RESPONSE

After a year of partially recovered PDers experiencing short-term benefits from “connecting the heart and brain,” I grudgingly admitted that this exercise and its dozens of variations would never give anything but temporary benefit. What I did not yet understand were the “rules” for dissociation. The key rule for dissociation, a “prepare to die” condition, is this: the heart should be shut down as much as possible. Therefore, if dissociation was the dominant pattern in the brain, any attempts at keeping the heart open using visualization will necessarily be short term. There was no way partially recovered PDers were going to shut down this body-wide response without changing it at the source.

Dissociation in prey animals is usually activated under extreme stress, such as when the teeth of a predator cut through the skin, or when escape has become impossible and death is imminent. The dissociation response prepares an organism to die. It is a body-wide automatic physiological response that diminishes the level of heart activity, in addition to its other actions. A crucial part of this response is that, if the animal doesn’t die after all, the heart automatically turns itself back on to normal levels after a period of time. The period of time may have to do with how long it takes the body to neutralize the endorphins that are released during this response. Possibly it is only when the endorphins wear off that the heart resumes normal activity level. When endorphins are neutralized and their numbing qualities diminish, feeling is once again possible. It may be that this return to a condition of feeling is what kicks the heart back into a normal activity level.

The real problem with partially recovered PDers, the core problem, appears to be that they have learned to consciously prevent the heart from turning back on when the imminent crisis is over. By using their minds to induce a heart-inhibiting response and mentally instructing the response to not end, they have unwittingly locked themselves into a physiological condition from which there is no natural means of escape. To turn their hearts back on, a person in this suspended state of dissociation must use his brain to trigger his heart function. This is the reverse of the natural order of things. Ordinarily, the heart regulates brain function except during times of stress. In most forms of stress, the heart’s activity is increased, not decreased. In these more usual forms of stress, as soon as the stress is over, the heart activity drops back down to its normal level.

However, in a dissociation response, the heart function is suppressed. It cannot resume a normal level of activity until the danger is over. Partially recovered PDers behave as if they have trained their brains to always behave as if danger is ongoing. Therefore, they must maintain the lowered heart activity level. It is outside of the natural order of things for the heart to come back to a normal level of activity while the dissociation response is in effect.

Therefore, a partially recovered PDer who merely pretends to open, connect, or visualize his heart may experience a pleasant feeling in the short term. However, none of these games
induce actual heart-feeling. As long as the danger-habituated brain is in charge, any pleasant mood shift induced by the visualization games will end as soon as the person stops pretending. Based on our experiments, it appears that what is needed, to permanently turn the heart back up to a normal level, is for *feeling* to resume.

A very quick example may help explain what I am talking about. One partially recovered PD, Ron, had been working with us for more than a year. He was experiencing extreme fatigue and steadily worsening ability to initiate movement. He had been regularly working at his visualization practice and had gotten to the point where he could imagine his body full of light and could even imagine his body moving. He asked Chris, my colleague, if he had mastered the visualization exercises. Chris asked him how his body parts felt when they were filled with light. Ron was taken aback. “What did you say?”

“**I asked how you feel when you do the visualization.**”

“How do I *feel*?”

“Yeah, how does your body feel when it’s full of light?”

“*Feel*?”

“Yeah, how do you feel?”

“What word are you saying: *feel*?”

“Yeah, *feel*.”

“What do you mean, *feel*?”

“I mean, *feel*. How do you feel?”

“What do you mean, *feel*? What are you talking about?”

“I’m asking, how do you feel when you imagine your body full of light?”

“*Feel*?”

We could bring a partially recovered PDer to his imagination, but we still couldn’t make him feel instead of think.

**A new technique**

In January, 2007, I attended another week-long silent retreat. Once again, I prayed for answers, but got no response in my heart. However, a few weeks later, I heard myself instructing a PDer to perform a ridiculously easy mental exercise involving his heart. As I heard myself issuing the instructions, I was stunned at the extraordinary simplicity of the technique.

I told this PDer to imagine that his heart was in a steel box. I told him to silently affirm that, with the heart safely ensconced in an impermeable box, he could *feel* no sensory experiences. He could not *feel* any resonance between his heart and his sensory experiences. He *could* cognize his seeing, hearing, and tactile experiences, but all of that cognition must take place in the brain alone, with *no* heart involvement, *no* heart resonance.

He spent four minutes with his heart in an imaginary box, consciously experiencing through his mind alone all of the sensory input from the sights and sounds in the room. Then, I abruptly asked him to rip off the imaginary box and let his heart resonate with all of his surrounding sensory input. For several minutes, the PDer had to experience any and all sensory events via resonance in his *heart*, while ignoring any mental recognition of the sensory experiences.
Based on beneficial results (an unexpected ability to feel with his heart), I asked my other PD patients to do the same exercise.

Amazingly enough, most PDers did not struggle with this technique in the way that they had struggled with the others. Even those who had only minimal or no results with this exercise, in terms of a change in ability to feel, actually did their “homework;” they practiced the exercise at home, on their own. This was the first time that I had come up with an exercise that nearly all PDers were willing and able to do.

For the first time since we started using visualization exercises, it seemed that, in this exercise, the PDers could do an imagination-based technique and not have to struggle against their own inner warning system. Because we were asking them to start by shutting down the heart, a thing their brain completely supports, they found themselves able to do this exercise without mental warning lights flashing. Also, when the exaggerated heart-shut down is turned off and the heart momentarily resumes having feelings, the PDer is not doing anything that is specifically denied by the brain. After all, the dissociation response is supposed to turn off when feeling returns.

We had tried all sorts of methods to get partially recovered PDers to feel their own bodies, without success. But we had never before tried to trigger feeling by first spending some time inhibiting heart-feeling.

And when PDers did experience a fleeting moment of genuine heart feeling, they also experienced a shift in perception. This shift seemed to alter their perceptions into those experienced by people who are in parasympathetic mode. It seemed as if, by turning down the dissociation response by experiencing heart-feeling, they seemed to be able to experience, momentarily, an increase in parasympathetic brain process and a diminution of dissociation and numbness – in a manner that apparently did not defy the existing dissociation rules in the brain.\(^1\)

Also, and most importantly, the experience was uniformly pleasant.

Of course, in order to do this exercise, the PDer has to be far enough along with his visualization practice that he could imagine himself having a heart.

But for those PDers who had gotten to the point of being able to “see” an imaginary heart, this exercise seemed to open the door to heart-based feeling. As that door started to open, the PDer was usually able to notice a difference between perceptions that were experienced via heart resonance compared to perceptions that registered in the brain. At first, the difference might be small. However, the experience was consistently pleasant and non-threatening. The more the PDer practiced shutting his heart in a box and going numb, and then getting rid of the box and experiencing sensory events via the heart for a few moments, the better he became at being able to experience momentary, brief events of actual feeling and the more he wanted to do it.

It looked as if we had found a way to get PDers to start using their hearts again. This seemed like a crucial first step in getting PDers to step up the level of heart-nerve function. If PDers could stop being numb to their bodies, if they could stop playing dead, they might be able

\(^1\) The truly cagey reader will appreciate that even this exercise is, in fact originating in the mind. But because the post-visual portion of exercise involves experiencing feeling via the heart, the exercise does become a heart-based technique even though it starts in the brain.
to have enough heart-nerve function that they could start ordering the release of adrenaline and dopamine for motor function once again.

**THOUGHTS AND QUESTIONS THAT AROSE**

Scientists know that, in an emergency, a person switches over to full-on sympathetic mode; in terms of wave pattern entrainment, the heart and brain then act as if they were disconnected.

My new technique seemed to suggest that the reverse is also true: having a disconnected heart, even pretending to have a disconnected heart, could cause a person’s perceptions and behaviors to switch over to the sympathetic mode.

More questions arose: could a person pretend to have a disconnect heart indefinitely? Could the changes set in motion stay in place even if there was no ongoing emergency? Was this the mechanism that PDers had used to stay lodged in dissociative mode?

Another very basic question was this: *could* a person to switch over to perceptions and behaviors typical of the sympathetic mode but keep the heart nerve activity at a very low level? Before I started suspecting a dissociation response, this had been an important question for several years. For many years I had received flak from PDers who were adamant that they were not using adrenaline 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.

Part of the problem was the the general public and even many doctors incorrectly assume that a switch to adrenaline must necessarily cause a huge surge of adrenaline – a surge of out of control, fight-or-flight behavior.

Behaviorally, PDers had sympathetic nervous system thinking and wariness. But anatomical events such as heart rate and breathing in PDers seemed to occur at minimal levels. The general thinking about adrenaline is that nerve responses to danger always increase heart rate, blood pressure, and breathing rate. But PDers often have lower blood pressure, low heart rate, and low breathing rate. This type of low-level adrenaline use while in sympathetic mode is associated with dissociation, but dissociation is usually considered a very rare condition, and not something that can be induced mentally or sustained for the long term.

I too was questioning the combination of sympathetic perceptions and behaviors with the low-levels of acting-out behaviors associated with adrenaline that I saw in PDers. I have been studying medicine for most of my life, and until I read, in 2007, about the ability of some regularly-abused children to consciously induce a measurable dissociation response, I never knew that a dissociation response could be mentally induced.

Therefore, I wondered if mental symptoms typical of a sympathetic system response *together* with low breathing and heart rate could be consciously induced by the new heart-in-a-box technique, a technique that consisted of mentally pretending to have dissociation-type heart behavior.

And then another question arose. If we could mentally induce this condition, would the resulting behaviors – sympathetic-mode mindset and sympathetic-type sensory perception – be considered a good match for the Parkinson’s Personality? The Parkinson’s Personality’s most
defining characteristic is harm avoidance. Would mentally induced heart-shutdown create a condition of wariness that fit the description of harm avoidance?

Most important, could we prove that the heart-mind disconnect, the disconnection from feelings, might possibly be *causative* for Parkinson’s disease? Some doctors might want to say that the measurably diminished heart response observed in PDers was the result of PD, and not the cause.

The researchers who discovered the diminished action of heart-nerves in PDers were able to prove that this low level of heart function was not affected by use of L-dopa; they concluded that the heart-nerve problems were not dopamine related. However, many doctors still imagine Parkinson’s to be caused by inexplicable decrease of dopamine release in the brain.

Could we prove that the heart-mind disconnect might possibly *not* be the result of slowly encroaching Parkinson’s, but that the Parkinson’s was the results of decades of slowly encroaching heart-mind disconnect?

Although we had yet to prove it, we actually knew already that dissociation could be set in motion by pretending: many recovered PDers had suddenly, calmly, during recovery, made a statement to the effect of, “Oh. I remember when I decided to start doing this,” and then went on to describe how they had created some mental game that allowed them to pretend that they didn’t have feelings.

Very often, there had been a very good reason that they had started. But their wording clearly stated that they had *consciously* started doing it: it had not been something that had happened accidentally or inadvertently.

Given what we were learning about the heart-brain relationship, and given what we had seen in PDers (mental and emotional behaviors that suggested almost lifelong reliance on the sympathetic nervous system), and given what we had seen in some recovered PDers (heightened sense of joy, feelings, and body-awareness, *and* a memory of having decided to pretend to not have feelings), we wondered if there was any way we could prove that some PDers become locked into sympathetic mode, adrenaline mode, by pretending to be detached from their hearts, detached from their feelings.

If so, this might add weight to our finding that PDers had consciously set these patterns in motion. And this would be powerfully important: if PDers understood that they had started this mind-game consciously, they would also understand that they, and no one else, could put a stop to it. This might help encourage them to keep working away at the hard work of overcoming their mental/emotional blockage instead of hoping, as so many had, that we should somehow “fix” the problem with an acupuncture needle.

Thousands of hours of interviews with PDers led us to suspect that, after setting this heart closure in motion, they had then enjoyed, at least in the beginning, the sense of enhanced alertness and accelerated motor and mental function that occurs when adrenaline is the dominant neurotransmitter. Many even imagined that, by keeping heart-nerve responses at a low level, their resultant calmness and focus was a sign of spiritual superiority.¹

¹ Heart-brain disassociation is anathema to serene living. The amount and frequency of a person’s episodes of heart-brain disassociation are directly related to the extent that a person is living an ego-based, mind-based, fear-based life. Heart-brain disassociation is an indication of cowardice, not refined spirit.
Many PDers had further convinced themselves that normal, healthy behaviors that they could no longer do, such as visualization, imagination of positive outcomes, moving in an unself-conscious, imaginative manner, and even the assumption of universal goodness were bad things. Somewhere along the line, they had convinced themselves that the sympathetic-system behaviors that they were locked into, behaviors such as caution, cynicism, stoicism, fear of being judged, judging others, and the whole array of negative emotions, were good things.

Feeling joy was usually impossible: they convinced themselves that feeling happy was corny. Feeling wary was the only option they had: they convinced themselves that being always on the alert to trouble was a sign of wisdom. They might consciously want to feel joy. They might want to be idealistic and serene and genuinely content. However, these pleasant conditions are not allowable during a dissociation response.

Even if PDers had set this heart-mind disconnect in place consciously, long before the Parkinson’s began, they were no longer aware that they were still using it. When, in later life, they became conscious of being depressed or negative, they had no idea as to how to stop doing it. They may have done this, at first, during a life-threatening event, and never stopped doing it. They may have simply decided to do it, and found it easy and rewarding.

**The benefit of an intentional heart-brain disconnect**

Why would anyone want to create a mock state of constant emergency? The benefit of so pretending is this: when the sympathetic nervous system is dominant, a person does not experience his own physical and emotional pain. What I was really proposing here was that

The saint never needs to switch over to emergency mode. For example, when a saint sitting calmly in the woods suddenly sees a cobra gliding towards him, the saint’s understanding of the nature of all things allows him to joyfully appreciate the phenomenon of the snake. The saint’s ego does not leap to the fore, setting off alarms of snakebite and mortality. The saint, attuned with soul’s feeling, thus truly knowing himself as soul and knowing himself as immortal, does not allow his fear-based, delusion-based ego to interfere with his enjoyment of nature’s little drama. And lo! the snake does not bite the saint, even if the saint claps his hands together to attract the snake’s attention.

The saint never allows his brain to become dominant over his heart. Even a saint such as St. Thomas Aquinas (1225-1274 AD), a catholic saint who approached God, as many do, via the difficult path of Wisdom and discretion, experienced, as a result of his mental self-disciplines, a heart-opening attunement with God. He said afterwards, in reference to his *Summa Theologica* (the crowning glory and summary of all religious philosophy of the middle ages), that, “Everything I have written is as straw.” That is to say, all his literary work was worth nothing compared to his heart experience.

Whether one works on opening the heart directly or approaches this goal of open heart via discriminating wisdom, service, or the arts, the goal of Self-control and Self-awareness is a heart that is ever open and guiding one’s life and a brain that is ever the well-trained servant of the heart.

Because the saint never allows his brain to dominate his heart, he never enters into a mental condition of emergency, whatever the situation around him. The saint is calm, not because he slides into a condition of denial, but because, whatever the circumstance, his heart is always in charge of his brain. As for amount of heart, the saint is always striving to have his heart open to the largest extent: the greater the opening of the heart, the greater the experience of joy and bliss. True, a saint may then behave in a manner that provokes censure from the small-minded: Mother Teresa of Calcutta was criticized by cynics of every country. However, she was never worried by the snide remarks of those who accused her of self-aggrandizing motives when she loved and helped others.

In so many people with Parkinson’s, I see souls who are consciously trying to emulate the calm and wisdom of the saint by pursuing the exact opposite mental and emotional condition of a saint: many a PDer has a mind that is dominating the body and a heart that has been, in his imagination, shut down or highly restricted.
PDers, by pretending to not feel physical and emotional pain, had necessarily put themselves into this special form of the sympathetic mode whether they had intended to or not.

Controlling one’s amounts of neurotransmitter by controlling the size of the heart-nerve signals could prove advantageous for one who is trying to limit his exposure to pain. After all, high levels of adrenaline or dopamine can allow one to exhibit out-of-control behavior. Out-of-control behavior can lead to physical or emotional pain. By closing off the heart and eliminating the possibility of emotionally high-risk or out-of-control behavior, the person with minimal heart-nerve signals, with a closed-off heart, can live a life of relative safety from physical or emotional pain.¹

However, long-term pretending might possibly lead to long-term physiological changes. By pretending that the heart is closed, and thus inhibiting the amount of heart-nerve signal to the brain, the heart-nerves may, in fact, become somewhat dormant; heart-nerve activity is measurably decreased in PDers. When the amount of heart-nerve dormancy hits a critical level, it becomes increasingly difficult to muster the amount of heart-nerve signal needed for basic motor function.

**Could I construct an experiment to answer our questions about the dissociation response?**

Could I prove that a person could consciously set in motion the sympathetic nervous system, instituting conditions of mental wariness and mild negativity, and the inhibition of dopamine and the parasympathetic system, merely by pretending that his heart was closed off?

If this had been done willingly, it could be undone.

Taking into consideration all of the above, I devised an experiment to address as many of these questions as possible. Mainly, the experiment was designed to see if it was possible for a healthy person to create and then uncreate a condition that mimicked reliance on the sympathetic nervous system and a diminished level of heart-nerve activity. I needed to see if the condition so created would cause the person to have sensory perceptions that corresponded to those experienced in the sympathetic mode. I also needed to see if heart rate and breathing rate remained slow, as if the heart-nerve activity was at a low level.

---

¹ Curiously, many PDers love to engage in high-risk behaviors such as parachuting, roller skating in dense city traffic, and other physically dangerous activities. Some have told me that they can only feel alive when they are doing something really risky. The irony is that they have intentionally shut down their hearts to the point that they feel nearly dead. To enable themselves to feel alive, they then need to do physical activities that are genuinely dangerous. To override their self-instituted game of non-feeling, they must behave in such as way as to incite real terror in their hearts if they want to feel alive.

I had one PD patient who enjoyed going to really scary movies. If he became scared enough, he would feel electrical energy flowing in his body, all the way down to his feet. The rest of the time, he purposely never felt his body. He remembered shutting off recognition of his body when, as a teen, he developed his lifelong severe, untreatable back pain.

It has long been recognized that, in a real emergency, PDers can move perfectly normally: when their life is truly at risk, they stop pretending that they don’t care. Very likely, the PDers who engage in high-risk sports and activities have learned that these activities allow them to increase their heart-nerve signals for the short while that they are at risk. The brief feeling of truly being alive is such a relief from their default condition of pretending to be shut off that, evidently, they are gladly willing to take the risks.
A Little Experiment

I decided to conduct an experiment to see whether or not a person with a healthy heart-brain relationship could consciously shift himself into the perception behaviors of the sympathetic mode, and whether or not a person could consciously choose to limit the extent of his heart’s responses.

The technique would be the same as the exercise I had recently started using with PDers. In choosing subjects in the experiment, I first used people who do not have Parkinson’s (and never did). To assure that the subjects had an active heart-brain relationship, I chose musicians to perform the experiment.

Then, out of curiosity, I also ran this experiment on people who had recovered from Parkinson’s and on people who were stuck in partial recovery from Parkinson’s.

**The experimental technique**

In phase one of the experiment, the subject pretends that he has a steel box around his heart. He then says silently to himself, “I *cannot* experience my sensory perceptions [sight, hearing, taste, smell, touch] in my heart. I *do not* experience my sensory perceptions in my heart. I *notice* my sensory perceptions, but I do not feel them in my heart.”

In phase two of the experiment, the subject pretends that the steel box is gone. He silently says to himself, “I *can* experience my sensory perception in my heart. I *do* experience my sensory perceptions in my heart.”

Total duration: five minutes. Phase one lasts for four minutes and is immediately followed by phase two. Phase two lasts for one minute.

During the four minutes of phase one, the subject has to maintain this mental heart-in-a-box image and the affirmations. During this time he can walk around, look at things, listen to any environmental sounds, including music or speech, talk to others, and pretty much do whatever he wants.

He can also walk around and do whatever he wants during the one minute of phase two.

After the five total minutes of the experiment, the subject will be asked to share any observations whatsoever that he had about the experiment, including any observations about differences in sensory perception.

Unbeknownst to the subjects, they were also being observed to see if they manifested any symptoms of an adrenaline surge: increased breathing or signs of increased heart rate. I did not actually measure the heart rate, because people become self-conscious when their heart rate is being measured, thus increasing the heart rate. Instead, I observed the type and rate of breathing, and assumed that the heart rate should correspond.

I did some of these experiments long-distance, over the phone. If I wasn’t present during a subject’s experiment, I simply asked, afterwards, whether or not the subject had felt any increase in heart rate or breathing during the experiment.

**Our expectation**

Our underlying assumption was that this game of pretending should create a heart-brain disconnect in the subject. This would move his heart-nerves more towards the direction of the
sympathetic nerve and away from the vagus nerve - into a mild condition of adrenaline dominance.

Heart-brain entrainment is characterized by feelings of peace and joy. Heart-brain dis-entrainment is characterized by heightened alertness of wariness or unease. For purposes of evaluation, we would look for any feeling of wariness or negativity in response to phase one, or a definite shift over to joy in phase two.

Our thinking was this: if the subjects noticed a decrease in joy in phase one or an increase in joy in phase two, we might conclude that, possibly, they had created or increase a condition of heart-brain dis-entrainment and a concomitant switch over to sympathetic mode during phase one, and a heart-brain entrainment and a move towards to the vagus nerve (parasympathetic, dopamine-using system), away from the sympathetic nerve, in phase two.

The subjects’ style of sensory perception would also tell us whether or not he had been in sympathetic or parasympathetic mode during the experiment.

Another assumption had to do with the large surge of adrenaline that some western MDs still assume to be the typical adrenaline response. Animal research suggests that switching over to the sympathetic mode usually triggers heightened alertness, but does not necessarily trigger the huge physiological responses that MDs refer to as “fight or flight.” The fight or flight response actually only occurs during highly specific situations.

We proposed that even if this experiment caused people to switch over to feelings that were characteristic of the sympathetic nervous system, they would not have a large surge of adrenaline and the accompanying increase in heart rate and breathing. Because of the methodology of the experiment, which pretended that the heart itself was cut off, we proposed that the subjects would have controlled breathing, and possibly even a slowing down of the heart rate.

The results

The results of this study were uniformly gratifying.

First, there was no indication of increased heart rate or increased rate of breathing. Instead, just the opposite occurred. Possibly because the subjects were concentrating deeply on what they were seeing, hearing, and feeling, their breath became very slow and measured – as breath does when a person concentrates. If the heart rate can be assumed to correspond to the breathing rate, one might well assume that the heart rate was also slowed and steadied. If our notion that these people had switched over to the sympathetic mode was correct, this calm-breathing observation was nice proof that not all adrenaline-based responses are of the violent “fight or flight” nature.

Second, the subjects observations strongly suggested that the game of pretending did in fact shift their perceptions over to the mental conditions that prevail when adrenaline and the sympathetic mode is dominant. I will share some of the observations of the subjects.
Observations

The musicians

I called the first subject at his home and asked him if he would participate. He agreed, mentioning that, while his heart was in a box he was going to be washing the dishes and then making breakfast.

He called me back as soon as he was done. He reported, “With my heart in a box, the mundane became mechanical. I was washing the dishes. I felt like I was achieving something, which felt good. I didn’t mind it. Then my mother-in-law came over from next door; she’d just squeezed some oranges from her tree. Normally, I would have said ‘Thank you so much for bringing the juice,’ but today I only said ‘That OJ tasted good’ because I didn’t have any feelings. After the experiment was over, I went over and thanked her, and explained that I’d had a box around my heart.

“All I can say is afterwards, when I got rid of the steel box, I felt joy in doing the dishes, which is weird. I didn’t expect anything to happen, but there I was, singing a little song, feeling joy. I really felt more joy afterwards.”

I asked him if he’d minded putting his heart in a box. Had he had any resistance to the idea? He replied, “I didn’t mind. But it was hard to do. I had to repeat it out loud, verbally, like a mantra, ‘My heart’s in a box, my heart’s in a box,’ for the first two minutes.

“And when I was giving my wife a hug as she was heading out the door, I told her I was hugging her but that my heart wasn’t feeling it because my heart was in a steel box, and she said, ‘I really don’t like this experiment!’”

This person did not feel repulsed by holding his heart in a box, and in fact, he felt good about achieving the goal of washing the dishes. At mild levels of adrenaline, a person can be very focused and get a lot done. This subject’s responses suggest that he was doing exactly that.

Many people with Parkinson’s find great pleasure in achieving. In fact, many of them live for their achievements. People with Parkinson’s are not necessarily locked into an overtly negative attitude for their whole lives. However, their perceptions do tend increasingly to be skewed over time into perceptions that reflect the adrenaline mind-set.

The second musician observed that, “In phase one, I noticed sensory stuff more. My skin was more sensitive; I noticed that the stubble on my chin was more prickly and my clothes were more scratchy. I decided to see how it would feel holding my wife’s hand. It was slightly uncomfortable.

“In phase two I felt more comfortable. I was less sensitive. My wife’s hand felt better, holding it wasn’t uncomfortable at all.

Actually, I didn’t realize, during phase one, that anything was different. I thought I was just taking more accurate note of tactile sensations because I was trying to pay attention. It wasn’t until I did phase two that I realized that my perceptions of my sensations had actually been different than usual during phase one. I mean, it feels good to hold my wife’s hand. So only in retrospect I realized that all my sensations had been more uncomfortable, more negative, in phase one.

I personally found it fascination that this person, when shutting down his heart, was not aware that he had made a shift into negativity. He merely assumed that he was noticing things
more acutely. In fact, he was putting negative connotations on things that ordinarily did not bother him – but he didn’t perceive it that way until he reopened his heart. This was so similar to the way that people with Parkinson’s tend to assess everything, from thoughts to sensory input to the behavior of others, that I was astonished; I had assumed that PDers needed a lifetime of adrenaline-based thinking to cultivate their negative perceptions. But this subject had made the switch in a matter of moments.

Most PDers assume that they are very, very objective, very keen observers and rational thinkers. When I try to tell the ones that are exceptionally cynical or critical that their cherished objectivity may seem to an outsider like negativity, they assume that those outsiders are just excessively emotional and that they, the PDers, are being coolly rational. However, as this musician pointed out, in phase one he felt uncomfortable holding his wife’s hand and didn’t question it, although he knew perfectly well that he enjoys holding his wife’s hand. Instead, he “realized that he was uncomfortable holding his wife’s hand,” as if this act was, to a rational thinker, necessarily uncomfortable. At the time, with his heart closed off, the uncomfortable feeling seemed accurate, objectively correct. It was only went he went into phase two did he realize that his seemingly objective perceptions had been terribly skewed during phase one.

The third subject, a musician and a practicing Zen Buddhist, doing highly focused meditation for decades, told me that he couldn’t do the experiment. He reported that his first thoughts were, “This seems familiar. I’ve done this before. I really don’t like it. Why am I doing it now?” Next, he felt “Numb but not really numb. Like I had less feeling. Increased awareness. But not really more aware, but differently aware. More like wary. And like being in a tunnel. But I couldn’t continue the experiment for four minutes. I kept getting distracted. I did the experiment while sitting in a lecture hall. I welcomed distractions, and, when distracted by the person sitting next to me, I instantly snapped back into the feeling of open-heartedness – almost to a higher than normal degree. It was like when you’ve become really thirsty and then you get a drink of water.”

I pointed out to him that, considering his normal ability to be utterly focused, his unexpected susceptibility to distraction was possibly significant. I asked him to try again. He did, and reported the following: “

“[While I was pretending my heart was in a box] there was a rigidity and determination, but the determination felt less multi-dimensional than my usual sense of determination. It was thinner, less full, as though I were struggling beneath a veneer of determination. When I stopped [phase two], everything was way better than when I was doing the experiment [phase one]: colors, sounds, everything. It felt so good to get out of there.”

In summing up the responses of the musicians, these three subjects’ observations suggest that merely pretending and making a numbing affirmation had temporarily altered their perceptions.

The words they had used to describe phase one perceptions included “wary,” “mechanical,” and “uncomfortable.” These words correspond to the shift in perceptions that occur when using the sympathetic nervous system. Oppositely, their perceptions when they got rid of the box corresponded to perceptions that occur when the heart and brain are entrained and the heart is using the parasympathetic system: better, brighter, comfortable and joyful. These are characteristic of the perceptions that occur when dopamine is dominant.
I also found it interesting that each one of them had noticed a shift towards wariness with regard to different sensory functions. The first one noticed a change in motor function. The second one noticed a change in tactile observations. The third one observed a shift in visual observations and auditory disruptions. This selectivity fits nicely with our budding idea that, especially in the beginning, a consciously-induced dissociation response can be mentally compartmentalized: even when people are shutting down their hearts, they have choices as to what they are shutting out and choices as to what things they should be wary of.

**Recovered PDers**

Next, I did the same experiment with ex-PDers, people who had completely recovered from Parkinson’s.

The first recovered PDer reported, “I felt a pressure over my heart when there was a box there. It was like a hand pushing on my heart. I felt glad to get rid of it after four minutes. I did not enjoy having a box around my heart.”

This observation was not so dramatic as those of the musicians, but still suggested that simple pretending could create a physical sensation.

The next recovered PDer said “I walked around an outdoor shopping mall while doing it. I noticed that, when my heart was in the box, I was walking briskly, and that I was very mindful of the other people. I noticed that I was paying attention to what people might be doing, which directions they were walking, their body language as it might relate to me, though most likely no one was actually paying attention to me. I didn’t have as much sense of enjoyment as I usually feel when walking.

When I got rid of the box around my heart, I continued walking. I suddenly became aware of the gentle breeze. Maybe it hadn’t been there earlier. I also noticed the piped-in music for the first time. It had a country western theme. I found myself walking in time with the music, and moving more easily, swaying at the hip. Also, I stopped noticing the people around me in the same way. I didn’t care what they were doing because I was enjoying the music and the breeze and the feeling of walking to the music. Now when I saw other people, I assumed that they too were probably enjoying whatever they were doing. A woman walked by wearing a goofy hat. I thought that she must be enjoying herself, and that thought pleased me. I remembered that, when I’d had Parkinson’s, I thought that people wearing goofy or showy clothes must be shallow, if not downright stupid. I assumed that, because they were doing something that I wouldn’t do, they weren’t “good,” somehow. Now I wonder, who was I, to have been passing judgment on other people because of how they dressed?

I was really glad to get to get rid of the box around my heart.”

The third recovered PDer had no suspicions as to the purpose of this experiment. He had first come to see me in 1999 because, as he put it, “My wife says I should see you. She thinks there’s something wrong with me, but aside from my right foot dragging once in a while, there’s nothing wrong.”

A quick intake brought out that his handwriting was getting smaller. His leg dragging was getting worse, he was having trouble sleeping and that his movements, especially when playing softball, seemed sluggish. He was an active athlete. He dismissed all of the above symptoms as being normal consequences of aging. I noticed that his
voice was pinched off and that he carried his head too far forward and walked somewhat stiffly. His right arm did not swing as well as the left. When I mentioned these symptoms, he assured me that they were the normal processes of aging.

He was in his mid-forties. I examined his leg and found Qi running backwards along his right Stomach channel. His right foot was rigid. I asked about history of injury, and he said he’d had none. I did Yin Tui Na on his right foot. After the second session he told me that he’d had many ankle injuries. The next week during his Yin Tui Na treatment he told me that one of the ankle injuries had been quite severe.

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 previous injury. I did not discuss my Parkinson’s work with him. So far as I know, he never read any of my articles or books. His symptoms cleared up over the next few months. After that, I saw him now and again for flu symptoms or other, non-Parkinson’s related situations.

He came to my office in 2006 to ask if I could help with his psoriasis. His case was moderately severe; the psoriasis covered much of his torso and legs. The condition had started when he was twenty years old. I discovered that the psoriasis had appeared following a case of pneumonia that might have prevented him from competing in some sports event. He recovered from the pneumonia in record time, and was able to participate in his event. He had never associated the appearance of the psoriasis with the pneumonia. I told him that I didn’t know if I could help him, and that skin conditions are notoriously hard to cure. He was willing to come in once a month for a year or so to see if the psoriasis might respond to acupuncture and herbs.

Just after I had done the heart-in-a-box experiment with the musicians, he came in for his monthly psoriasis treatment. Without telling him why, I asked him to do this experiment. He did it, and 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.”

I asked if he had noticed any sensory shift.

“No, no awareness of sensory shift because I was trying to shut out sensations. When I stopped, it was great. I really enjoyed hearing and seeing everything [in phase two].”

I was so stunned at his recollection 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 feeling cut off. My jaw was hanging open. In terms of my research, this was like winning the lottery.

**Partially recovered PDers**

Finally, I tested three people who had partially recovered from Parkinson’s.
Hope, the first one, observed, “The first way seemed more normal. The second way [with no box around the heart], I immediately felt hungry.”

This was incredible. Hope is a tiny, tiny woman. She is not scrawny, and she works out twice a week at the gym. But she eats like a bird. She had told me that she never understood what it meant to feel hungry or to feel “full.” Until partway through recovery from Parkinson’s disease, she had always thought that “feeling full” had to do with having smelled enough of the food odors to be sated. She had never experienced the sensation of a full stomach or of her own moving intestines until her foot injury began to heal. She had never, ever, in her remembered past, experienced hunger. She had told me this before, when she first noticed the “disgusting” sensation of being able to feel food in her own stomach.

Hope continued telling me her observation about the experiment. “I never feel hunger. I just felt hunger! It was like a feeling in me!”

We repeated the experiment. This time when she got rid of the box around her heart, she said, “I’m hungry again! And my neck feels kinda funny too, like it’s twisting, tipping to the right. I feel the cool breeze on my feet. I hadn’t noticed that before.”

It is very important to note that Hope had been working on connecting her brain and heart for nearly a year. There is no way that Hope could have put a steel box around her heart a year earlier. A year earlier, Hope’s heart, the one in her actual body, was a two-dimensional valentine-shaped decoration. The version of Hope’s heart that she used for emotions and self-direction was over in her mirror-image’s body, several feet away. The Mirror-Hope’s version of a heart, like the hearts of so many PDers, was surrounded by barbed wire and warning signs.

To demonstrate the painful nature of recovery from Parkinson’s, I am going to take a side trip and share one of Hope’s recovery events. About nine months prior to this heart-in-a-box experiment, after mentally merging her imaginary “mirror self” and her physical self, Hope had started crying for the first time since she was six years old. She started crying while in my office. I asked her if she was able to drive herself back to work. She said that she’d be fine, that she’d be able to stop crying. She had always been able to control herself when she needed to.

The next week she reported that, after returning to work that day, she had not been fine. She had continued crying. Her sobbing was so severe that a co-worker had had to drive her home. When she got home, she continued crying. She cried on and off for the next few days. Five days later, two of her best friends, friends that she had introduced, died in a car crash on the way home from Disneyland. Their infant also died in the crash. Their eight year old was seriously injured, but survived. When she heard about the accident, Hope went into agonies. She cried, sobbed, wailed. She felt horrible pain in her chest. Her chest hurt, her face hurt, her arms hurt, her skin hurt. She couldn’t tell if the pain was coming from the crying or if she was crying because of the pain. She had not cried since she was six years old. She didn’t know if she was crying for all the pain of her childhood or if she was crying for all the unhappiness in the world, or if she was crying for herself or others.

Hope had been a despised, unwanted third child in a well-to-do family that did not believe in touching or emotions. Her parents rarely spoke to her and grudged her the pennies that they spent on her. If Hope was injured, she had to forfeit her Christmas money from Grandma to help pay for any doctor bills. Her mother had never touched her. Her father had hugged her once, briefly, furtively, when mother was out of the house, when Hope brought home a straight A
report card. As a baby, Hope had been cared for by her Portuguese maternal-grandmother. When Hope was six years old, the grandmother had been thrown out of Hope’s home – which actually belonged to the grandmother – because Grandma had “interfered” with the family dynamic by making a dessert for dinner. Hope’s mother accused Hope’s father of preferring grandmother’s pie to her own pies. After that, Grandma had to go.

At any rate, over the last year Hope had been learning how to cry and experience her own emotions. It had not been painless.

This extra information about Hope is merely to make the point that this simple heart experiment would not have been possible without the struggles that Hope had already gone through in learning to visualize her own brain and then her heart, and then realizing that her feeling heart was actually being kept in her mirror image. She had done the painful work of reintegrating with her mirror image. She had done an enormous amount of visualization work before she got to the point where she could now, with ease, participate in a meaningful way in the heart-in-a-box experiment.

Omar, the second partially recovered subject, observed that he didn’t notice much difference when he put his heart in a box and when he removed the box, but added, after a moment’s thought, “Maybe I felt sadness when I got rid of the box.”

His general tone suggested that he had already decided that he wasn’t going to feel much, one way or the other. So I decided to shake things up a bit. I asked him to pretend that, with regard to his children, he had a steel box around his heart and could not experience his sensory perceptions in his heart. That got to him. He is devoted to his two children, ages ten and seven.

He replied, “I refuse to do that.”

I countered, “I’m only asking you to pretend. Only for four minutes. And then you can pretend that you don’t have a box around your heart.”

“No. I refuse. What a horrible thought. How can you even suggest such a thing? That’s sick.”

“Well, I said, “I don’t see that it’s any more sick than asking you to have a box around your heart with regard to everything, which you just did with perfect ease.”

“That’s different. I like to think I care about a lot of things. I care about my children.”

He finally agreed to try the experiment. He observed afterwards that, “I was very uncomfortable with the first part. In the second part, when my heart was open, I couldn’t focus. I felt a different kind of discomfort; I felt sad. (I later found out that he considers sadness to be a sign of spiritual advancement. He feels that because compassion is a virtue, and sadness is a sign of compassion, that a “good” person should be sad. He had told me at an earlier date that, to him, the concept of feeling is synonymous with sadness. So it should have come as no surprise to me that, when he pretends that his heart is open, he feels sad.)

Next, I asked him to do this experiment with regard to something he hated. He answered that he didn’t hate anything, or at least he tried not to.


“No! I would never name my child Augustine! I once knew a real jerk….”
“OK,” I continued, “Let’s pretend that, with regard to the name Augustine, your heart is in a steel box. With regard to the name Augustine, you cannot and do not experience your sensory perceptions in your heart.

He happily did the experiment. He reported, “I didn’t care about not feeling. I expected to be uncomfortable in the second part, but I didn’t feel much of anything either way.”

My sense was, in phase two, he had only gotten rid of the experimental box around his heart. He still had a major heart-brain disconnect with the name Augustine, so of course he didn’t feel anything for “Aaron” even when he got rid of the box. As for his children, they too were still subject to his ever-present heart-brain disconnect, and so he didn’t feel waves of joy when he got rid of that experimental pretend box: his experimental box around his heart was only an overlay on his pre-existing box, so no real breakthrough occurred for him – at first.

I asked him to do the experiment with regard to his children one more time, but this time, he would spend only one minute with his heart in a box, and spend four minutes without the box. This time, there was a budding difference. He reported that, when his heart was not in the pretend box, he felt unfocused; it was confusing. I asked if he had felt sad. He replied that no, it wasn’t sadness. It wasn’t baffling, it was… He never finished the sentence.

A moment later, he said, “As for sadness, it’s not a problem. I’m comfortable with it. I’m comfortable with being sad.”

I have since learned that confusion is a not uncommon initial response to having an open heart. Sometimes a PDer spends months feeling deeply confused as his heart starts resonating with surrounding experiences. Omar did this heart-in-a-box technique for four months before he suddenly turned into a person with a rich emotional life and an ability to feel his own body and emotions.

At the time of the first experiment, however, I just suggested that being sad was not a sign of spiritual greatness. I asked him to name one saint, sage, or prophet who he admired. He named Ex-president Carter. I asked him if Carter walked around feeling sad. He said, “No,” and then looked stunned at his own answer.

He asked me if I didn’t think that sadness was the same as compassion. I told him that a loving and wise person may feel compassion for the sadness or the happiness of others, but that he was under no compunction to carry sadness in his own heart. I gave him the example of how Jesus wept when informed by his sobbing friends that Lazarus was dead. Jesus knew darned well that he was going to raise Lazarus from the grave, but nevertheless, deeply touched by their sorrow, Jesus wept – with compassion for their sorrow. Omar then said that he didn’t like examples from scripture. “I admire people who can have a religion, but I can’t have one.”

I next pointed out that some people, including religious adherents of many faiths, past and present, think that they must suffer or be sad in order to be virtuous. Some people, even today, wear shirts with bits of metal imbedded in the cloth or whip themselves bloody in hopes of acquiring a spiritual disdain for the body. Did he think that this made them more virtuous? If so, I could understand his insistence on being sad. He said that he doubted that people needed to suffer in order to be loved. He loved his own children and did not hope for them to suffer.

I asked if he hoped for his children to be sad, so that they might be virtuous. He was appalled at the idea. He did not want his beloved children to be always sad. I asked him if he wanted me to be always sad because of his inability to open up his heart. He didn’t want me to be always sad.
So then I asked him how he could possibly imagine that anyone should be always sad in order to be virtuous, since even he didn’t want any of his loved ones – or even his doctor – to be sad forever.

He was visibly shaken. He mumbled that he had never thought of it this way, in terms of his children and what he wants for them.

It felt like a critical, breakthrough moment. He might still have a tremor and an arm that didn’t swing, but we had found out that he had learned, as a child, that an open heart, a feeling heart, had to be a sad heart.

As for the rest of the experiment, he remained aware that he had noticed a change when he got rid of the box: he had felt strange, confused. Very possibly, his heart had been in a box for so long that when he lost his heightened level of alertness, he felt a relative inability to focus.

A little background is necessary to understand what a huge step it was for Omar to do this experiment first crack out of the box. During the previous year, when I introduced the heart-brain connecting exercise, he had struggled for an hour, once a week, for three long months before he could actually dare to pretend to see his brain (one hemisphere kept getting dark and disappearing – or worse). After he could imagine himself having a brain, we had worked every week with every tool in our visualization workbench to get him to the point that he could imagine his own heart without the heart crumbing into ash, withering, or fading into darkness. For him to easily participate in this heart-in-a-box experiment was proof of how tremendously far he had come.

The next partially recovered PDer noted that when he first did the experiment, he felt no difference either way. He seemed bemused, almost proud of the fact that he didn’t notice any difference when his heart was encased in steel or when it was accessible. I noticed something curious about him.

Even though I had given him instruction to, during the experiment, notice everything outside the windows, listen to all the sounds inside the room and outside the window, notice everything that’s going on around, he did not. Instead, after starting the clock, he immediately closed his eyes and became rigid, choosing to stay lying down on the treatment table.

I kept telling him to open his eyes, to move around, to notice how his perceptions registered when his heart was closed. He remained rigid, eyes closed.

“Open your eyes!” I commanded. He kept them closed. I nudged him hard on the shoulder. He opened one eye and stared at me. I asked him how he was going to register visual perceptions if he kept his eyes closed. I asked him to look out the window. He did so, but it seemed as if he did so grudgingly. Somehow, in his mind, closing off the heart must have also meant shutting down the body. And he had been able to shut everything out so well that he may not have even heard my increasingly loud commands. This was a person who was good at shutting himself off.

I asked him to repeat the experiment and notice sensory events. The second time, although he insisted he felt no change in phase two, I noticed a change in his exhalation when I announced “Four minutes is up; now get rid of the box and pretend that you can and do feel your sensory perceptions in your heart.” His exhalation, previously very slight, almost imperceptible, came out with a chesty “huh huh huh huh huh” sound as if he were releasing a held-in sob. We
did the experiment several times, and upon entering phase two, he always sounded as if he were about to start crying.

I asked him to do the experiment again, putting his heart in a box with regard to his ability to feel emotions about his beloved daughter, age 26.

In response to this request, he looked at me with a face that seemed impassive, slightly bemused, but his eyes began to water a tiny bit and his smile seemed less certain of itself.

“Pretend I don’t feel love for my daughter? No. Why would I want to do that?”

“Why not? You don’t seem to mind pretending that you don’t care about anything; isn’t your daughter a part of “anything”?”

“Yes, but I love her.”

“And you choose not to love the rest of the world?”

“How can you say that!”

“Then why were you pleased with yourself when you noticed no difference when you pretended to put your heart in a box just a minute ago, but now you aren’t willing to pretend that you can do that with regard to your daughter.”

“But why should I?”

“Well, because I’d like you to do this horrible thing so that you can enjoy all the more how you feel when you cast off the box. If you learn to notice how bad it feels to pretend to have your heart in a box maybe you can see how good it feels when you pretend that you don’t. We need to get a reward system going in your mind around getting rid of pretending to be shut off. The best way to feel good, for someone in a negative mindset, might be to really focus on feeling bad, and then consciously reverse that bad feeling.”

He didn’t understand, and I’ll admit I hadn’t put it well. Also, so far as he could tell, putting his heart in a box felt good, not negative. It felt smart and familiar and safe.

I gave him a physical example of using an opposite condition to induce the desired response: I asked him to relax his right (healthy side) arm. He did so. Then, I told him to tense his right arm for a few moments and then relax it. I asked him if his arm felt more relaxed the first time or the second time. He replied that his arm felt more relaxed after tensing it and then relaxing, than when he simply tried to tell it to relax.

I said that this was the same principle I was using in getting people to pay attention to their hearts and practice opening them up. By paying attention to the heart and pretending it was temporarily closed off, they could experience a large payoff when they then pretended to open their hearts.

He nervously agreed to do the experiment again, and to do it with regard to his daughter, on the condition that he only keep his heart in a box for one minute, not four.

This time, he felt that it was much harder to pretend the box was in place. He noticed no changes in sensory perception (colors, sounds, feelings) when he got rid of the box but he did feel relieved when the experiment ended.

We repeated the experiment with regard to the daughter, and limited it to twenty seconds in a box and twenty seconds after getting rid of the box. Again, he was relieved when it ended, and this time, he again released a long exhalation of relief that, to me, sounded like repressed sobbing.

The next time, I asked him to do the experiment with regard to his torso’s left upper quadrant. When I had met this patient, he was stooped, he shuffled, his voice was soft and his face was very masked. His left arm was becoming weak and didn’t swing, and he had a slight
tremor in the left hand. Now, his previous Parkinson’s symptoms were gone except for a worsening problem with intermittent tremor in his middle finger on his left hand and/or tremor in his wrist that worsened when he stretched the muscles of his left chest, as when lifting his left arm over his head. Recently, the tremor had become increasingly variable and more affected by mood. Sometimes it wasn’t there, but sometimes it was starting to interfere with typing.

For the past month, PD Team members and I had been focusing on what seemed to be evidence of a deep muscle tear somewhere in his left upper quadrant – a tear that seemed resistant to healing.

I told him that with regard to his upper left quadrant he only needed to have his heart in a box for twenty seconds, since the main point of the experiment now was to see what happened when he got rid of the box.

I watched the clock and told him when to start, when to get rid of the box, and when to stop. Then I asked him for his observations.

He didn’t answer. He was lifting his left arm up and down. He very, very slowly twisted his arm one way and then another. He was clearly experimenting with something in his arm.

I waited about twenty seconds and asked again if he had any observations. I might as well have not been in the room. I don’t know if he even heard me. Now he was lifting his left arm gently, very carefully, up over his head and stretching oh-so-gently. He started moving his left arm in slow, graceful circles.

I waited another thirty seconds and asked if he had any observations. He continued to silently play with his arm. He was moving that arm as if he was discovering it, as if he’d never had a left arm before. I gave up, and went to write up my notes at my desk. A few minutes later, when his appointment came to a close, he left in a hurry; he needed to get somewhere across town. As he dashed out the door, I told him to practice that upper left quadrant – heart in a box experiment as homework.

I called him up the next day and asked if he was doing his homework. I was surprised when he replied, “You bet! And I’m doing much better. My arm is really doing better today.”

Previously, when I had asked him to connect his brain and his heart, to imagine his heart being big, or to imagine himself or his arm being full of light, or any of the dozens of techniques that we had attempted, he had told me that doing those things seemed stupid. He had never practiced any of the other techniques when he got home. And why should he, since they felt “stupid.” This heart-in-a-box exercise, on the other hand, had made him feel good. And the active part, pretending that some part of him was in a steel box, did not feel stupid. Why would it? Heck, it was what he did all the time! Phase one had not seemed stupid; it seemed normal. And the payoff was that he felt good during phase two. So he was doing his homework, finally, and his arm was becoming responsive.

End of the experiment

I was elated. This had been a very simple experiment. All of those who had a presumably healthy heart-mind relationship, the musicians and those who had recovered from Parkinson’s, had somewhat similar responses: they hadn’t particularly enjoyed phase one, even though it was only pretend, but they had been able to do it and had noticed sensory differences between phase one and phase two. They had all felt a rebound of joy when they started phase two, when they
stopped pretending. They had also noticed various sensory changes for the better when they stopped pretending.

**Conclusion**

This little experiment had demonstrated that a person could switch his perceptions over to the sympathetic mode, the mode that uses adrenaline, the mode of wariness, by simple pretending. It also proved that a person could be in this mode without creating the rapid heart and breathing symptoms that are seen during a huge surge of adrenaline.

The experiment was over, as far as I was concerned. Others might wish to run the same experiment with hundreds of subjects instead of three groups of three. Others might wish to measure the heart rates and do brain scans to look for “real” changes being set in motion by a game of pretending. But as far as I was concerned, I had seen enough.

Healthy people had been able to induce, via pretending that the heart was inaccessible, sensory changes consistent with a switch over to heart-brain dis-entrainment and the sympathetic nervous system.

During this switch over to adrenaline-based behavior at a very low level of adrenaline, breathing rate and heart rate had remained slow.

After pretending in phase two that the heart was accessible, sensory changes occurred that were consistent with joy, dopamine release, and the parasympathetic system.

Significantly, people who were stuck in some stage of partial recovery had not been able to experience a quick change into joy. They had, however, experienced unexpected sensory changes such as hunger and relief, changes that suggested turning on the parasympathetic system. Most curiously, they had not felt any shift when they put a steel box around their heart. I propose this is because a steel box or some other imaginary mechanism is already there.

The variations in response between people with a healthy heart-mind relationship and people in partial recovery were extremely telling. But most importantly, for now, the Little Experiment had proved that a person could evoke a physiological response that effected perceptions and thought simply by pretending that he couldn’t feel. Also, the heart rate and breathing rate remained very calm during this game of pretending.

**Looking over the results in light of partial recovery**

The group that was partially recovered from Parkinson’s behaved, at first, as if their hearts were already in a steel box, and so phase one didn’t create a perceptible change. However, they all had some type of response, however small or weird, when they got rid of the box in phase two.

Curiously, despite their having no initial objections whatsoever to the box, and not noticing any change when they pretended the box was in place, they had nevertheless been powerfully opposed to sealing off their hearts with regard to their children. This suggested to me that, although they might already have their heart in a box with regard to themselves, there are some arenas in which they have decided to keep their hearts open. This rather suggested to me that a full range of choice might be involved in the fantasy of the closed heart – meaning that the PDer might have far more control over this heart-closing game than he might want to admit.
Overall, their responses when the box was removed had been slight, if at all – maybe a
sense of relief or confusion – and not nearly as large a response as the joyful rebound
experienced by the musicians and recovered PDers.

The third partially recovered may have actually noticed a significant change after he did
this experiment, with regard to his injured upper left quadrant. Evidently, his relationship to his
arm experienced a shift.

Looking again at the recovered PDers’ responses

The recovered PDers were the most gratifying. They, like the musicians, knew what to
expect when they closed off the heart. They knew it was going to be weird and it was. And they
were relieved when it was over. And the best response of all had come from the subject with
psoriasis, who never knew he had Parkinson’s, who never knew about the possible significance
of his age-ten ankle injury, who had never read my books, for whom six years had passed since I
had worked on his injury, who had said brightly, “It seems so familiar; I remember doing it when
I hurt my ankle.”

Follow up

I have since performed this experiment many times with PDers who are in recovery. I ask
PDers who have finally learned how to imagine themselves with a healthy heart to practice this
exercise as much as possible – even every few minutes. In our limited experience, even those
partially recovered PDers who do not notice any shift at first (and who become very discouraged,
certain that they will never learn to feel anything) usually find that, with practice, they can
become very good at opening the heart and keeping the heart open.

The results of this continuing experiment are uniformly suggestive that PDers are indeed
pretending to be cut off from their feelings, their heart. Those who make progress at keeping
their hearts “open” or “turned on” also become increasingly aware that there periods of poor
mentally preoccupied with negative thoughts.

WHAT FOLLOWS GETTING THE HEART OUT OF THE BOX?

“I must be crazy”

In the new clarity that comes from practicing this exercise, a clarity that allows people to
recognize whether they are in negative mindset or positive mindset, heart in a box or not, many
PDers have had an unwelcome surprise. They realize that they are living as if their heart is in a
box, they set out to change this negative mindset; that’s when the power of mental habit
suddenly surprises them.

Many who had imagined themselves to be disciplined thinkers suddenly realize that they
have, in truth, almost no discipline over their negative, anxious, even self-immobilizing thoughts.
They had imagined themselves to be disciplined thinkers because their brains were always
humming away. However, when they start observing their own thoughts carefully, they are
alarmed to realize that the mind is running along in its habitual negative patterns and is highly
resistant to change. After decades of the negative tilt to thinking that is inherent in the
dissociation response, they have built up powerful habits of mental negativity. Attempts to
replace that negativity with positive thoughts and the calm awareness that comes with an open
heart are met with firm resistance: the habits of the mind create a force strong enough that the mind appears to have a “will of its own.”

*Not a surprise to everyone*

Then again, this shocking, two-part revelation, that a negative attitude is prevalent and that it is a tough habit to break, does not astonish all PDers; many PDers are already deeply aware that they are highly negative. They usually did not suspect the role that their attitude played in their rigidity and difficulty in initiating movement, but at least they were aware that they had some attitude problems. Some of these latter PDers, after reading the previous edition of this book with its minimal notes on the role of negativity and fear, have told me, “I’ve got my work cut out for me; I recognize that I am a very negative person.”

However, some PDers have been furiously resistant to the idea that their mindset is entrenched in the sympathetic nervous system, a system that requires thoughts to be of a negative, wary orientation. For these people, the various heart exercises, and especially the heart-in-a-box exercise, have been a helpful. Through these exercises, these PDers are able to become aware that they are living with a mind well-trained in negative, movement-inhibiting thoughts. This realization does not bring about an automatic cure. However, it can make PDers appreciate that they have some bad habits. If they are lucky enough to be emotionally mature, they also realize that the responsibility for changing ones thought patterns rests squarely on the shoulders of the one with the mental habit.

Some PDers resist any responsibility for their own mental habits. Their response to the idea of changing the mind is a defiant “My mind is just the way it is. That’s who I am! I can’t change how I think! Thinking is automatic!”

Fortunately, new research in the field of neurology is proving, objectively, that the brain’s thought linkages and habits are set in motion by conscious thoughts, and they are changeable. The brain is very plastic, and its linkages and neuron growths are the result of one’s thoughts – not the reverse. Thoughts themselves determine the direction in which brain links will form, and with which thoughts they will link.

It is mental habit and mental training, not genetics or diet, that determines whether or not a person will respond to the idea of a tropical vacation with a series of thoughts about what might go wrong or a series of thoughts about how much fun the vacation will be.

These mental habits are changed or confirmed every single time a thought is formed. Changing mental habits is a formidable task. However, if a person who is locked into a dissociative (sympathetic system, danger system) mindset truly wants to recover from Parkinson’s disease, he must take control of his own thoughts and retrain them to resemble the thoughts of a person with a balanced outlook.

The mind will strenuously resist any attempt at change. The mind hates changes. PDers who are trying to change a lifetime of wariness report to me that “When I try to change my thinking patterns, or even focus on some of your exercises, I feel like there’s a devil in my mind that won’t let me do what I want,” or “I feel like I must be crazy; I can’t control my own mind for even a few seconds!”

While these people are stunned by the lack of control they have over their minds, they should not really be surprised. By instructing the heart to “play dead” and the mind to be ever wary, they have created millions, possibly billions of neurological connections and links in the
brain that are patterned to follow a peculiar, one-foot-in-the-grave, if-I-err-I-die, never-let-my-guard down style of negative line of thinking.

While it may require only one second to decide to reverse the direction of negative thinking and replace it with positive thinking, a person may need tens of thousands of mental repetitions of positive thoughts to reverse and conquer the habits of a lifetime.

**PDers who’ve recovered easily**

The PDers who’ve recovered easily often have stories about how they made a decision at some point earlier in their life to change their negative mindset. Some of them have told me how they taught themselves to cry. Others learned to sing as a way to overcome their fear of being criticized. The PDers who recovered easily have, in the years prior their diagnoses of Parkinson’s, done an enormous amount of work at remaking their own mental patterns. They have disciplined their own minds to trust the universe. They have taught themselves to stop micromanaging their lives and the lives of everyone around them. They have taught their fearful minds to turn off the fear and surrender to the goodness all around.

Without even suspecting that they were carrying the foot-injury seeds of an immobilizing illness, these people retrained their thoughts because they recognized that their lifelong wariness was not bringing them happiness. This mind retraining then paid off in an unexpected measure when they were able to recover easily from Parkinson’s disease.

Those who have prided themselves on what they thought was their unique mindset and who have made no attempt to get themselves out of dissociative and sympathetic patterns of thinking find that, upon recovering from their foot injuries, they have a large job ahead: learning to have genuinely positive thoughts.

This subject will be addresses further in the chapters on treatment techniques.

**Moving on to the last remaining problem**

Finally, as partially recovered PDers started being able to open their hearts, feeling joy and feeling a return of physical ease to their movements, some of them still had one remaining problem: toe and foot spasms and cramping. Armed with the new knowledge of Qi flow changes that we learned about when people had their heart in or out of a box, we quickly found the cause and a solution for it. Happily and unexpectedly, this solution also provided support for the dissociation response portion of our theory.

The next chapter will explain this last problem and its revealing solution.

**The only difference is happiness**

Before closing chapter, I want to share one more case study about the heart-in-a-box exercise.

This PDers, after doing the exercise several times, observed that there was “no difference [doing the exercise he felt nothing different from how he usually felt, with or without a steel box]. No difference…just happiness.”

He explained further, as he wiped away an uncharacteristic tear. “There was only one difference: I felt happy looking at and hearing everything when my heart wasn’t in a box. But that’s the only difference… just happiness.”
John Bateson donated the tree artwork. He wanted show that, like a PDer, the noble tree is sturdy, dependable, rigid, punctual, and rooted to the spot. But even a tree acknowledges and responds to wounds.
And the time came when the risk to remain tight in a bud was more painful than
the risk it took to blossom.”

- Anais Nin

Chapter Sixteen

Untitled

The mystery of the toe spasms

Toe spasms and toe dystonias are not uncommon symptoms in medicated and
unmedicated Parkinson’s disease. Researchers have even found that the dyskinesias of
Parkinson’s that are brought on by excess medication most often develop first in the feet and
toes.

We noticed that many partially recovered PDers continued to have problems with toe
spasms, toe curling, and other toe-related problems. These toe problems have various triggers; in
some PDers, these toe problems worsen in response to a negative thought. In others, the toe
problems worsen in response to exercise. For others, the toe problems diminish in response to
exercise. One partially recovered PDer thought that wearing shoes was the cause of her toe
cramping; then she noticed that the cramping actually started when she thought about putting on
shoes.

We discovered the cause behind the toe curl mystery just a few months after starting to
do the heart-in-a-box technique. And bringing the foot injury and the mental/emotional blockage
parts of Parkinson’s full circle, we found that the most effective way to treat this symptom
involved both physical treatment of the foot and simultaneous practice of the two-part heart-in-a-
box technique.

A case study

We had known about our patients various toe cramping and toe spasm problems for
years, but had considered it an inconvenience, not a major clue.

However, one of my partially recovered patients was becoming increasingly worried
about his worsening toe spasms. He could no longer walk more than about ten minutes without
triggering very painful toe spasms. Walking was the only exercise that allowed him to relax his
mind and move easily. If, due to toe spasm, he could no longer go for his long walks, he would
have no way to induce his daily hour of normal, relaxed movement.

He was very concerned about the worsening of the toe trouble. He asked me to forget
about his very small remaining hand tremor and do whatever I could to stop what he called “toe
jamming.” I examined his foot yet again, but I was stumped. I could think of no reason for his
otherwise straight toes to be move in this strange way during walking. When his toes “jammed
up,” the toes were bending at the each of the joints, scrunching up as if some force in the middle
of the foot was powerfully pulling on the toes.

After staring at and playing with his foot for about five minutes, ruling out the possibility
of joint problems, muscle problems, nerve problems, and psychological problems (he liked
walking), I exclaimed, there is no force that I can think of that could be pulling your toes in
towards the center of the foot unless, by walking, you are creating some sort of electromagnetic field in the center of your foot that is sucking your toes in.

The very idea was so ridiculous that I laughed as I said it. And then I paused. Where was the Qi that was supposed to be flowing across the skin of his toes? Was it actually in his toes? I had never checked. Because my earliest PDer patients, ones who had recovered easily, had experienced a spontaneous return of normal Qi flow patterns throughout the feet and toes after the center-of-the-foot injury healed, I had just assumed that removal of the injury and the resumption of Qi flow in the right direction would necessarily cause correct Qi flow over the toes. I had never bothered to check what was going on with the Qi flow in the toes of anyone whose Stomach channel was no longer running backwards.

Now, working with Omar, I had to wonder if I’d been hasty. What if the Qi that was supposed to run across the skin of his toes was running, instead, down through the center of his foot. This would be a completely wrong pattern, a horrible pattern, something we had never considered in school. Channel Qi is supposed to flow just under the skin. It’s not supposed to dive deep inside and plunge into a different channel altogether.

If Omar’s Qi was running in this improbably manner, this aberrant Qi would probably not be a direct result of the injury. I wondered if the source of this aberration was the decades of incorrect Qi flow that had developed around the injury site. In other words, this wrong Qi flow pattern might be a secondary aberration, not set in motion by the injury, per se. This secondary pattern would have developed over years, forming an electrical “habit.” Such a habit would not necessarily go away simply because the injury was healed.

Obviously, in some PDers, the whole feet came back to health when the injury at ST-42 was effectively treated. But, I had to wonder, was it possible that, in some PDers, in some of the partially recovered PDers, this secondary problem of aberrant Qi flow in the toes remained in place even after the Qi in the rest of the Stomach channel was running in the right direction?

The Qi flow pattern in the toes has not yet been discussed in this book. In a healthy person, the Qi flow of the Stomach channel diverges at ST-42 into two paths. The larger of the two paths flows “sideways” across to the medial arch of the foot and joins up with acupoint SP-3 on the side of the foot. The smaller of the two paths fans out somewhat, flowing over the top of the foot and over the second and third toes. When the Qi arrives at the tips of these two toes, it flows across the tops of the toes towards the big (first) toe. Arriving at the medial side of the tip of the big toe, the Qi then flows down the toe towards the arch and joins into the larger stream of Spleen Qi at SP-3.

If the Qi in Omar’s foot was flowing from ST-42 down into the center of the foot and emerging at the sole of the foot, it could conceivably create the toe-cramping pattern that he experienced when he increased the Qi in his Stomach channel. Using the legs in a rhythmic pattern, such as walking or running, increases the amount of Qi flowing in the Stomach channel. It seemed as if Omar’s walking was causing more Qi to flow in his Stomach channels. The increased Qi flow was flowing through the middle of his foot and into the Kidney channel, on the sole of his foot. This diverted Qi might have been creating an electromagnetic pull on his toes.
Now I really was puzzled. Acupuncture is used to assist or increase the flow of Qi in a channel. If the Qi flow was now going in a completely wrong direction, purely from habit, and not from any injury or obstruction, how on earth could I make it form a new habit?

The only way I could think of was inserting a nearly continuous line of acupuncture needles along the correct pathway of the toes portion of the Stomach channel. This technique, in which a line of closely placed needles is inserted, and the needles very nearly touch each other under the skin, is called “threading” technique. This technique can be used to rebuild a channel that, for some reason, has become completely blocked over a large area and has started running in some unusual direction.

I decided to construct a correct path of the toes portion of the Stomach channel on Omar. There was no Qi in his toes. His feet, distal to ST-42, did not feel the needles. Sensing that no Qi was following my needles, I quickly, in a few minutes, constructed a double, parallel row of needles running from ST-42 to the tips of the toes, and another parallel row of needles running over to the side of the foot, connecting ST-42 and SP-3. There were now nearly fifty needles in his foot. Next, I added a needle at ST-42. Omar felt nothing in his toes. I added a needle just below the knee at ST-36. He still felt nothing in his toes. The Qi in his Stomach channel was running in the correct direction in his leg. The Qi ran in full quantity. However, it did not flow into his toes.

I asked Omar to do the heart-in-a-box exercise. I asked him to especially focus on his toes. When his heart was shut down, I wanted him to cognize any sensation in his toes using his brain, not his heart. When the box around the heart was cast aside and the heart was able to function like a radio, resonating with various wavelengths, I wanted him to “tune his heart radio” to the toes and concentrate on the feelings in his heart that was being broadcast by the electromagnetic vibrations in his toes.

After about twenty minutes of alternating between shutting his heart in a box, having no feelings and exposing the heart, letting it resonate with feelings coming from the toes, Omar had a most stunning response. After four years of working with us, during which he had struggling mightily to learn how to visualize, struggled to open his heart, and had no ability to fully cognize the existence of his left leg and arm, he suddenly “came to life.” He became aware of an un-nameable emotional pain in his shoulder and hip. He also felt a physical sharp pain, a “slicing pain” in his shoulder and hip, as if they had been badly injured, maybe even severed from his body.

Most strange of all, he suddenly began speaking to me using a completely different vocabulary, and in a different style. He spoke in an unguarded fashion, as if he was baring his heart. His vocal delivery style was more musical, and he was using words such as “feeling” and “sensitivity.” I stared at him in wonder and said, several times, “You’ve never talked with me like this.”

A light went on in my head. The dissociation response can most easily be induced by perforating the skin. People who regularly slash or burn the skin of their wrists do so to obtain the endorphin rush that accompanies the dissociation response. By putting fifty needles into his foot in less than fifteen minutes, I had probably triggered a genuine dissociation response. By having Omar practice shutting and opening his heart at the same time, he may have been able to
finally terminate a genuine dissociation response, not just a consciously-induced response, in the correct and normal way – by having a resumption of feeling. Omar experienced the usual rush of feeling and joy that is typical following the cessation of a dissociation response.

In a sense, I had taken Omar back to the time when he had first dissociated and hurt his foot and had him replay the scenario with a different ending – the correct ending.

I feel certain that, had Omar not been practicing the heart-in-a-box technique to develop some ability to feel with his heart, he would not have been able to make a correct exit from the dissociation response. But because he had been practicing, he emerged correctly from his most recent, and from his lifelong-long, dissociation responses with a rush of feeling.

Qi in his foot started flowing correctly, through his toes. He was able to walk without any toe cramping.

A few days later, the toe cramping returned briefly for one day.

When Omar returned for his next weekly treatment, I repeated the same toe needling. The toe cramping never returned.

**Repeating the treatment on other PDers**

I did this same treatment on other partially recovered PDers. I discovered a bizarre, utterly unexpected collection of incorrect toe Qi patterns that these people’s feet had created, through the years. In some feet, the Stomach channel Qi diverged at ST-42 and poured into the Bladder channel on the lateral side of the foot. In others, the Stomach channel diverged at ST-42 and flowed into the Gallbladder channel. I discovered wild aberrations in the Kidney channel. In some cases, Stomach channel Qi was flowing down through the center of the foot and into the sole of the foot, into the Kidney channel. But the Kidney channel was often distorted by this action. In some cases, possibly because the Stomach channel was flowing into the pathway of the Kidney channel, the Qi that should have been able to flow into the Kidney channel flowed, not through the Kidney pathway, but into the nearby Spleen channel. This move caused the Qi in the Spleen channel to flow into the nearby Liver channel. The origin of this particular series of shifts seemed to be, in some cases, the shunt from ST-42 to the ankle, at KI-6, a short-circuit that was mentioned in the early chapter on channel irregularities in Parkinson’s. This particular short circuit Qi at the ankle is not uncommon in Parkinson’s disease. The body’s ways for diverting energy around this confusion are many. Each person’s foot seemed to find its own way to get around the ankle snafu.

Still other aberrations in the Kidney channel seemed to have their starting point with a problem similar to Omar’s: Stomach Qi was flowing from the top (dorsal side) of the middle of the foot down through the center of the foot and emerging at the sole (planar side) of the middle of the foot.

I was fascinated by all the variations on incorrect channel Qi. But the treatment for all of them was the same: recreate the correct path of the affected channels by “threading” a maximum number of needles along the correct path of the channel, sometimes making two parallel rows of needles.

In all of these people, I constructed correct channels on their feet and then “activated” the new pattern by points at other locations on the leg. For example, to activate the newly constructed Stomach channel, I adding ST-36, below the knee and LI-4, on the opposite hand. The theory involved in the choice of “activating” needles is too lengthy to explain in this chapter,
but it will be clear to any good acupuncturist. The theory is explained in detail in the chapters on treatment techniques.

Most importantly, I only did this extreme form of needling on people who had already been practicing opening and shutting off their hearts: if I was going to perforate the skin at this excessive level, at a level that could induce a dissociation response, I wanted to be sure that the patient was prepared to resume genuine heart feeling when the needle effect wore off.

Other patients made similar emotional leaps forward. Some of them had mild improvements with each extreme (fifty needles, more or less) toe-needling treatment and needed repeated toe-needling sessions.

I had to wonder if we could dispense with all of the Tui Na and slow bother of starting the healing of the foot injury. Wouldn’t it be wonderful if a person could recover from Parkinson’s disease merely by getting fifty or sixty acupuncture needles in the foot? But then I remembered the earliest days of this research project. I had hoped that the Qi in the channels might be turned around simply using needles. But I had come to learn that needles in the feet and toes were not beneficial if the ongoing injury was still in place. If the Qi is running backwards in the Stomach channel due to an obstruction, the Qi will not be able to flow to the needles in the toes: the Qi cannot even reach as far as the toes.

Also, the ability to release dopamine in the brain cannot be attained so long as Qi is running backwards in the channel. Merely needling the toes was not going to turn off a dissociation response if the body remained injured and Qi was telling the brain to go to sleep.

I was disappointed. I had hoped that we might find a method of recovery that required only Tui Na and mind retraining. Tui Na can be performed by nearly anyone, and retraining the mind can be done by the patient working by himself; no acupuncturist or medical specialist is needed for these two aspects of treatment. If some people were going to need channels rebuilt and possibly even a skin-perforation-induced dissociation response, this meant that these people might need to seek out the services of an acupuncturist.

My goal has always been to come up with the easiest possible way to treat Parkinson’s disease. My hope was that no specialist would be needed in the process. However, it appeared that, for some recovering PDers, intensive needling of the foot and toes might be necessary after the Qi started flowing correctly and the PDer was able to practice using his heart to experience feelings.

Still, even if it turned out that acupuncture needling was a necessary part of the recovery process for some people, this was still a small problem. This was not an insurmountable problem.

**Qi Flow in the Feet During Dissociation**

I was still baffled by the Qi flowing through the center of the foot, going from the top of the foot to the sole of the foot. Nothing like this had been discussed in any of my years of study.

Also, after I discovered that my partially recovered patients had strange Qi flow patterns in their injured foot, I checked all the rest of the Stomach channel in extreme detail.
I was stunned when I found that the portion of the Stomach channel that flows directly over the heart was, in most of these people, not flowing, or was flowing backwards. This is a small section of the channel, the portion that runs from the collar bone down to the fifth rib, at “the root, or bottom, of the breast.” Even more strange, the Qi picked up steam again below the fifth rib and continued down the leg as if everything was perfectly normal. And then, when it got past ST-42, it did not go over the toes in the correct fashion, but diverted into some incorrect path. Or I should say, a path that I thought was incorrect.

I tried needling to correct the portion of Stomach Qi that was not running correctly over the heart. I sometimes got a temporary correction, but the incorrect pattern usually resumed quickly.

Now I was truly baffled.

I won’t go into all the details of my hunches. I will just say that I devised an experiment to see what was supposed to happen to Qi flow in the Stomach channel during various conditions. I decided to test three situations on each subject: relaxation, dissociation, and a call for rapid action.

Not only was this experiment designed to see what happened to Qi flow in the above situations, it was a test to see whether or not the Qi flow alterations could be induced by merely thinking about the situations.

This is the experiment: I asked healthy volunteer subjects to imagine three different scenarios. Immediately following the scenario, they were supposed to notice what was happening to the energy in their feet. I orally led them through the scenarios. Here is my script:

“Imagine you are resting by the seashore or a nice lake. It is a nice sunny day and you want, as much as possible, to be feeling the warm air on your skin. Imagine that a warm current of energy is flowing over the top of your foot. This warm feeling runs from the ankle to the center of the foot, and then spreads out over the top of the foot and goes to the tips of the second and third toes, and over to the arch-side of the foot, near the first toe. Can you imagine that?”

After the subject said that he could imagine this, I introduced the next idea:

“Suddenly, a lion has appeared. He has grabbed your left shoulder in his teeth. His teeth are sinking into your chest, and he is going to pull your left arm off. Can you imagine that?”

As soon as the subject said yes (which was usually very quickly), I asked, “What is happening to the energy in your foot?”

The answers were thrilling, gratifying, beautiful. Each person was surprised to feel that the energy in his foot had stopped flowing. In some cases, it was vibrating back and forth very quickly, but flowing nowhere. In others, the feeling of energy was going backwards, towards the ankle. In others, in most, the energy seemed to have stopped at the top of the foot and was flowing into the sole of the foot. A few people noticed that their feet and toes were curling under.

Next, I said:

“No! There is no lion! It was all a wrong idea. You’re fine. Your shoulder is fine. You weren’t bitten. You’re alive, after all! You’re fine! Now tell me, what is happening to the energy in your foot?”
The responses were all the similar: energy was once again flowing over the top of the foot and flowing out to the toes.

Next, I said:
“Imagine that you are sitting on the grass at the park, and your sixth month-old daughter or granddaughter is sitting ten feet away. Suddenly, you realize that some crazy creep is sneaking up on her with a two-by-four piece of wood, and is planning to whack her in the head. Can you imagine that?”

As soon as the subject said that he could imagine it, I asked him what was happening with the energy in his feet.

In nearly every case, the subject was surprised to feel surging energy pouring over his feet. Some subjects felt the soles of the feet throbbing with energy, as if the feet expected to be put into instant use.

Next, I said:
“Wait! There is no crazy person. There never was a crazy person. Your child or grandchild is OK. It’s a beautiful day at the park, and you are sitting on the grass, enjoying yourself. What is happening with the energy in your feet?”

In each subject, the energy in the feet was back on the surface, spreading over the top of the foot and flowing peacefully into the toes.

The above three scenarios – contentment, dissociation (prepare for death) and emergency (prepare for action) – all created immediate energetic responses in the feet. These responses were too fast to be the result of neurotransmitter release. In fact, these responses happened so quickly that in the rebuttal of the crazy person stage, the adrenaline released by the scenario had not yet been neutralized, but the energy in the foot was able to immediately go back to the “relaxed” flow pattern.

This suggested that Qi flow responds immediately to thoughts. It also suggested that Qi flowing from the Stomach channel down into the sole of the foot, even to the point of causing foot and toe cramping, was a normal and healthy part of the dissociation response.

I thought about how a mouse looks when it “plays dead” after being snapped up by a cat. The mouse does not actually go limp; the mouse’s tiny legs pull tightly inward, as if it is imitating the rigor mortis condition that occurs hours after death. This look, even more than the limpness of death, might discourage a playful cat from continuing with his sport. No cat wants to waste time with a mouse that has been dead for hours.

And so, had we found the reason for those toe-curling problems?

It appeared that the toe spasms and foot cramps were not actually signs of pathology; they were the correct foot response to any thought that is associated with potential death. Because some PDers have learned to keep their minds focused on potential danger and even potentially dire danger, the foot and toe spasm response may be an electrical shift that is a correct response to this type of thinking.

**Qi flow over the heart**

While I was conducting the above experiments, I held my hand over the portion of the Stomach channel that flows in the vicinity of the heart. The rest of the Qi flow mystery was
answered. During the contentment phase, the Qi flowed correctly. During the dissociation phase, the Qi stopped flowing and dove into the chest. During the “call to action” phase, the Qi of the Stomach channel dove into the chest and flowed into the source current of the pericardium. The pericardium is the muscle that separates the heart from the lungs. In some subjects, a shift away from the path of the Stomach channel was felt and a simultaneous increase could be felt in the Starting Point of the Pericardium channel, when a call for action was imagined.

In other words, the Stomach channel does not flow in the same pathways at all times. The Stomach channel is closely associated with the vagus nerve. During those times when the body is in parasympathetic mode and the vagus nerve is running merrily, the Stomach channel is also running close to the skin, making its way from the forehead to the toes. During times of impending death, the Stomach channel ceases to flow. As researchers already know, during such a time, blood is shunted deep within, and the body goes into a state of suspended animation. This condition is called dissociation.

During times of emergency, when the mind imagines a call for action, the energy in the vagus nerve diminishes and the Qi in the Stomach channel is shunted towards the heart to provide extra energy to the heart. Further down the torso, just below the level of the lowest rib, the Stomach channel re-emerges, at full strength. The Stomach channel seems to become fortified, to pick up extra Qi from the Stomach point on the Ren channel (just below the level of the lowest rib). The Qi at this point on the Ren channel normally provides additional support for the functioning of the stomach organ, and especially the functioning of the duodenal muscles at the exit from the stomach.

During a call to action, these muscles are not used. The energy for operating the opening at the bottom of the stomach is instead shunted into the main path of the Stomach channel just below the stomach, and then flows down the Stomach channel into the legs and feet. In this way, the Stomach channel is able to flow into the legs, but the portion of the Stomach channel that would normally direct the functioning of the stomach itself is shunted into the heart to give extra energy to that organ.

This shift in the Stomach channel pathway makes absolute sense, and corresponds exactly with the shift in heart-nerve function when the body switches between the parasympathetic system and the sympathetic system.

It makes sense. However, I had never heard of such a thing. Imagine my amazement when, not three weeks after running this experiment, I heard a lecture (in translation) by Dr. Yuji Wang (mentioned in a footnote in chapter one), in which he stated, “The Qi runs in the normal channel pathways maybe 70% of the time. The rest of the time, the Qi may be running in directions that do not match the basic texts. In illness, you cannot assume that the Qi is running in the normal pathway.”

Adding foot awareness to the mix

Now that we knew that Qi was supposed to run in specific patterns during various mental/emotional states, we had a new way to help partially recovered PDers assess whether or not they were still locked into, or floating in and out of, a dissociation response. If they were able to feel their feet, they could notice, at any given moment, whether or not the Qi was running over the top of the feet or was “standing still” (diving deep inside). If they found that the Qi was not running over the surface, they could practice putting the heart in the box right then, getting the
heart back out of the box, and then checking the feet to make sure that the Qi flow had gone back to the correct pattern.

With many partially recovered PDers, we added the intensive needling of the toes to the process. When the needles were being inserted, the PDer was allowed to dissociate if he wanted. He could imagine his heart getting shut in a box and turning himself off to pain. But as soon as he felt his tension start to fade from the fear or sensations of the needles, he had to get rid of the box around the heart and focus his heart’s sensitivity on the sensations coming from his feet. As he focused on the sensations in his feet, sensations enhanced by the presence of the needles, he needed to affirm to himself over and over, with as much positive emotion as he could muster, “I’m not dead after all; I’m not dead after all!”

In this way, PDers had many angles to work on in learning how to turn off their consciously-induced dissociation responses.

This led to a new thought, one not actually related to Parkinson’s, but which can be used during recovery. The question arose for me, what is the normal, correct way for a dissociation response to get turned off? Unlike the other sympathetic nervous system responses, which stimulate the heart fires and which ebb when the danger is over, a dissociation response is a response that prepares an animal for death. The heart itself is nearly turned off. In a dissociation response, the heart behaves as if its pilot light is turned off. How can the heart become turned back on if the heart itself is turned off?

Thinking about patients who have had near death experiences, who have recounted to me their intense emotions and sensory experiences when they were “brought back” to life, I have arrived at a hypothesis: the trigger for turning on the heart is sensory input.

During the dissociation response, the body releases endorphins. These chemicals are very like opiates, morphine, heroin. These chemicals numb the body to physical sensation. When these chemicals wear off, when they are neutralized, the body experiences an period of enhanced sensory awareness.

The user of heroin may experience an period of enhanced sensory awareness when his drugs completely wear off and he goes into withdrawal. During this painful period, light may be painfully bright, sounds may be painfully loud, the sensation of clothing on the skin may be irritating or painful. All of the sensory functions in the body go into a hyper-sensitive phase when the heroin wears off.

In the same way, but far more pleasant, a person who has experienced a surge of endorphins during dissociation may have a period of enhanced sensory awareness when the endorphins are neutralized. I propose that it is this surge of sensory awareness that restarts the heart energy.

A new baby is catapulted into a different condition of heart-nerve function when he goes through the wildly sensory experience of birth and the first burning breaths of air. His heart gets kicked into “fully operational mode.” For the rest of his life, in order to keep breathing, the child’s heart must keep the pilot light going and keep some amount of heart-nerve energy dedicated to the sympathetic mode.

If an animal goes into a dissociation mode, a prepare for death mode, in which the heart signals to the brain diminish and the heart function is set to “minimal,” it may be that a surge of sensory input, such as that experienced by a new baby or that experienced by a junkie when his fix wears off, is the necessary trigger.
And how does that relate to Parkinson’s disease? It is the answer to the entire story. Many people with Parkinson’s remember, before, during, or after recovery, when they decided to pretend that they could not feel physical and emotional pain. If the sensation of physical or emotional is the trigger that can turn the heart back off and terminate a dissociation response, what will happen to a person who dissociates and tells himself to feel no physical sensation?

He will be unable to turn off the dissociation response. He will have created a mental situation that prevents himself being able to fully feel. Because he cannot feel, he cannot turn off the response.

By using his mind to deny his experiences and by telling himself that he does not want to have future sensations – if they might be painful – he is setting himself into a situation in which he locks himself into a self-induced dissociation response and simultaneously sets up a condition that will prevent himself from getting out of the response.

The person who initiates such a mind-game may, in the beginning, want the physical and emotional numbness to apply to only one or two specific situations. However, because of the way that the mind is designed to work, negative (sympathetic system) mental patterns create links. Over years, and then decades, negative mental patterns spread through the brain until a majority of situations may be deemed (by the subconscious or conscious mind) suitable for dissociation. Increasingly, fewer and fewer situations and scenarios will be considered “safe.”

Parkinson’s disease, a disease that, in its latest stage, resembles living death, may be a manifestation of a decision, made decades earlier, to be numb to one’s own physical and emotional pain: a decision that creates the electrical patterns identical to those of a dissociation response. In this condition, a foot injury that might have healed, or at least hurt, in any healthy person, will not be able to heal. This foot injury, because of the layout of the foot bones, is very likely to disturb the format of the foot at the terminus of the Stomach channel. If this disturbance goes unhealed, it will set in motion electrical patterns that, in a healthy person, should only occur during recovery from an intense injury. These electrical patterns are supposed to make a person fall easily into healing sleep. These electrical patterns are supposed to shut down, temporarily, neurotransmitter release.

These electrical patterns make fixed changes in the manner of brain function. If a person decides, years after his foot injury, that he no longer wants to be shut off from his feelings, the electrical patterns in his foot and brain may have already become established. Now, even if he wants to have normal parasympathetic function, feeling contented and keenly aware of his heart’s ability to sense feelings, he may be unable to make the switch: his brain has become locked into a pattern that keeps him behaving as if injured.

In the beginning, the mentally-induced dissociation response may prevent his foot from healing. In later years, the electrical patterns set in motion by the unhealed foot injury may prevent him from turning off the dissociation response.

To recover from Parkinson’s disease, a person must recover from both his foot injury and his mental/emotional preference for dissociation.
A human is not a tree.
CHAPTE R SEVENTEEN

UNTITLED

This wrap-up chapter is still under construction.
It will be posted as soon as it is finished.

Also, the unfinished chapters on the techniques that some people have found useful in turning off the dissociation response will be posted as soon as they are finished. They will be in Part 2 of the book, which deals with treatment techniques.

W/re page numbering in part 2: because I do not know how many pages will be in this chapter, I have started the page numbering for part 2 with the randomly assigned page number of 401.

The chapters on recovery symptoms, frequently asked questions, and statistics will be in Part 3, and will be posted when they are finished.
CHAPTER EIGHTEEN

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, and Na means hold or take. 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. 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

---

2 Ibid. p.486
Balancing, Healing Touch therapy, Positive Touch therapy, Bowen therapy, and hugs, to mention just a few in an 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. 4

**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

---

4 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.
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.

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. 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,

---

5 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.
indigestion, or other maladies and pains that are related to the traumas being retained in the soft tissue.

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 “Spongeworth-Hugeussson” 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.⁶

**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

---

⁶ 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.
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 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 acupressure 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 acupressure 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.7

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.8

---

7 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.

8 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.
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.

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.  

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 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
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 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.10 A mere

---

10 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.
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 Guandong 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.

When I attended acupuncture school, the most common definitions were “bone medicine” or “skin rolling/pediatric massage.” The latter is a relatively new meaning of Tui Na, going back only to the Yuan dynasty. During the Yuan period, the Tui Na/massage department of medicine came under the administration of the bone setting department. A department of pediatrics was opened also and incorporated into the Tui Na department. It was during this time that Tui Na became divided into bone-setting and pediatrics.¹¹

Now, in modern times, Tui Na is the name given to almost any form of physical-touch work in which the doctor makes intentional hand contact with the patient. Technically, acupressure, a process in which acupoints are stimulated by finger pressure, could be considered a form of Tui Na.

**Historical use of Tui Na**

Tui Na is an ancient art, preceding the development of acupuncture. A Chinese text asserts “The unearthed oracle inscriptions … of the Shang dynasty record that the female witch doctor, Bi, could treat patients with massage.”

**Light-touch therapy in modern times**

One might say that the technique principles of ancient Yin Tui Na are becoming widely known once again in the west as well as the east. Or one might, more accurately, say that the basic healing properties of human touch, known in all countries since time immemorial, have recently become better understood than they have been in a long, long time. As the modern biophysics principles behind light touch therapy answer some of our questions about “how does it work,” the mystery behind light-touch therapy is melting away. As the mystery melts, this work is becoming more acceptable to religions and governments, including the Chinese government, that have, for many years, shied away from what seemed to be charismatic medicine.

Now, in both east and west, various new schools of hands-on techniques exclaim over and extol the seemingly miraculous health improvements that can result from directed holding, or extreme light-touch therapy. These schools are reintroducing in modern times, in various vocabularies, the same timeless principles that have been referred to historically in China as Yin Tui Na.

**A frequently asked question: Is there a Yin Tui Na practitioner in my area?**

There is no such thing as a specific technique called “Yin Tui Na,” per se. Asking for a Yin Tui Na practitioner is comparable to asking for a person who does any type of technique that is relatively gentle hands-on therapy, as compared to strong, vigorous therapy.

It does make sense to ask a Tui Na practitioner if he has studied Yin-style or Yang-style techniques. If the respondent says that he doesn’t know, or if he says that he does both kinds, this means that he probably has studied only Yang-type techniques. In general, most people who study Tui Na are studying the Yang type. As a generality, while practitioners of Yin Tui Na are very aware of the existence of both the Yin and Yang kinds, most practitioners of Yang Tui Na are not familiar with the ideas of light-touch work.

---

12 The actual text would have said that Bi could treat patients with Tui Na. Again, the use of the word “massage” when translating into the English is not accurate. The source of the quote is Chinese Massage. Publishing House of Shanghai College of Traditional Chinese Medicine. Shanghai. 1988. p.2. Note: the Shang dynasty dates from approximately 1766 BC to 1027 BC.

13 I am certain that every human culture has known about the therapeutic value of light-touch healing work. However, due to many cultural rules against touching, and the poorly understood and therefore “charismatic” mystique attributed to highly skilled practitioners of hands-on work, this form of therapy has been, in many cultures, relegated to shamans, midwives, mystics or – as in 20th century China – officially forbidden: as noted earlier, even the texts were revised to leave out the more Yin types of Tui Na.
People who have studied the Yin forms of bodywork have usually done so by studying western types of light-touch work. They would be more likely to refer to their techniques by the specific, western names of the modalities that they studied. For example, a western, hands-on, light-touch therapist might say: “I do both Gregson’s cranio-tarsal work and Master Wu’s Medical Unwrapping protocol.” However, despite this precise language, one hopes that, in practice, the therapist is exercising some amount of individual intuition and flexibility to move beyond the basic parameters of his coursework. Even so, simply from lack of bilingual understanding, they would probably not refer to their work as Tui Na. Most western-trained light-touch therapists have never heard the term “Tui Na,” although they are practicing what would be called, in China, techniques of Tui Na.

I will continue to refer to the techniques we use in treating Parkinson’s as being variations on a type of Tui Na. Hopefully, this use of language will give unspoken permission to therapists working with PDers: permission to change, adapt, and explore variations on the techniques described in the next chapter.

Rather than conveying the wrong idea that the following techniques must be performed to an exactitude, I hope that by referring sweepingly to our suggestions for PDers’ treatment as “a type of Yin Tui Na,” the students and practitioners of these techniques will understand that there is tremendous latitude.

The health practitioner will get the best result if he understands that each person is unique and that all modalities work best when they are modified or adjusted to fit the patient. Although, in chapters ahead, I will offer instructions for specific hand positions and descriptions of how much pressure to use, they will only be suggestions.

The therapist who wishes to become a master – even a master with only one patient – will understand that he is not learning a rigid program, but is learning to respond to what his patient’s body asks for. At that point, when he is doing what the patient wants instead of what the instructions say, he will be practicing FSR. He will be doing “a variation” of FSR. Which variation? He will be doing his own and the patient’s own. The general term that I use that embraces that unique variation is Yin Tui Na.

---

14 Both of these named techniques are fictional, created for the sake of example.
...Bearing thy heart, which I will keep so chary; as tender nurse her babe from faring ill.

- William Shakespeare, sonnet XXII

CHAPTER NINETEEN

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 PDers, 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.\(^{15}\)

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 PDers. When the

\(^{15}\) 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.
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 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 PDers, 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 PDers 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.\(^\text{16}\)

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 PDers. 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 an 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.

\(^{16}\) 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.”
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.)

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.\(^\text{17}\)

**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.

---

\(^{17}\)This footnote is directed to those people who worked with the 5\(^{\text{th}}\) 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.

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.
**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” 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.\(^\text{18}\)

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.

**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

---

\(^{18}\) 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.
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, ironically enough, 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, false delicacy 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. 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 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

---

19 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 antianxiety, and most of the Parkinson’s drugs, are not able to ascertain when they are receiving a “go away” signal.
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.
Chapter Twenty

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 PDers 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 PDers 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.

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.

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.
**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 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.

---

**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.

---

---

21 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.
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 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, 2\textsuperscript{nd}) 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 (1\textsuperscript{st}) Cuneiform  
F. Intermediate (2\textsuperscript{nd}) Cuneiform  
G. Lateral (3\textsuperscript{rd}) Cuneiform  
H. 1\textsuperscript{st} Metatarsal  
I. 2\textsuperscript{nd} Metatarsal  
J. 3\textsuperscript{rd} Metatarsal  
K. 4\textsuperscript{th} Metatarsal  
L. 5\textsuperscript{th} Metatarsal  
M. 1\textsuperscript{st} Phalange 1\textsuperscript{st} toe  
N. 1\textsuperscript{st} Phalange 2\textsuperscript{nd} toe  
O. 1\textsuperscript{st} Phalange 3\textsuperscript{rd} toe  
P. 1\textsuperscript{st} Phalange 4\textsuperscript{th} toe  
Q. 1\textsuperscript{st} Phalange 5\textsuperscript{th} toe  
R. 2\textsuperscript{nd} Phalanges
The Bones of the Foot
Fig. 14.1

BACK TO PRACTICING

Another possible place to position your hands is with one hand under the calcaneous (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/calcaneous 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 calcaneous. 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 calcaneous. 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 metatarsals 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.\(^2\)

**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

---

\(^2\) 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.
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 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 to10 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.  

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.

**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:

---

23 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.
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. 14.2.)

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. 14.3.)

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.
A straight line from leg to toes
Fig. 14.2

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. 14.3
Outward rotation of the foot
If the foot is not relaxed, it might:

1. Not straighten out (Fig. 14.2), but will remain at more or less of a right angle to the tibia.

2. Instead of rotating laterally (Fig. 14.3), 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 Fig 14.4 and 14.5). 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. 14.4
Moderate hammer toe of the big (first) toe

Fig 14.5
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 proverb

CHAPTER TWENTY-ONE

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.

---

24 The saying and the attribution “Spanish proverb” was taken from a postcard printed by the Tushita postcard company, http://tushita.com/tibetimage.
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.

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.

“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 PDers.

*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.”

Ironic

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

25 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.”
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 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 PDers. 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 Poultice. 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 PDers 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 PDers 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.”

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.

---

26 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 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.
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 twelve, 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 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 thirteen. 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. 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.

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.

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.

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
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 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

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.
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,
ablet 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’ts

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.  

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.

---

29 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.

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.
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 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.
CHAPTER TWENTY-TWO

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 holdee 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.\footnote{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.”} Where the mind’s attention is focused, there the life force and healing energy of the body is likewise focused.

**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

\footnote{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 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.
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. The blood vessels themselves may become twisted and then hold onto that twist.

**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.

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

---

31 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.

32 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.
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, but the love of the cosmos. The perfect sense of unity and cosmic love is both personal and impersonal, with no sense of obligation.33

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

---

33 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.
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 the injured person, a simple repositioning of the broken bones is all that is necessary for healing to commence.
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 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.\textsuperscript{35}

Then again, \textit{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 please.

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 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 \textit{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 – \textit{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.

\textsuperscript{35} 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.
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.