

PART I

THE CAUSE AND CURE OF
PARKINSON'S DISEASE

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

— James Parkinson, 1817¹

CHAPTER ONE

PARKINSON’S DISEASE: UPROOTING THE CAUSE

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

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

MINDING MY OWN BUSINESS

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

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

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

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

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

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

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

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

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

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

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

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

A small pilot study

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

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

Control group

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

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

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

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

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

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

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

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

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

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

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

A Breakthrough

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

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

Study conclusion

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

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

On the outer (lateral) section of the leg, a downward current is the normal pattern described in Asian medicine. But the PDers all had currents running from the foot *upward* along the outer leg. The non-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.

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

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

Staying busy

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

Unexpected changes in response to simple foot holding

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

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

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

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

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

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

GROWTH OF A PROJECT

Results from afar

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

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

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

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

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

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

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

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

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

Why me, Lord?

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

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

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

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

The emotional component

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

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

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

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

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

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

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

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

JUMPING AHEAD TO THE PRESENT – THE YEAR 2006

An unhealed foot injury combined with a fear-based attitude

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

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

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

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

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

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

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

Electrical confusion in the area of the foot injury

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Backwards-flowing energy: a perfectly normal thing

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

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

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

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

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

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

Healing sleep

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Backwards flowing currents can be very, very bad

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

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

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

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

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

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

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

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

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

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

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

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

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

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

IN CONCLUSION

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

