This is the first eight chapters of the newest *Recovery from Parkinson's*.

This is a preview version of the upcoming edition. Finish date is projected as late 2019 or early 2020. The accompanying book, *Stuck on Pause*, should be finished by late 2020.

I will be making updated postings throughout 2019. Therefore, please do not make copies of this preview edition for friends or post this copyrighted material on a website. Instead, please refer any interested party to PDRecovery.org so that they can get the most up-to-date version of this book. Thank you.

Please ignore the many internal memos such as "xxx" and the place-saving designs that help during the construction of a book. Also, rest assured that editors and proof-readers will have their way with this material before it is presented in its final form.

This preview edition is being posted, even though it is a bit raw and unfinished, so that people with Parkinson's can have their questions answered with the most up-to-the-minute information.

Recovery from Parkinson's

Dr. Janice Walton-Hadlock, DAOM

Illustrations by Ben Bateson

Recovery from Parkinson's – first 8 chapters

Dr. Janice Hadlock, DAOM, LAc
Illustrated by Ben Bateson

Other books by Janice Hadlock available at www.JaniceHadlock.com:

Hacking Chinese Medicine
Tracking the Dragon*
Yin Tui Na: Hands-on therapy for traumatic injury *
Stuck on Pause *
Medications of Parkinson's or Once Upon a Pill: Patient experiences with dopamine-enhancing medications and supplements * **

*Also available for free download at www.pdRecovery.org.
** Not available in hard copy

Published by Raja Books, Santa Cruz, CA
www.JaniceHadlock.com
Fifth edition (heavily revised) 2019

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"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." [1]

James Parkinson, 1817

Chapter One

A curable illness

Idiopathic Parkinson's disease is not – and never has been – an incurable illness.

I saw my first Parkinson's patient in 1996. I treated her for her foot problem, not her Parkinson's. After all, everyone in the field of medicine knows that Parkinson's is incurable. When she unexpectedly recovered, I logically assumed she had been misdiagnosed.

This book tells what happened after that. More than twenty years have passed since that first patient recovered. I've worked closely with hundreds of Parkinson's patients. Hundreds more have conferred or contributed observations via emails. For over two decades, my search for answers about Parkinson's disease bordered on obsession.

I'll start this book with the answers.

What causes idiopathic Parkinson's disease?

Idiopathic Parkinson's disease (but not drug- or toxin-induced parkinsonism) is a collection of symptoms that are set in motion by the long-term use of a highly specific bio-electric schematic: a specific pattern in the electrical currents that flow in the connective tissue just under the skin.

This particular electrical configuration is only supposed to run when a person is in near-death shock or coma.

In people with Parkinson's, the currents flow in this near-death pattern all the time. In many people with Parkinson's, the currents have been running this way since childhood.

In schools of Chinese medicine, these *paths* of the currents are referred to as channels. The electricity flowing along these paths is called "channel qi." The word qi means "energy" and is pronounced chée-ee.

In this book, sometimes I will refer to these currents as channel qi, sometimes as electrical currents. Feel free to call these currents whatever you want: electrical currents work the same way in English as they do in Chinese.

The currents of channel qi can be easily felt by hand. Most people can learn to feel these electrical currents by hand after a few weeks of training.[2]

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[1] Parkinson J; *An Essay on the Shaking Palsy*; Sherwood, Neely, and Jones; London; 1817; page 56.

[2] I teach a class in feeling these currents, at Five Branches acupuncture college in Santa Cruz, California. I wrote a textbook for the class, *Tracking the Dragon*. The textbook can be used as a learn-on-your-own course for anyone wanting to learn how to feel the currents in the sub-

Very early on in my Parkinson's work, I noticed that the channel qi in people with Parkinson's was running backwards in one of the channels on the leg: the Stomach channel. I only noticed that other currents were also behaving strangely several years later.

I also observed that, when the old, unhealed foot injuries of my first few Parkinson's patients recovered in response to the gentle hands-on therapy that I was using, all the channels began flowing in the correct direction, and stayed that way. The symptoms of Parkinson's ceased and never returned.

At that time, I naturally assumed that straightening out the aberrant channel qi by fixing the foot injury had gotten rid of the Parkinson's symptoms. Therefore, this would be an effective treatment for *everyone* with Parkinson's. I was wrong.

Now, knowing that there are four ways to lock in the electrical currents that cause idiopathic Parkinson's, I can say that when these currents are turned off, in response either to therapies that turns off the neurological mode of near-death shock *or* therapies that encourage healing of an injury that is causing the body's currents to run in patterns very *similar* to those of near-death shock, Parkinson's ceases.

Long term use of near-death mode

In healthy people, the near-death electrical pattern usually only kicks in for a very short duration. It can be triggered by excessive loss of blood, excessive perforation of the skin, concussion, or other *near-death* types of severe shock-inducing trauma or coma. In this context, the word "shock" does *not* refer to surprise, fear, or the type of "shock" associated with post-traumatic stress disorder: responses that activate the use of sympathetic mode, also known as "fight or flight" mode.

The unhealthy, *long-term* use (stopping only during sleep – maybe) of the electrical-current patterns that should only occur during *near-death* shock or coma can be activated in four different ways.

Four corresponding therapies can turn off the electrical currents characteristic of this mode. Two of the therapies involve altering some mental habits. Two of the therapies are physical and directed at old injuries. The type of mental event or injury that triggered either the long-term use of this mode *or* an injury-based electrical simulation of it determines which therapy needs to be used to turn it off.

All of my hundreds of patients with Parkinson's disease had used one or more of these four activation methods.

I have seen people with Parkinson's completely recover after using the appropriate method for turning off the electrical currents typical of near-death shock.

The normal sequence of steps for turning off near-death mode

1) When an otherwise healthy person's body has physically stabilized following a near-death trauma, when the likelihood of death is no longer *imminent* (meaning in the

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dermal connective tissues. The book is written for the general public and does not presume any medical background.

The chapters with instructions on how to feel channel qi and the appendix with the maps of the channels are available for free download at www.PDRecovery.org, the website of the non-profit Parkinson's Recovery Project. Click on Publications, then click on *Tracking the Dragon*.

next *few minutes*), the person's body resumes somatic feelings (sensations inside the body) of being biologically stable and thus *internally* safe. At this point, the body might begin to tremor, either visibly or internally.

A specific type of tremor can be a normal part of the process for coming out of near-death shock. The type of tremoring performed while coming out of near-death shock seems to serve as a query directed to the brain: "Hey brain! I'm on the verge of coming back to life! So tell me, is the coast clear?" or "Is it safe enough out there for me to come back to life? To come out of the paralysis of near-death shock?"

2) The brain, using input from eyes, ears, smell, and touch, determines if the vicinity is safe, or at least safe enough to come out of the near-death mode.

If the immediate vicinity seems safe enough, the body will then perform three physical moves that turn off this mode and re-start the more normal awake-time modes: sympathetic (fight or flight) and parasympathetic (curious and playful). The moves include 3) taking a slow, deep, *audible* breath, 4) bobbling the head left and right very gently at the top of the neck, and then 5) allowing a shimmy to travel down the spine. Five steps in all.

The latter two moves turn back on the vagus nerves and spinal nerves, respectively. This reinstitutes the normal, waking-hours blend of parasympathetic and sympathetic mode channel qi schematics, which then trigger the release of neurotransmitters, thought patterns, and cellular and organ behaviors appropriate for these modes. The physiological behaviors of near-death shock mode then cease.

The near-death mode is not recognized in western medical theory, but it is in ancient Chinese medicine. I have taken the liberty of naming it "pause mode" [1]

This five-step sequence for turning off pause mode is *not* all that unusual. You might have done it yourself.

If you have ever found yourself shaking or trembling after a swim in an icy mountain lake (which triggers a blend of pause and sympathetic modes) or following on the heels of some intense, pause-inducing trauma, you might recall this sequence. It starts with a realization such as, "Hey, I'm gonna be OK, after all" and/or maybe, "I don't *need* to be shaking anymore."

This thought is followed by a deep, audible breath like a sigh of relief, a subtle head wobble, and a shimmy or shiver up or down the spine that almost feels like hitting an internal reset button. This sequence might be familiar to you...if you don't have Parkinson's.

Most of the people I've casually asked about this sequence recall performing it in response to the brief tremoring that might follow a shock, trauma, or full-body anesthesia. But many of my Parkinson's (PD) patients had no idea what I was talking about when I described the basic sequence for coming out of pause. Many even felt that it should be *impossible* for the brain to think that it's *ever* safe enough to come out of a mode characterized by wariness and heightened risk assessment.

As one of my PD patients said, "Only an *idiot* could ever think he was *safe*."

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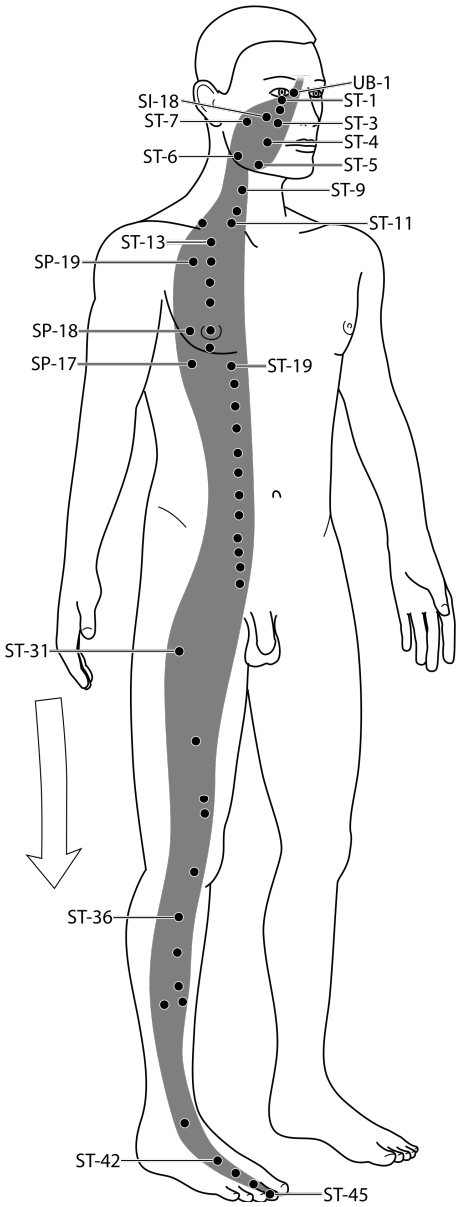
[1] Ancient Chinese medical theory recognized four neurological modes: sympathetic and parasympathetic plus the modes that are active in sleep and in near-death. Western medicine still only recognizes two, and those two only relatively recently, in the last two hundred-plus years. In the ancient Chinese medical text *Huang Ti Nei Jing*, pause mode is called "Cling to Life."

One of the channel qi alterations seen during pause mode – and in Parkinson's

In Chinese medical theory, each of the twelve "Primary" channels that flow just under the skin is named in honor of one of the organs. One of them, the Stomach channel, usually runs *from* the head *to* the toes.



When pause mode is activated, the Stomach channel qi flows *backwards*, from acupoint ST-42 on the foot up to ST-6, on the jaw.

Underlying muscles become rigid if the channel qi running over their surface is moving backwards.

In Parkinson's, the span from ST-42 up to ST-6 is one of the body sections along which muscles become rigid, pulling the neck forward, hunching the back, and tightening the muscles on the sides of the legs: part of the muscle behaviors that create the characteristic Parkinson's posture.

When running backwards, the Stomach channel does *not* flow up to the center forehead. Instead, it flows from ST-6, on the jaw, up alongside the ear to ST-8, at the hairline, and then towards the back of the head. If the Stomach channel runs backwards all the way up to the jaw, *no* channel qi traverses the face portion of the Stomach channel.

When the Stomach channel runs backwards, there is no channel qi flowing between ST-42, at the center of the foot, to ST-45, at the tip of the middle toe.

An *absence* of channel qi makes the underlying muscles become numb, cold, and/or limp. An absence of channel qi can allow the growth of fungus in the affected skin and nails.

The absence of channel qi in the span from ST-1 to ST-6 on the face and from ST-42 to ST-45 on the foot causes the characteristic numbness and muscle limpness – not rigidity – in these areas that you see in people with Parkinson's.

Fig. 1.1 The healthy, parasympathetic mode flow of the Stomach channel

On the face, the absence of channel qi can contribute to the seborrhea (subdermal fungal growth) alongside the nose that is not uncommon in Parkinson's.

On the foot, the absence of channel qi in the 2nd and 3rd toes contributes to both their immobility and to the severe toenail fungus often seen in those toes, and sometimes in the first toe, in people with Parkinson's.

In Parkinson's disease, people develop specific areas of muscle *rigidity* and *other* specific areas of numb, limp, or even non-functional muscles. These areas correspond perfectly to the areas in which channel qi flows backwards or is absent, respectively, during pause mode.

When the electrical patterns of pause turn off, people recover from Parkinson's. Healthy flow of channel qi resumes. Rigid muscles become soft. Inactive or atrophied muscles slowly resume tone.

When pause turns off, even the toenail fungus and seborrhea, if any, very often clear up on their own over the course of six months to a year.

These two pages were the merest of introductions to the Chinese channel theory that you'll get in this book. Other channels that are altered during pause mode will be introduced in later chapters.

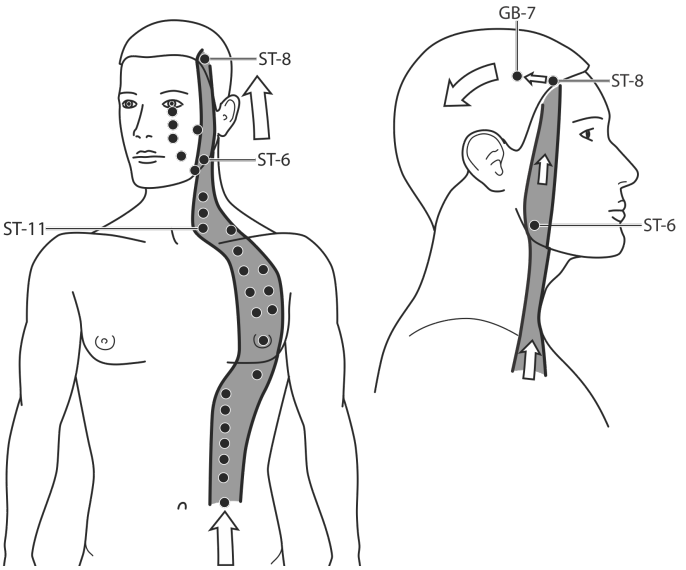


Fig.1.2 During pause, Stomach channel qi flows backwards and up to ST-6. When the channel qi arrives at ST-6, it is shunted up to ST-8 and then flows into the Gallbladder channel.

Compare the amount of energy flowing over the cheeks in fig. 1.1 and fig. 1.2. The absence of energy moving over the cheeks causes the expressionless "mask" face that can contribute to the characteristic look of Parkinson's disease.

The norepinephrine override

You might be wondering how a person is able to move around perfectly normally if he has been using the somewhat immobilizing mode of pause for decades, sometimes even since childhood. This question takes us back to western medicine.

When a person or animal is on pause, his release of brain-based dopamine for motor function *and* his release of adrenaline from the adrenal glands, located next to the kidneys, are both inhibited.

However, if an animal or person on pause is still conscious and *needs* to move for some reason of extreme emergency such as the injuring predator now moving towards the

victim's helpless offspring, the injured animal or person *will* be able to activate motor function. This motor function is very likely activated with neural norepinephrine. The word neural here means "in the brain." [1]

In nearly all of my PD patients, their symptoms began to appear when some life challenge that had long been used as a mental spur was finally laid to rest: the youngest child finished college; the mortgage was paid off; the predatory uncle died.

The reason their Parkinson's symptoms appeared is *not* that their dopamine levels had dropped. We now know that people have more than enough dopamine at the time they are first diagnosed with Parkinson's. Chapter three xxx will introduce the research showing that dopamine amounts are more than adequate in people with Parkinson's at the time they are first diagnosed.

People with Parkinson's haven't *used* dopamine for motor function for decades – sometimes since childhood. When they recover, most of them are shocked and a bit giddy because of how utterly foreign it feels to use spontaneous, (usually called "automatic") dopamine-based movement as opposed to the conscious, you might say "word-based" or "mental command-based" movement that they have used for decades.

For example, one recovered patient recalled how, at age seven, a classmate asked her how she was able to run so fast. She had replied, "I tell my arms to move back and forth as fast as I can, and the legs have to follow."

Some forty years later, when she recovered from Parkinson's, this habitually stoic woman told me how she burst into tears the first time her body used dopamine-based movement to get up from the sofa.

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[1] "Norepinephrine loss produces more profound motor deficits than MPTP treatment in mice"; K.S. Rommelfanger, G.L. Edwards, K.G. Freeman, et al; *Proceedings of the National Academy of Sciences of the United States of America*; 2007 Aug 21; 104(34):13804-13809.

Published online: 2007 Aug 16. Doi: 10.1073/pnas.0702753104.

In this study, mice brains' dopamine receptors were chemically inhibited using MPTP, a synthetic opioid. Instead of exhibiting the expected Parkinson's-like behaviors, the mice still had what appeared to be normal motor function. Only after the norepinephrine receptors were then chemically inhibited did the mice show the "poverty of movement" (extreme slowness and stiffness) also seen in people with Parkinson's disease.

*An introduction to norepinephrine***:** norepinephrine and adrenaline are structurally related.

In the blood stream, norepinephrine, a neurotransmitter, is released continuously from certain nerves at low levels to maintain blood pressure, among other jobs. Adrenaline, the "fight or flight chemical," is released from the adrenal glands, right next to the kidneys, during times of stress. Adrenaline increases heart rate and opens the bronchia (windpipes).

In medicine, norepinephrine is used to increase or maintain dangerously low blood pressure. Adrenalin, also known medically as epinephrine, is used to treat allergic reactions and asthma. Adrenaline usually acts as a hormone, but sometimes, when released by some nerves, behaves as a neurotransmitter.

Neurotransmitters do not easily cross the selective, semi-permeable membrane known as the blood brain barrier. The body supply and the brain supply of neurotransmitters are kept apart unless the bloodstream levels are *extremely*, unnaturally high, or there is a health problem affecting the blood brain barrier's integrity.

Neural norepinephrine and neural dopamine are produced and used in the brain.

Her tremor had already mostly stopped some days earlier. She was sitting on the sofa when she had the thought, "I should go into the kitchen," and the next thing she knew she was standing up and moving towards the kitchen *without* having mentally instructed herself to stand up.

Sobbing, overwhelmed with self-pity and relief, she exclaimed to the empty living room, "Is *this* how *easy* it's always been for everyone else?"

Based on patients' amazed reactions to using dopamine after turning off pause, it seems *very* probable that most of my patients with Type I Parkinson's have long used some alternative, non-dopamine neurotransmitter system for motor function. Then again, the people with Type II and Type IV Parkinson's have not been as surprised by the sensations when they resume using dopamine for motor function.

The four types are detailed in the next chapter.

In the early years of my research, I assumed the neurotransmitter that people had used instead of dopamine was adrenaline. That assumption was based on my use of the western medical theory that there are only two neurological modes.

But during recovery, after turning off pause, some of my patients had to relearn how to activate their adrenal glands. The rush of adrenaline felt just as foreign ("Scary!" "Animal-like!") as did the use of dopamine.

Based on research studies such as the one with mice that was footnoted on the previous pag, I hypothesize that the brain's norepinephrine system is what my patients had been using for motor function during all the years that they were stuck on pause.

Based on what I've heard from my PD patients, neural norepinephrine, if that's what they were using, enables a person to not only keep moving, but moving with abnormally heightened motor function.

Many of my PD patients had been top athletes or ace pilots or captains of industry – roles that required an almost super-human ability to always be tireless, as well as stronger, smarter, and faster than everyone around them: emergency behavior. Not all my patients, but still, *so* many.

During the pre-Parkinson's years, if a person who is stuck on pause can summon up a *constant* sense of emergency or intensity of purpose, he will be able to have what appears to be fairly normal, or even *superior* motor function in spite of being on pause. However, this type of motor function is triggered by a conscious process, a process very different from the one that drives "automatic" or unself-conscious, dopamine-based movement.

Most healthy people are always using a blend of two types of movement: dopamine-driven and adrenaline and/or norepinephrine-driven. At any given moment, the ratio of the blend depends on how relaxed or how uneasy they are.

I hypothesize, based on patient histories, that eventually, when the ability to rouse or sustain a mental sense of constant emergency *diminishes*, the ability to mentally activate the emergency norepinephrine override for pause also declines. *That's* when their long-hidden, pause-like symptoms that we call idiopathic Parkinson's begin to manifest.

Dopamine had been present in their brains in high enough quantities, until the PD became quite advanced. In fact, in people with Parkinson's, dopamine levels are *higher* than normal in the right anterior cingulate part of the brain, an area used for risk-

assessment, among other things. Dopamine use for *motor* function is inhibited during pause. Dopamine use for risk assessment is *increased* during pause. [1]

People with Parkinson's, for various reasons, are stuck in the electrical patterns of a neurological mode that can only be turned off 1) when the biological threat from the trauma, if any, has stabilized and/or 2) the person is willing and able to once again feel safe, or at least safe enough to turn off this mode.

Most of my patients with Parkinson's had an unhealed foot injury causing pause-like electrical flow *or* had gotten stuck on pause and never felt safe enough to come out of it *or* they gave themselves a command – often in childhood, often while staring into a mirror – of something along the lines of "Feel no pain."

The body *does* have a neurological mode in which it is numb to several kinds of physical and emotional pain: pause mode – the mode associated with near-death trauma and coma. When a person using great mental focus and grim determination commands himself to feel no pain, his body might very well obey. It might shift into the near-death neurological mode of relative numbness…and stay there.

I refer to pause mode that is triggered in *this* fashion as "self-induced pause."

PD from self-induced pause is by far the most common of the four types of idiopathic Parkinson's disease, presenting in nearly ninety-five percent of my PD patients. I've named it Type I Parkinson's disease.

Many people with Type I Parkinson's do recall giving themselves some such command. Many who do *not recall* doing this nevertheless say that they embrace numbness, or something like "going into a dead place inside," as their way of dealing with unpleasantness or negative emotions.

Type I Parkinson's has behavioral patterns not seen in the other types of PD. So even if a person with Type I PD doesn't *recall* any sort of self-instruction, this type is fairly easy to distinguish. This book has instructions for diagnosing the four types.

Whether or not a person *remembers* instructing himself to become numb or some other command that inadvertently induced pause mode, *if* this command is in place, the person will *not* be able to terminate the use of this mode until he makes some specific mental changes.

In addition to the Type I self-activation of pause mode, there are three other far less commonly used activation methods that can inadvertently lead to getting stuck with pause-like electrical patterns.

In total, there are four ways to get *stuck* in the electrical patterns of near-death mode. And four ways to turn them off.

So if you or a loved one have been diagnosed with Parkinson's disease and are *not* taking dopamine-enhancing medications, be of good cheer. It is *not* an incurable illness.

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[1] "Personality traits and brain dopaminergic function in Parkinson's disease"; *Proceedings of the National Academy of Sciences USA* 98:13272-7*;* Valtteri Kaasinen, MD, PhD et al; 2001.

This 2001 study, published in one of the most respected journals in American science, describes the utterly unexpected discovery that people with Parkinson's have *elevated* levels of dopamine activity in the brain's anterior cingulate area.

Note: Before going any further, I want to make clear that modalities of Chinese medicine such as acupuncture, Chinese herbs, and moxa (smoking mugwort leaves) are *not* used in the treatment of Parkinson's disease. I'll discuss this more, later, but I want to mention this nice and early, before you rush off in search of the nearest acupuncturist in response to my references to Chinese medicine.

Also, your acupuncturist probably won't know anything about pause mode.

We do *not* learn about pause mode in Chinese medical school.

More than ten years into my Parkinson's research, I stumbled across this neurological mode in a translation of the most important ancient tome of Chinese medical theory, the *Huang Ti Nei Jing*.

The only reason I understood immediately what the garbled language was discussing was that it answered so many of the questions I had accumulated in my years of researching Parkinson's…and I had acquired a *lot* of questions.[1]

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[1] Here's an example of what I mean by "garbled language," followed by my translation using a more contemporary English.

"Change of colors corresponds to pulses of the four phases, which is valued by gods because it is in tune with the divine being and which enables us to flee from death and cling to life."

From *Su Wen*, chapter 13-9, from *A Complete Translation of the Yellow Emperor's Classics of Internal Medicine and the Difficult Classic*; Henry C. Lu, PhD; published by the International College of Traditional Chinese Medicine; Vancouver, BC, Canada; 2004; p. 116.

The word "colors" in this context is a very loose – and incorrect – translation from the Chinese characters for *Qi Se*, literally "energy from light, or energy from light waves" (electricity) and is a reference to channel qi, but is often translated into English simply as "colors". The word "pulse" in this context refers to "changing behaviors."

A quick translation into medical English would read, "Changes in the electrical schematics of the channel qi correspond to the physiological changes of the four modes. The first mode, parasympathetic, is "in tune with the Divine being" (joy and ease). The second, sympathetic, enables us to flee from death (fight or flight). [The third is not listed in this quote, but it corresponds to sleep mode.] The fourth, pause mode, allows one to cling to life.

The English- and Chinese-speaking practitioners of Chinese medicine that I've spoken to have had no idea that this section of the *Huang Ti Nei Jing* is discussing neurological modes.

Having no basis for translating this section, they have usually guessed that this sentence from the thousand-plus-years old medical tome is a random interjection about natural history, something along the lines of "Plants change color during the course of the four seasons."

The rest of the chapter that follows this introductory sentence makes no medical sense if this chapter is about leaves changing colors in the autumnal months. The rest of the chapter makes stunning good sense if one recognizes from hands-on experience that the channel qi does flow differently in each of the four neurological modes.

Since the Communist revolution in China, channel qi is regarded as an historical superstition, similar to religion and any classical references to the Divine. Teaching channel qi as a medical reality has been illegal in China for more than half a century. This has led to some truly bizarre translations of the old medical books into modern, atheistic Chinese and into English. The

Not only are the modalities of Chinese medicine not helpful in treating Parkinson's, professional help of *any* type is usually not necessary.

Recovering from Parkinson's is pretty much a do-it-yourself or a do-it-with-a-friend project.

This book will explain how to do it.

political reasons for this are discussed in greater depth in some of my other books, including *Hacking Chinese Medicine* and *Tracking the Dragon*.

As with much of the classical Chinese literature, one needs to already know most of what is being discussed in order for the cryptic, terse language to be intelligible. The ancient writings were never meant as explicit guides for the general public. Rather, they were insider references for a primarily oral, closely guarded tradition.

Chapter two

Pressing questions

The most pressing question for many people is "How long will it take for me or my loved one to recover?"

The answer is, it varies. You'll have to learn what all the variables are and then see where your own case fits in.

The most important variable is what type of Parkinson's a person has. So this chapter will start with that. But other variables can also be significant.

Then again, my readers who are doctors of any type of medicine usually have completely different questions: Can your hypotheses be proven? How can I help my own patients with Parkinson's? and How dare you give my patients false hope?

I hope to answer as many questions as I can for all types of readers.

Four ways to develop idiopathic Parkinson's disease

I have found four reasons that people get stuck in the electrical configurations that are associated with pause mode. Of course, there might be more. I've numbered them in order of how common they have been in my experience.

Type I: self-induced pause
Type II: being *stuck* in normal, biological dissociation from a foot or ankle injury
Type III: self-induced dissociation from an injury
Type IV: being *stuck* in normal, biological pause

Type I PD: self-induced pause

This type of pause is usually set in motion via a self-command, often given in childhood, to "feel no pain," "be spiritual: rise above pain," "I am not a part of this painful world" or some similar instruction commanding that one distance oneself from physical or emotional pain or trauma. The command was *not* a casual remark. It is often memorable for the intensity and determination that was evoked. As mentioned earlier, it was often performed while staring into a mirror.

This type is by far the most common. Nearly ninety-five percent of my hundreds of PD patients have had this type of Parkinson's disease.

This type does *not* respond to the five basic steps that turn off *normal* pause. A completely different set of techniques is involved in turning off self-induced pause.

Recovery from this type usually takes longer than recovery from the other three types. The time frame for recovery from this type ranges from a few days to many years.

The length of time is related to the degree to which a person has developed a steady habit of a fear- or anxiety-based internal monologue and/or cultivating a habit of imagining himself to be apart from the rest of humanity, or even alone in the universe. In general, the more a person has embraced wary and/or judgmental thinking, the longer it might take him or her to recover.

I'll go into details about this type in later chapters, but first I want to give a quick overview of the other types. (If you notice that percentages vary slightly from previous editions of this book, it's because I've seen more patients since then.)

Type II PD: dissociation from an injury

This is the second most common cause for Parkinson's. People with this pattern have usually dissociated from a foot or ankle injury. The dissociation was *not* consciously activated. Instead, the dissociation that can automatically occur in response to a significant injury *until* the person finds himself in a safe place has simply never turned off.

The automatic dissociation behavior, held in place until safety is attained, might be demonstrated by a small child or toddler who hurts himself and then wanders through the house looking for his mother or some other source of comfort. As soon as he sees someone who can help him, someone he trusts, he bursts into tears.

The mother or comforter might say, "It couldn't have been too bad, you didn't even start crying until you found me…"

This statement reflects a lack of understanding. The injury might have been significant, but until the child knows that he is safe, he will be able to do what he needs to do – look for mom or some other source of comfort and safety – with minimum recognition (automatic dissociation) of the pain.

I have seen more than a hundred *non*-Parkinson's patients who are still dissociated from some old injury or surgery: this is a main reason behind a chronic pain, a "failure to heal" or, in the case of broken bones, "failure to knit."

In many cases, as soon as the patient's subconscious mind is brought to dwell on the problem area, the area begins to heal. Very often, when the long-sustained micro-muscle tension in the area finally loosens up, deep, full healing begins. The area might finally manifest its long-repressed swelling and/or bruising (become black and blue).

Because of the specific electrical schematics in the foot and ankle, a long-unhealed (unhealed because still mentally dissociated) injury in this area can cause the electrical disarray from the injury to grow larger over time. Eventually it can create so much resistance in the foot that an electrical current – usually the Stomach channel current – begins to flow *backwards*, just as it does during pause.

Over time, usually decades, the domino effect of channel disturbances from the unhealed injury sets in motion electrical behaviors – and therefore physical symptoms – that are nearly indistinguishable from those caused by pause mode.

This is the type of Parkinson's disease that might spontaneously, permanently disappear in response to Qi Gong or other movement disciplines in which mental focus is used to direct energy throughout the body – even to areas that might unknowingly have been dissociated.

When you read online about people whose Parkinson's went away in response to Qi Gong, visualization, a positive affirmation, or hands-on support therapies, these recoveries most likely occurred in people who *only* had Type II.

People with only Type II tend to turn off the Parkinson's behaviors fairly quickly, in a matter of a few weeks or a few months, in response to appropriate treatment.

This type of Parkinson's is the second most common. I have seen *only* Type II PD in nearly five percent of my patients.

The one-two combination

I have seen Type II *combined* with Type I in nearly ninety percent of my patients.

This combination, I hypothesize, might be so common because if a person is stuck on pause, whether the normal type or self-induced, any subsequent injuries *must* be dealt with via dissociation, until such time as pause turns off.

To understand this, consider an animal that is on pause and appearing nearly lifeless due to some sort of attack. The animal should *not* be responsive to ongoing *or* subsequent injury or pain. Being responsive might cause the animal to give itself away to, or re-excite, a nearby predator. An animal on pause automatically dissociates from his injuries until 1) he turns off pause and then 2) gets to a safe place.

After pause turns off *and* the animal then gets to a safe place, he will automatically become painfully aware of, you could say re-associated with, his injuries.

Once he is in a safe place, the animal might tend to his injuries by licking them, coddling them, or allowing another animal to lick them.

Being *stuck* on pause can cause a person to *stay* dissociated from his subsequent injuries. Add to this that that nearly everyone bangs a foot at one time or another.

Also, some patients have recalled giving themselves the original instruction to feel no pain in direct response to a painful foot injury. This means that a foot injury, if any, might stay unhealed either because the person was *already* on pause *or* because the person *responded* to the foot injury by commanding himself to feel no pain: to go into self-induced pause.

Therefore, finding dissociated, unhealed foot and ankle injuries contributing to the pause schematics in a person who also has self-induced pause is *not* surprising. Most of my patients with Type I PD also had Type II. In these cases, Type I is the deeper problem and **must** be dealt with first, before dealing with the foot or other injuries, in order to prevent the nightmare of partial recovery – which will be described in great detail later on xxx.

Type III PD: self-induced dissociation from an injury

In some cases, a person has commanded himself to "ignore the foot injury," "pretend the injury never happened," or even "I don't have a foot."

In these cases, the person commanded himself to not pay attention to the specific foot or ankle injury.

This is *very* different from a pause-inducing command in which a person tells himself to feel no physical or emotional pain, period. In self-induced dissociation from an injury, the person has commanded himself to no longer recognize a *specific* injury event or a *specific* body part.

I repeat: in self-induced *pause*, the person has commanded himself to be numb, period. The exact wording might vary, of course. Very often, the person did not use the word "numb" but instead used words like "rise above my feelings" or "be more spiritual: don't be affected by pain." But the intention was nevertheless to obtain a body-wide condition of being impervious to physical and/or emotional pain.

In self-induced *dissociation*, the patient has only compartmentalized one or a few events or body parts away from normal consciousness.

I have seen self-induced dissociation from an injury in dozens of *non*-PD patients. I have seen it in one person with idiopathic Parkinson's disease.

He told me how twenty-five years earlier he had dissociated from a foot injury that wasn't healing up fast enough. He was in college at the time and "…couldn't be bothered with this pain," so he powerfully commanded himself to not feel his foot. After making this command several times over the course of one day, the pain abruptly ceased.

He gave a rueful chuckle, "I was so proud of myself."

Recovery from this type might take a few weeks longer than Type II because destroying the mental instruction for dissociation might take a week or two.

Type IV: stuck in biological pause.

Being stuck on pause is a *somewhat* uncommon condition, but over the years I have seen it in quite a few patients. I discuss a sampling of these cases in my book *Stuck on Pause*. I have seen it in *one* person with idiopathic Parkinson's disease.

She had never *commanded* herself to be numb. She didn't have a foot injury.

Twenty years earlier, she had experienced a severe concussion from a blow to the side of her head. She had been in a coma for three days. After she regained consciousness she had quickly recovered her brilliant mental acuity. However, based on the pause-type flow of her subdermal electrical currents when I met her nearly twenty years later, her bio-electrics had *not* come out of the coma event.

Her symptoms had been slowly developing over the years and, a few years before I met her, she had been diagnosed with idiopathic Parkinson's disease.

Although this is an introductory chapter, I will go into a few details regarding her case because they demonstrate how pause mode and its termination, even with *no* mental components, relate to causing and turning off Parkinson's disease.

Case study #1: normal, or "biological" pause

When I met this patient, she had all the symptoms of idiopathic Parkinson's including rigidity in her arms and legs, cogwheeling in her wrists and ankles, poverty of movement (moved slowly, needed help getting out of a chair), micrographia, faint voice, the classic Parkinson's hunched posture, a blank facial expression that was worse on the right side of her face, and a slowly worsening pill-rolling tremor, also on the right side. She had never taken antiparkinson's medications.

I did an extensive intake history – more than an hour. I always did this with my new Parkinson's patients or any patient that had what seemed like a complicated case. While asking about her medical history, I learned of her head injury.

Based on the head injury, I started her treatment by doing some craniosacral work on her head. Craniosacral therapy is a type of Yin Tui Na. Yin Tui Na is a generic Chinese term meaning slow, supportive holding. For people with Type II or Type IV Parkinson's, Yin Tui Na can help people re-associate with and/or recover from the injuries that are keeping pause-type electrical patterns in place.

I discovered that my patient's right-side parietal bone (on the upper side of the head) was jammed under her right temporal bone (surrounding the ear hole).

When I did the basic craniosacral protocol, one segment of which allows the parietal bones to move freely relative to the temporal bones and properly reposition themselves, she immediately – right there in my office – went through all the long-delayed behaviors that a person experiences when coming out of pause: the giddy realization that she was safe, the deep breath, the head bobble, the shimmy down the spine.

All of her most acute Parkinson's symptoms were reduced within minutes, some disappeared entirely.

Her very elderly mother, who had never been informed that her daughter had Parkinson's, was present at the therapy session in which the Parkinson's stopped.

Her daughter, my patient, sprang to a sitting position on the side of the treatment table, exclaiming over and over, "I'm OK! I'm OK! I'm *good*! I'm *good*!"

This gushing, radiant joy in a person who has just come out of pause can be absolutely normal – just ask any emergency room nurse.

As she was still exclaiming over how good she was, her mother said to me, "Finally! She's smiling again. I've been telling her for years to put a smile on her face, but would she listen to me? No. I guess she'd rather smile for you than for me."

After this session, her neurologist told her that not only had she been *mis*diagnosed with Parkinson's, but she must also have been neurotic for having *pretended* to have had Parkinson's disease.

Except for the ones who never went back to a neurologist, every one of my patients who recovered was subsequently told by the neurologist that he or she had obviously been misdiagnosed or else had merely had "psychogenic parkinsonism."

Psychogenic parkinsonism is a syndrome that looks exactly like Parkinson's, but it often comes on suddenly, following a severe physical or emotional trauma, and then goes away when the severe shock ebbs. It is often dismissed by MDs as being a purely mental phenomenon and not related in any way to the presumed brain deficiencies of idiopathic Parkinson's disease.

In fact, with the exception of the extremely rare Type IV PD, all cases of idiopathic Parkinson's disease are technically psychogenic, meaning that they have a significant mental component.

Some doctors who declared "misdiagnosis" when confronted with one of my recovered patients had then, because of recovery symptoms that can include sudden weakness in long-rigid muscles, whimsically changed the diagnosis to either multiple-system atrophy (MSA) or progressive supranuclear palsy (PSP) – two other movement disorders for which, like Parkinson's, there is no test other than autopsy. When the patient has fully recovered and is strong again, and a diagnosis of MSA or PSP is no longer appropriate, a typical MD response has been anger and accusations, or a diagnosis of psychosis.

According to western medical theory, and based on the *long*-disproved late-nineteenth century idea that brain cells and brain behaviors cannot ever heal or even change, Parkinson's, being considered a brain disorder is, by definition, incurable.[1]

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[1] Research over the last twenty years has shown that brain behaviors do change in response to usage or even create compensations in response to damage.

As for the idea that one can never grow completely new brain cells, new research suggests that not only is the brain able to grow new cells (neurogenesis), but that "impaired neurogenesis might be a potentially relevant mechanism underlying memory deficits in Alzheimer's disease."

The above quote was taken from the article "Adult hippocampal neurogenesis is abundant in neurologically healthy subjects and drops sharply in patients with Alzheimer's disease"; *Nature Medicine*; March 25, 2019; www.nature.com/articles/s41591-019-0375-9.

Getting back to the subject of my patients, I have only seen the above one case in which the patient was *biologically*, not mentally (self-induced), stuck on pause *and* was manifesting fully-developed Parkinson's disease. In this one case, she had never commanded herself to feel no pain. Her body was simply waiting for some help to dislodge the cranial-bone displacement that had put her into a three-day coma and which was still displaced twenty years later.

Recovery from Parkinson's caused by biological pause might take a few weeks longer than recovery from Type II, the type caused by merely an unhealed foot or ankle injury. In part, this is because a person who is stuck on pause might have *several* pause-causing injuries in the body that are keeping pause in place. It might take several weeks of once a week treatments to track down all the places in a person's body that are causing the person to be stuck on pause.

Very likely, the reason that I have only seen one example of a person with fairly advanced Parkinson's being *biologically* stuck on pause is because I have only seen a very limited number of people with Parkinson's: only a few hundred.

I am certain that, if I were to see several thousand people with PD, I would meet several who are biologically stuck on pause rather than having self-induced pause.

This brings up the subject of people who have long been stuck on biological pause and do not yet have Parkinson's. I *have* seen many patients who *were* stuck on pause and running the electrical currents of pause, who did *not* overtly appear to have Parkinson's. Yet.

However, *many* of them had some subtle and some not-so-subtle pre-Parkinson's symptoms, often including an internal tremor that ceased when pause was turned off. Remember, Parkinson's usually takes many *decades* to develop.

In *some* cases of people being stuck on biological pause, the injury was already healed. All that these people needed to do was initiate the normal steps for turning off pause: confirm that they are both internally stable *and* externally safe, take a deep audible breath, wobble the head high on the neck, and let a shiver run down the spine a few times, until they feel something shift inside.

You can read about many of these cases and the *pre*-PD symptoms of many of my pause patients in my book *Stuck on Pause*.

As for the speed of the above patient's recovery, that is typical.

When I say a person recovered from Parkinson's instantly, which is the most typical way to recover for all types of Parkinson's, I mean that the *driver* behind the symptoms of Parkinson's and the oppressive sense of impending doom were instantly gone. And didn't come back.

Following that instant change, people still had to go through the recovery symptoms: weeks and even months of overcoming the new weakness in muscles that had long been rigid, pins and needles in body parts that had long been numb or disconnected from proprioception, achy soreness in muscles that had been limp, un-usable, and atrophied and which were once again *automatically*, *unstoppably* being used for movements such as arm swing: symptoms that are the *opposite* of the symptoms of PD.

So it's not as if these people who no longer had Parkinson's could suddenly move as easily as they had twenty years earlier, with no repercussions from their years with PD. They had been through a war and needed to recover from it. But when I say "the

Parkinson's ended," I mean that the war itself was clearly, obviously, without a doubt, over.

Case study #2: a ten year old boy with internal tremor

For a quick example of a pre-PD symptom, I had a ten year-old patient with a shoulder injury from sports. I noticed his Stomach channel qi running backwards *and* an obvious displacement of bones in his foot that he could not account for. The displaced shoulder moved itself back into place very quickly. I asked his attending father if I might also work on the obviously displaced bones in his feet.

Before starting in, I asked the boy about certain pre-PD symptoms, including any feelings of agitation, shaking, or tremoring inside his head or body. He said no to every query.

He was an extremely articulate and intelligent lad, competed in league sports, played violin, and had just won a place on the student council in his grade school despite being younger than most of the also-rans. He struck me as having an intelligence and an articulate, focused personality very similar to that of many of my Parkinson's patients.

I treated him over the course of several, one-hour, once a week sessions until his foot bones moved themselves back into place. At the beginning of every treatment session I asked him if he had any internal tremoring. He always said no.

During the session of foot holding in which his foot bones finally slid themselves back into place, he remarked, "I just remembered when I hurt that foot. I was five or six years old. My mother drove over my foot with the car. I couldn't tell her; she would have been devastated. So I pretended it never happened."

After a few moments he spoke up again. "You know how you always ask me if I'm shaky inside? Well, I must have been, because it just stopped! It feels *so* good. It was always there. I just thought it was normal. Ahh. It feels *so* good now. It's so *still* inside. The shaking inside my head is completely gone!"

Based on his remarks about pretending that the specific injury didn't happen, he probably had Type III *pre*-Parkinson's.

Although some Type III cases might require an additional, medical Qi Gong type of healing technique, a person with a less advanced Type III case such as the person in the above case study might recover in full in response to just basic Yin Tui Na.

The book *Yin Tui Na: Hands-on treatment for traumatic injury* explains both the hands-on support and the medical Qi Gong techniques, and how to diagnose who needs what.

Case study #3: dropped as a baby

A woman in her late twenties came to see me because one foot was starting to drag. Her posture was starting to hunch forward. Her arms tended to stay crooked at the elbow. She had been very athletic, and felt that she was becoming "old before her time." She moved stiffly when she tried to turn over in bed.

I found several displaced bones in her foot. She had no memory of a recent foot injury.

I worked on her foot for several weeks before she remembered a very old injury that she had assumed was long since healed.

When she was around three months old, she was baptized in her parents' Greek Orthodox faith.

As the priest was about to dip her into the font, he dropped her. Before she landed headfirst in the water, the priest snatched her by her left foot and jerked her back up to safety, and then proceeded to immerse her in the cold water. A painful foot injury, followed by a shock.

Her grandmother, who had attended the ceremony, had told her several times over the ensuing years, "You were never the same after that day. You had been a very easy-going baby. After that day, you were always agitated, always guarded."

After her foot injury healed up in response to several Yin Tui Na foot-holding treatments, she had quite a few of the very specific recovery symptoms – symptoms that are the opposite of Parkinson's, that I've seen in people who've recovered from Parkinson's disease.

Whether she was dissociated from her injury or was stuck on pause, I cannot say. At the time I worked with her, I had no idea that it made a difference. Either way, her pre-Parkinson's symptoms cleared up quickly when the foot healed.

This case study and the one previous are provided in part to point out that a person can have many pre-Parkinson's symptoms and yet still not be diagnosable from a neurologist's standpoint. And remember, Parkinson's disease can take years, usually decades, to develop. Nearly all of my patients that were stuck on biological pause had *many* pre-Parkinson's symptoms. When they recovered, they usually went through some of the counter-intuitive symptoms of recovery from Parkinson's.

If a patient did *not* have an official diagnosis of Parkinson's disease, I *never* mentioned the possibility that he or she had dodged having Parkinson's in the future.

I merely did Yin Tui Na on the old, unhealed injuries or helped the patients go through the steps that turn off pause. Even if they had electric currents consistent with pause mode, even if they had subtle pre-PD symptoms *or* a few *glaring* Parkinson's symptoms, I just treated the injuries or health problems and never mentioned Parkinson's disease.

If the patient was surprised at how many other mild problems cleared up at the same time as the injury, I just suggested that lots of physical and mental behaviors might be connected to an old injury.

For me to act the prognosticator and tell these people who had been stuck on pause that, by turning it off, they probably prevented themselves from developing Parkinson's disease down the road would of course have been ethically wrong.

Even so, I am fairly certain that this was the case. In all their cases, turning off the electrical circuitry of pause mode – and their pre-PD symptoms – was simple, once they received Yin Tui Na treatment for the pause-inducing injury and/or let me coach them through the steps that turn off pause – steps that for various reasons they had never taken.

Looking ahead, if doctors of every school learn to recognize the electrical behaviors of pause mode and can direct their patients with this situation towards information on turning those behaviors *off*, then many or even most cases of Parkinson's disease might be prevented. Ideally, people with pre-PD symptoms might someday be able to avoid developing the more glaringly obvious symptoms of Parkinson's altogether if the injuries or attitudes that triggered pause and/or dissociation are treated in a timely manner.

Now, having given very brief, very generalized answers to the most common question, “how long will it take to recover?” and promising to revisit the question later with more specifics, it’s time to consider the second most common question: what about all those dead dopamine-producing cells in the brain?

I’m glad you asked.

Chapter three

Western medicine guesswork

The theory of the dead dopamine cells

Researchers in the field of Parkinson's have repeatedly disproved the hypothesis that idiopathic Parkinson's is caused by the inexplicable death of dopamine-producing cells.

That theory, dating back to the 1960s and concomitant with early research on L-dopa, was based in large part on a small group of illegal-drug users. They all developed a few symptoms of parkinson*ism overnight* because of presumed brain damage from a weekend binge using a type of synthetic heroin.

Their symptoms were *not* actually a good match for idiopathic Parkinson's disease, but because their immobility from sudden brain damage responded favorably to a mind-swamping level of L-dopa – the pharmacological equivalent to dopamine – in a manner similar to that of people immobilized with Parkinson's, it was concluded that Parkinson's itself must similarly be caused by some unknown factor that, like the synthetic heroin, destroyed dopamine-producing brain cells. There was *never* hard evidence for this quickly adopted theory.

At that time, dopamine was assumed to be a neurotransmitter that *relaxed* muscles. In order to make sense of observations of people with Parkinson's moving easily after receiving large doses of L-dopa, researchers assumed, wrongly, that the normal, passive state of muscles must be rigidity: dopamine was required to relax muscles, thus allowing for movement. Dopamine therefore was also wrongly assumed to induce sleep by creating a state of heightened relaxation.

Two corresponding "facts" at that time were that dopamine levels in the brain were 1) lower in the daytime and 2) higher during sleep. [1]

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[1] This is a lengthy footnote, but I hope you'll forgive me because it makes an important point about how hard it is to change a widely accepted, presumably "scientific," wrong hypothesis.

In the first article I ever submitted for publishing, I mentioned that dopamine levels were higher during awake times and lower during sleep. This was based on my very new, (1990s) understanding of the role played by dopamine in stimulant drugs such as cocaine and methamphetamine, and disagreed with what I'd learned in college.

The editor choked on my brazen contradiction of the long-known "facts" about dopamine.

She said I would need to show her at least one research article that showed dopamine levels being higher in the daytime than at night, if she was going to consider running my article.

Of the one hundred articles that I pulled up in my search, there were ninety-eight articles in which the subjects had *higher* dopamine levels in the daytime than at night.

In every one of these studies, the main subject of the research was not dopamine or Parkinson's disease, but was some other illness being studied. The night and day *blood* dopamine levels (not brain levels) had merely been checked as a part of the general blood work.

In every case, the researcher had noticed that, *contrary* to the pattern that was presumed to be normal, all of his or her human subjects had higher blood levels of dopamine in the daytime, lower levels at night. In each article, the researcher concluded that the unanticipated and obviously

These wrong "scientific facts" about dopamine from the 1960s were still in place in the 1990s, when I started my Parkinson's research.

Now, in 2019, we know that dopamine is the neurotransmitter of *consciousness* and expectations of joy. Not sleep. Dopamine release for motor function is greatly *inhibited* during both sleep mode and pause mode.

Nevertheless, based on the *wrong* idea that dopamine makes people sleep or relax, and the fact that people who were rigid with Parkinson's *could* relax and move freely when they took mind-smashing levels of L-dopa, the researchers claimed to have proof, however indirect, of their theory that the death of dopamine-producing brain cells causes Parkinson's disease.

The unproven leaps and guesswork came to their somewhat illogical conclusions in the writing of Dr. Langston, in his book *The Case of the Frozen Addicts*. This book described the small group of illegal-drug users who developed drug-induced parkinsonism. This book's premise then served as the basis for *all* Parkinson's research

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pathologically reversed dopamine levels might be contributing to the illness that was being researched. The various illnesses being studied included PMS, narcolepsy, epilepsy, mood swings, muscle cramping, mental retardation – the list went on and on. In every case, the researcher for each article had suggested that maybe the cause of the illness at hand was the pathologically elevated daytime levels of dopamine and decreased nighttime levels.

Since it was a recognized fact that dopamine is a relaxant (wrong), and is therefore present in higher quantities at night (based on the excess acetylcholine/ insufficient dopamine imbalance = excess rigidity theory of Parkinson's disease, also now recognized as wrong), every one of these studies concluded that the cause of the illness in question might be this reversal of the officially-correct dopamine pattern.

In other words, if the patients were in the headache study, the report proposed that this abnormal reversal of the correct dopamine levels was causing their headaches. Ninety-eight of the one hundred studies followed this pattern. It didn't matter what the researchers were looking at. The conclusion in *every case* was that the illness at hand might be related to the abnormal, reversed situation of blood dopamine levels being higher during the daytime and lower at night.

But, to be fair, there were two studies that had the opposite result. In these two studies they measured not *blood* dopamine levels, but the actual brain levels of dopamine. This was done by chopping off the heads of the subjects, tossing the heads in a blender, and quickly assaying the results. This gave the most accurate possible reading of brain dopamine levels.

In these studies, the dopamine levels were higher at night, thus confirming the fact of dopamine as a nighttime relaxant. There was only one detail that had evidently missed everyone's attention: the subjects in these two experiments were rats. Rats are nocturnal – they are active at night; they sleep during the day.

Armed with these research abstracts, abstracts that suggested that 100% of the time human and rat dopamine levels were higher during awake, active time and lower during sleep, my editor agreed to run my article.

This was in 1998. Hopefully, this footnote illustrates just how hard it is to defy the established facts, even when the "known facts" are unproven and *every* study produces data that *opposes* those so-called "facts."

Most of the current western medicine "facts" about idiopathic Parkinson's were never more than guesses, hypothesized in the 1960s and 1970s, when *very* little was known about brain function.

for nearly forty years...even though the "frozen" drug addicts in question did *not* have idiopathic Parkinson's disease.

In retrospect, these hypotheses were an embarrassing series of wild guesses that were primary used to explain why L-dopa, in mind-blasting amounts, was able to temporarily override *some* – though *not* all – of the neurological inhibitions of Parkinson's. Even so, many medical people of the day saw that the Parkinson's hypotheses were very shaky. Also, this new idea did not explain *many* of the characteristic symptoms of Parkinson's, such as some muscles being weak while others were rigid. It did not explain the tremor at all.

In 1998, one of my PD patients who was a senior neurologist at one of our top, university-linked medical schools confided in me, "We don't actually know anything about Parkinson's disease. We're just making stuff up."

He also told me that he would probably lose his seat on the school's neurology board if his colleagues found out he was seeing an acupuncturist.

Since then, even though Dr. Langston's hypotheses have been repeatedly disproven *and* even though we now know that idiopathic Parkinson's is *very* different from drug- or toxin-induced parkinsonism, his hypothesis remains at the forefront of the drug industry's push for new, "improved" dopamine-based drug treatments – not cures – for people with Parkinson's.

Research doctors in the field of Parkinson's, often far removed from the interface with actual patients, have known for years that the dead dopamine cell model does not hold up.[1]

Many if not most *clinical* doctors – general practitioners and neurologists who are working with patients and prescribing drugs – often have *no* idea that the dead dopamine cell theory is considered passé in the *research* realm. They merrily pass along the misinformation they were taught in medical school – information that is supported by companies that make the various antiparkinson's medications and by companies making deep-brain-stimulating implants.

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[1]"Myth about Parkinson's disease debunked"; *ScienceDaily*, 9-16-2014, www.sciencedaily.com/release/2014/09/140916084909.htm, from "Three mechanisms by which Striatal Denervation causes Breakdown of Dopamine Signaling"; J.K. Dreyer; *Journal of Neuroscience*; 2014; 34 (37): 12444 doi: 10.1523/JNEUROSCI; 1458-14.201.

This article is just one that I plucked at random from my large collection of research articles that disprove the dead dopamine cell theory.

This article, like others, points out that, at the time of diagnosis, people with Parkinson's have more than enough dopamine in the brain. This article's point is that, for some inexplicable reason, dopamine is present in the brain but isn't used to provide motor function in people with Parkinson's.

This is not actually inexplicable. The reason for the striatal denervation is that dopamine release for motor function is *supposed* to be inhibited when a person is on pause. After decades of *not* using striatal neurons, the brain, ever efficient and thrifty, begins to break down some of the un-used striatal neurons. Based on what I've seen in recovering patients, the brain restores these neurons when they once again are called for.

As an aside, the deep brain implants do *not* elevate dopamine levels. This is a well-known fact. They work by creating a sense of emergency in the brain.

The electrical stimulation creates enough sense of emergency that what I call the norepinephrine override is able to once again mask the symptoms of pause. However, the brain soon becomes accustomed to the implant's electrical bursts in the brain. This is why an implant's signals need to be regularly adjusted or put on a varying schedule so that the brain does not become accustomed to the stimulation and then stops treating the implant stimulation as an emergency.

More than enough dopamine

Research shows that people who have been recently diagnosed with Parkinson's not only have more than enough dopamine in their brains, but we have known, since 2001, that the risk-assessment areas in the brains of people with Parkinson's have *elevated* dopamine levels when compared to those of people without Parkinson's. [1]

The problem in Parkinson's is *not* the absence of dopamine. The problem is the *inhibition* of brain-based dopamine for motor function and for some types of mental processing such as positive visualization, and the *inhibition* of adrenaline release from the adrenal glands…the exact same life-saving inhibitions that occur, correctly, during near-death shock and coma: during pause mode.

A peek inside the brain

A few people have asked me to explain a bit about the chemicals and structures in the brain that come into play in Parkinson's. This section will give a quick overview of one neurotransmitter plus three brain areas: dopamine plus the substantia nigra, the striatum, and the thalamus.

Dopamine

Dopamine can activate certain nerves and neurons. (The nerve-type cells located in the brain are called neurons.) We call nerve and neuron stimulators "neurotransmitters." Dopamine is produced in several areas in the brain, including the substantia nigra.

Dopamine is also produced outside the brain, where it behaves more like a hormone, affecting blood vessel and various organ behaviors. In the brain, dopamine behaves like a neurotransmitter, activating neurons.

As noted earlier, blood dopamine and brain dopamine are, for the most part, kept apart by the blood brain barrier. However, extremely high levels of blood dopamine will lead to a small amount crossing into the brain. (Carbidopa, when added to L-dopa, allows

[1] This citation was also used in the first chapter. "Personality traits and brain dopaminergic function in Parkinson's disease"; *Proceedings of the National Academy of Sciences USA* 98:13272-7*;* Valtteri Kaasinen, MD, PhD et al; 2001.

But on this page I've added the following: We now know that different brain areas produce dopamine for different brain functions. This is why the dopamine-enhancing medications, which indiscriminately flood the *entire* brain with dopamine, have so many unwanted and even disastrous adverse effects and can cause long-term brain damage.

the L-dopa to pass through the blood brain barrier. Most pharmaceutical preparations that use L-dopa for Parkinson's treatment combine it with carbidopa.)

The substantia nigra

The substantia nigra is made up of two (left and right side) very small areas, just to the sides of the midbrain. "The midbrain" refers to tissues located through the center line of the brain, going from front to back, as opposed to those brain structures that are located on the left and right sides of the brain.

Looking up at these two substantia nigra areas from the bottom of the brain, they look like two wispy eyebrows, slightly darker colored than the surrounding brain cells.

This darker color is the source of the name substantia nigra, which translated loosely means "black stuff."

The neurons in the substantia nigra have long tendrils that send dopamine-triggered messages to the muscle-moving neurons to provide for automatic (not mental command-based) movements.

In autopsies of people with Parkinson's, the substantia nigra area is less black than it should be. The number of specialized, black cells in this area is reduced.

As an aside, no one, and I mean no one, has ever done an autopsy study of this area comparing people with Parkinson's who never took antiparkinson's medications and those who did, using brains of people who had the same *degree* of Parkinson's symptoms. So we have no way of knowing how *much* of the reduction in blackness might be due to drug use.

Even so, based on recovery symptoms, and despite recent research showing that people *newly* diagnosed with PD have sufficient dopamine levels, I hypothesize that people with Parkinson's *do* eventually develop an insufficiency of dopamine: a decrease in available dopamine for movement and temperature regulation, but not necessarily for other brain functions.

I hypothesize this because, during recovery, people with somewhat *advanced* PD find that they have only a limited amount of dopamine for motor function at first...even if they never took antiparkinson's medications.

When, during recovery, the dopamine supply temporarily runs out, a person does *not* become rigid, as he used to do with Parkinson's disease. Instead, he becomes very comfortable, although motionless and limp: more like a rag doll than anything. After some time, ranging from a few minutes to half an hour or so, when enough dopamine has gotten recycled into the brain's dopamine-holding tanks (vesicles), the person suddenly, effortlessly, springs back to full movement.

If, say, during one week the recovering person was consistently able to move for an hour at a time before going limp, and then have ten to twenty minutes of downtime, by the next week he might be able to move effortlessly for two hours at a stretch. In a few more weeks, he might be able to go six hours and the downtimes in between will be much shorter. The body recycles dopamine, so as it generates more and more, the amount of functional time increases quickly. And every time a person runs out of dopamine, that's a signal to the brain that it needs to make still more.

Fairly quickly, usually within a few months, the person no longer runs out of dopamine, ever.

This running out of steam will *not* happen during an emergency, or while driving or performing an activity in which being motionless is not an option. During recovery, a person is once again able to access adrenaline from the adrenal glands. And he can still use the norepinephrine override, even though doing so will feel mildly unpleasant and a bit stressful – a reminder of the old Parkinson's days.

If the dopamine runs out during some *critical* activity, adrenaline and/or neural norepinephrine will kick in.

If the dopamine runs out while the recovering person is just hanging out around the house, he will have several seconds of warning that he is starting to fade, and should immediately sit down. This is not a dangerous problem, although people who still have Parkinson's who read about this are sometimes horrified by the idea that they are going to lose even more control over their body. Worry and anxiety is a part of the on-pause Parkinson's personality (a concept that will be discussed later).

The inhibition of visualizing positive scenarios, as well as the inhibition of other positive-thoughts, plus the high level of wariness and/or anxiety, will go away when pause turns off. The recovery period is actually a very pleasant time, full of daily surprises and gentle joys.

But the point is, even if a person never took antiparkinson's medications, the recovering person's body might show evidence of short periods of dopamine insufficiency *and* an ability to re-build the dopamine supply. This suggests that the dopamine supply had, in fact, been reduced because it wasn't being called on. As soon as the person is once again *able* to mentally use dopamine for motor function, the body starts to rebuild the supply.

If you're wondering why, a few paragraphs earlier, I associated dopamine from this part of the brain with temperature regulation it's because, in a sudden spell of very hot or cold weather, the slowly burgeoning dopamine supply in a recovering person runs out a bit faster than usual. This type of information will be covered in great detail in the chapters on recovery symptoms.

For now, I mention it merely to show that, yes, a person diagnosed with early-stage Parkinson's still has more than enough dopamine to move normally and yes, the decline in dopamine levels does exist in people with advanced Parkinson's, even in people who never took antiparkinson's medications. And - here's the most important point – dopamine levels, *if* insufficient, will be restored to a healthy, more than adequate amount when a person recovers from Parkinson's.

An early theory from the 1960s declared that the dark cells in the substantia nigra must have *died*. That was the only option, based on western medical theory at that time. Back in those days, we were taught that brain cells could never change or regrow. The brain was thought of as static: an inert switchboard of unchanging connectors.

We now know that to be completely wrong. Brain cells are constantly changing, being altered. They are constantly increasing in activity and influence in areas that are increasingly stimulated, and decreasing and being re-assigned in areas that are not being used or that are being inhibited over a long period. (See footnote on page 15 xxx.)

The slides of the substantia nigra in people with Parkinson's that I looked at in the 1990s showed that some of the dark cells had been replaced with normal colored brain cells. This suggested to me even at the time that these cells that were no longer being used to make dopamine had converted (re-undifferentiated) back into a more basic type of cell.

This is just good, efficient and thrifty use of materials on the part of the brain. If an area isn't being used, the brain can let cells in that area become reassigned from their specialized jobs, reverting back to the more basic, less specialized type of cells…until they are needed again.

So in people with Parkinson's, are there eventually fewer of the black, dopamine-making cells? Probably yes.

Are the cells dead? Almost certainly no.

Can they resume their role as "black cells" if they are once again called upon? Based on steadily increasing duration of recovered patients' dopamine-based movement, it certainly appears that they can.

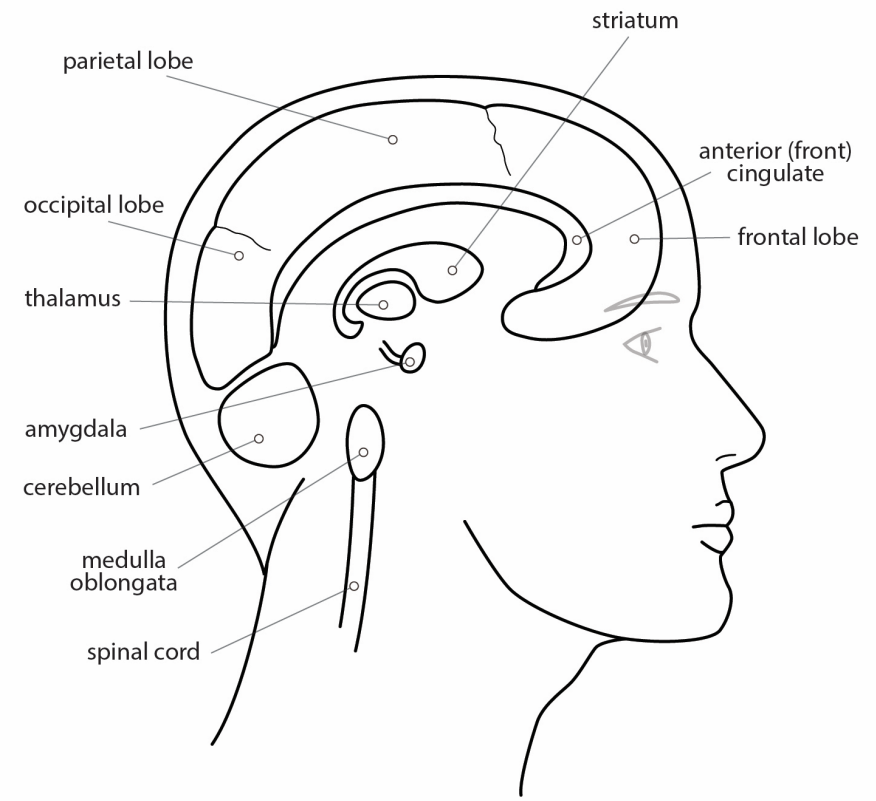

Fig. 3.1 A few brain areas

The striatum

The striatum is a brain area that regulates automatic and voluntary motor function and the "reward system" (determines how good you feel.) It sits on the midline of the brain about halfway along an imaginary arc that parallels the midline of the skull, an arc that connects the brain stem area (at the top of the medulla oblongata) to the point between the eyebrows on the forehead, a point sometimes called the Third Eye or, in Chinese, Yin Tang.

The striatum receives dopamine from various areas in the brain and distributes it to other areas in the basal ganglia (most of the midbrain areas, but not the big lobes).

The thalamus

The thalamus is like a little nut right in the center of the midbrain. It regulates sensory awareness, including how you feel inside your body. The striatum is larger than the thalamus and wraps around the back, top, and front of the thalamus. The thalamus is the location of irregular electrical "static" in people with Parkinson's. This static has long been thought to be the cause of the tremor.

I hypothesize that the origin of the tremor is in the sacrum.

When the sacral tremor is turned off, the brain's "internal tremor" ceases. But when, during napping or deep calm, the *brain's* internal tremor merely slows or seems to *temporarily* stop, the tremor in the sacrum continues. The static in the thalamus seems to merely "echo" the electrical agitation in the sacrum. Whether or not the internal tremoring also manifests visibly in the *physical* body depends on 1) the degree of alertness and the degree of concern the person is experiencing at the moment, as well as 2) the degree of atrophy and mental disconnect in the specific body areas that are tremoring.

Although the assumption has long been that the problem in Parkinson's starts in the substantia nigra, the recovery symptoms and behaviors I've seen when people recover from Parkinson's suggest that the actual sources of the movement inhibition and visible, external tremor are the striatum and thalamus. The decline in the number of *active* dark cells in the substantia nigra is likely just a side effect from the brain being on pause over the long term, causing automatic and voluntary motor function to be chronically inhibited.

And when Parkinson's turns off, patients' recovery symptoms suggest that the long-unemployed substantia nigra cells once again differentiate back into dopamine producing/distributing cells.

More channel theory

Based on Chinese channel theory that says channel qi flow through the midbrain is significantly reduced during sleep and *extremely* minimized during pause, I propose that during sleep and during pause mode, activity in the striatum and thalamus, both smack in the middle of the midbrain, is correspondingly decreased.

The striatum is recognized as being the primary source of input for the rest of the lower midbrain area (the basal ganglia) and is especially crucial for regulating voluntary movement. When the striatum is inhibited, as it is during sleep or when on pause, voluntary movement is inhibited.

This is a good design: you don't want to be running around like a chicken with its head cut off when you are asleep or while trying to not die from a potentially mortal injury.

At the same time, the inhibition of the *thalamus* while on pause causes inhibition of sensory function, which is also all to the good while you are trying to focus your energy on sleeping or on not dying. This inhibition of the thalamus contributes to the inhibition of some types of pain, an inhibition that has long been associated with mortal injuries: as the old folk-lore tells us, "mortal injuries don't hurt."

(Other neurological systems, such as the release of endorphins from the bases of the spinal nerves during pause, also contribute to the inhibition of pain from a potentially mortal injury.)

I propose that under normal (not self-induced) situations of near-death, normal thalamus behavior is inhibited by the use of pause mode. *But* when the body has finally

become physically stabilized following the trauma, that's when the sacrum and thalamus begins their electrical signaling behavior. The low-level, nattering electrical signal (neurologists call it "static") affects other areas in the brain and can even get your conscious attention as you begin to tremor in time with this signal, either visibly or internally.

Based on how non-Parkinson's people respond to their tremors just before coming out of pause, tremor, whether visible or internal, seems to be a signal that says to the brain, "The body now feels biologically stable on the *inside*. Is the perimeter now safe?"

While western doctors see the electrical static in the thalamus of people with Parkinson's as a pathology, it is actually normal behavior following a severe trauma. It is the first sign that the body has stabilized enough to come out of pause…so long as the coast is now clear.

The midbrain on pause

In order to overcome the pause-induced inhibition of dopamine release from both the striatum and the substantia nigra, a person must first come out of pause. The brain isn't allowed to do this until the body feels physically stabilized – the blood pressure is stable, the person has stopped bleeding out, and so on – an internal *feeling* processed by the thalamus: a feeling that initiates the tremor.

The tremor can be thought of as a little starter motor, one that gives off a tiny, wobbly, static-y vibration. When parasympathetic mode and sympathetic mode kick back in, the big electrical engines of these modes surge through the midbrain and the brain motor purrs smoothly: the tremor turns off.

One patient, after learning how to isolate and mentally turn off the sensation of static tremor coming from her sacrum, said it reminded her of how, when stopped at a traffic light on her bicycle, she wobbles on the bike until the light changes to green, when she can power forward and resume the smooth, gyroscopic balance of a moving bicycle.

In response to the query from the tremor, when the conscious sensory functions of vision, hearing, smell, and touch confirm that the surroundings are now safe, the brain allows the resumption of the the normal, awake-time flow of energy through the midbrain. This strong flow of energy coming from the strongest current in the body, the spine- and brain-traversing Du channel, fully activates the striatum and thalamus.

The three physical moves, the deep breath, the head bobble, and the spinal shimmy, are then spontaneously performed. Pause turns off.[1]

But in self-induced pause…

While on self-induced pause, the brain is evidently not able to respond correctly to the tremor because it has been commanded to be numb: to stay on pause. Based on patient responses and behaviors, the brain seems unable to respond. Instead, it refuses to even consider the possibility that "the perimeter is now safe." Which means, I hypothesize, that the person is stuck in between the first and second steps of coming out of pause.

The body is internally tremoring, essentially saying to the sensory functions, "Yoo hoo! We're physically stable; is the perimeter safe now so we can come out of pause?

[1] Detailed information on how the channel qi flows in the different neurological modes is provided in *Tracking the Dragon*.

The body seems to be saying "Now?"

And the brain is replying, "The perimeter can *never* be safe; we've been instructed to be numb to physical or emotional pain (be in pause mode, stay on the verge of death)."

"Now?"

"Never."

"Now?"

"Never."

A relentless internal tremor and the urgency to come out of pause that it impels is countered by the brain's refusal to even *consider* that the world is once again safe.

This impasse seems to be the underlying cause of Parkinson's from self-induced pause: Type I PD.

As so many people with Parkinson's have told me, they feel as if they are always "stepping on the gas and the brakes at the same time." Or that they are relentlessly, commanding their body to "get going" but the brain is *refusing* to make it happen or doesn't know *how* to make it happen.

A bypass for pause

The brain scan research to be discussed in chapter seven xxx helped me to devise exercises for people with Type I PD, mental exercises that vigorously stimulate activity in the striatum and thalamus directly.

Normally, the sensory confirmation of safety allows the striatum and thalamus to then activate, completing the second step in the process for turning off pause, based on my research on this mode.

The exercises I developed do an end run around the sensory functions. They *bypass* the conscious, sensory confirmation step and *directly* fire up the striatum and thalamus.

Based on my patients' experiences, it appears that increases in activity in these two brain areas can eventually get enough neural stimulation going through these areas that the body and mind both *feel* as if safe again, and go on to activate the physical moves that complete the termination of pause – thus *overriding* the long-abiding instruction to "feel no physical and/or emotional pain."

The direct reactivation of the striatum and thalamus seems to induce the last three steps for coming out of pause mode – the deep breath, the head bobble, and the spinal shimmy.

Once the moves are complete and pause is turned off, the brain areas and body behaviors promptly go back to full, healthy function again; they "come back to life" – just as they are designed to do.

Going through the motions

If the striatum is not anticipating feeling safe, just going through the physical steps will *not* turn off pause. In fact, the body might physically resist attempts at wobbling the head and shivering along the spine. These moves will feel forced and unnatural, even "unsafe."

The genetic angle

Western researchers have observed that Parkinson's disease seems to run in families. This has led to a search for a genetic cause for Parkinson's disease. However, outside of the Contoursi Family genes that cause a Parkinson's-like syndrome and which are not found in most people with Parkinson's, researchers have found no genetic smoking gun.

Instead, if we look closely, we see that Parkinson's runs, not specifically in families, but in social groups. Social groups *or* families that emphasize suppression of emotions are more likely to see higher numbers of people with Parkinson's disease.

In the United States, the demographic group with the most Parkinson's per capita is the Mennonites, also known as the Amish. Their social requirements include *never* showing anger, resentment or other so-called "unChrist-like" emotions.

A famous example is that if someone murders your daughter, you should lovingly invite him/her to dinner and forgive him/her because that is what Jesus would supposedly have done. As you can imagine, *powerful* suppression of negative emotions is rampant in this culture, in those people who are capable of it.

If there *is* a genetic link to Parkinson's, and there may well be one, it will be related to the high level of word-based intelligence and focus that a person needs in order to activate self-induced pause. A scatter-brain can't do it.

You will almost *never* meet a dull-witted person with Type I Parkinson's – or at least not one who was dull-witted up through early adulthood or before taking the anti-parkinson's medications.

Although there are exceptions, a tremendous intensity of purpose and will power, and a high-level of word-based intelligence are typically some of the components of what is referred to as the Parkinson's personality: traits that are common in Type I PD. *These* attributes might have a genetic component. But genes that contribute to word-based intelligence and mental focus don't *need* to be used to induce pause. The genetics might provide the skill set used for inducing pause mode in oneself, but the genetics don't *require* that those skills be used in this way.

The identical twin study

In the late 1990s, I received an email from a university researcher on the team doing the largest ever – at that time – identical twin study. The point was to see if there was a genetic component to Parkinson's disease.

Back then, before we had DNA analysis and before the idea of epigenetics emerged, identical twin studies were considered the gold standard for proving the genetic basis of an illness.

The premise was this: if the syndrome is always present or always absent in *both* of a pair of identical twins, the syndrome was genetic.

If the source of the syndrome was *not* genetic then, if one twin had the syndrome, the odds of the other twin having it would be about the same as the national average for that syndrome.

The reason I was contacted was that the researcher was having a numbers problem.

The study showed that if *one* of the identical twins had Parkinson's, the *other* twin was far *less* likely than the national average to have Parkinson's.

This made absolutely *no* sense to him. Even if Parkinson's *wasn't* genetic, the other twin should have PD as often as the national average.

I explained that Parkinson's had a sociological component. In cases of identical twins, even in families who promoted stern suppression of emotion and discouraged coddling (or worse, used physical and emotional punishments in response to children's mistakes), the subordinate identical twin might have something that most people don't have: a deeply personal connection with an at-hand champion or comforter.

In the very special identical-twin relationship, the dominant twin could serve as a secret confidant, an emotional support for the subordinate twin.

The *dominant* twin might induce pause in himself or herself in order not to feel or show pain, and go on to develop Parkinson's disease. But the *subordinate* twin might always be able to secretly lean on and find *safety* in the love and strength of the dominant twin.

Because of this life-long, exceptional level of emotional support, a subordinate twin *would* be less likely than the national average to powerfully command himself to be stoic and then go on to develop Parkinson's.

The researcher and I exchanged several emails. He completely understood how my hypothesis would account for their findings.

However, his final email said something like, "Thanks, but I can't use your information after all. My grant requires me to prove that Parkinson's is genetic."

So let's leave the disproven western hypotheses about dead dopamine-producing cells and genetics-based PD behind us. Let's return to the subject of what actually does cause Parkinson's disease, and what's needed to turn it off.

Chapter four

Requirements for recovery

Recovery from idiopathic Parkinson's disease requires three things: 1) correct diagnosis, 2) no brain damage from antiparkinson's medications and 3) in the case of *self-induced* dissociation or pause, the ability to understand the problem and the desire to mentally bring about changes in thought patterns.

1. Correct Diagnosis

Starting with a good diagnosis is crucial. You will learn in upcoming chapters far more details than your neurologist ever learned in school regarding how to make an accurate diagnosis of idiopathic Parkinson's disease.

Having a solid diagnosis will also be of comfort to you when you recover and your doctor tells you that you were merely misdiagnosed.

My patients who have recovered have been told that they had been misdiagnosed. Many have been accused of pretending to have Parkinson's in order to get early retirement, pity, or to get access to the mind-altering, euphoria-inducing (if you don't have idiopathic PD) medications. Obviously, these nasty accusations do *not* fit with the fact that the patient booked an appointment with the MD in order to share the happy news of having recovered. Worse, a cynical MD might also conclude that the presumably deceitful patient now wants to be falsely lauded for his/her miraculous recovery.

I have learned that a patient doesn't stand a chance against a doctor with a rigid mental attitude.

One person who I never met emailed me after she recovered. She had been diagnosed six times. She had moved frequently due to work, and at each new location she had met with a new neurologist. Every one of them diagnosed or confirmed her diagnosis of Parkinson's.

When she recovered, she didn't even bother to go back to her latest neurologist. She knew he would tell her she had been misdiagnosed.

Still, she wanted me to know how *many* doctors had diagnosed her with PD prior to her complete recovery.

Misdiagnosis

I have also seen in my clinic many, many people who had been diagnosed with Parkinson's disease who clearly did *not* have Parkinson's disease. Usually, they have been very *obviously* misdiagnosed.

In many cases, these people had very *quickly* developed *one* or *two* symptoms similar to those of Parkinson's in *immediate* response to a surgery, injury, or a particular medication.

For example, I've seen post-surgical rigidity in the joint that was operated on then misdiagnosed as Parkinson's…in a person with *no* other symptoms of PD. I've seen sudden-onset tremor in response to a new heart medication misdiagnosed as Parkinson's in a person with *no* other symptoms of PD.

In so many of these obvious cases of misdiagnosis, the diagnosing MDs or surgeons had dismissed their own responsibility for the patient's new, sudden-onset symptom(s) by declaring that the sudden new symptom was Parkinson's – a syndrome that cannot be confirmed with any test – thus clearing the MD from any responsibility for triggering the new symptom.

I saw an egregious misdiagnosis of Parkinson's with regard to an immediate-onset lack of arm swing due to a shoulder injury the night before. Her lack of arm swing wasn't Parkinson's-like. Her upper arm bone had become displaced from the shoulder socket and she literally could not move the arm. The woman had *no* symptoms of Parkinson's, but had been given a diagnosis of PD by a qualified neurologist.

In the *many* cases of glaring misdiagnosis that I've seen, the patient's channels were *never* running in the pause schematics, although some of the channels, especially those in the immediate injury area, were often very obviously running incorrectly.

Oppositely, *all* of my correctly diagnosed Parkinson's patients were clearly running the electrical patterns that occur during pause.

The prevalence of misdiagnosis

Based on autopsy studies, over thirty percent of people with a diagnosis of Parkinson's had none of the brain changes associated with Parkinson's, and were presumed to have been misdiagnosed. Over thirty percent![1]

For another example of uncertainty of diagnosis, consider the infamous Eldopa study of 2002. At the time, increasing numbers of research studies were proving that dopamine-enhancing drugs are addictive and can cause lasting brain damage and drug-induced parkinsonism.

In response, a drug company-led study was cleverly designed to make it *appear* that that dopamine-enhancing drugs do *not* accelerate symptoms of Parkinson's. (Brain scans taken as a part of this study proved that, in fact, the drugs *did* cause levels of damage that correlated to dosage levels, so the scan results were left out of the published conclusion.) For this study, six top-level Parkinson's research MDs were enlisted. The

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[1] I regret that I can no longer put my finger on the several research papers that presented information on this subject. But I can say that in my own practice, at *least* twenty percent of the people who came to me because of a diagnosis of Parkinson's did not, in fact, have it. (I did not track these people and do not have exact numbers.)

Many of them had obvious misdiagnoses, as described above. Many others had some stuck or twisted joint that was preventing knee bending, wrist flexion or some other movement, and the grossly negligent neurologists had put the one or two symptoms down to Parkinson's.

If I could remove the one or two symptoms that had been misdiagnosed as PD in one hour-long session of physical therapy, I felt perfectly justified in telling the patient that he/she had been misdiagnosed.

Not one of these patients, to my knowledge, ever went back to inform the MD. They were just so tearfully glad to "get rid of the hopelessness of degenerative PD" that they didn't really care about follow-up. But then, they also didn't have the grim determination and drive of most people with Type I Parkinson's, necessary qualities for generating self-induced pause *or* for taking on an MD with poor diagnostic skills.

Joke: What do you call a doctor (or acupuncturist) who graduates at the bottom of his or her class? Answer: a doctor (or acupuncturist).

plan was to have unanimous confirmation that each of the two hundred plus participants did, in fact, have idiopathic Parkinson's.

My point here, with regard to the difficulty of diagnosing Parkinson's disease based on ambiguous western medicine criteria is that the MDs were so conflicted over *who* actually had Parkinson's that they finally agreed that any person for whom three of the six doctors gave a diagnosis of idiopathic Parkinson's could be used in the study.

This is just to show you how hard it can be to make an accurate diagnosis of idiopathic Parkinson's using the vague, western medicine standards.

Diagnosing idiopathic Parkinson's

You cannot trust your doctors to make an accurate diagnosis, nor should you blame them. The doctor is doing what he or she learned in school.

So before you even try to decide which of the four *types* of idiopathic Parkinson's disease you have, you will first want to confirm that you actually have Parkinson's.

To qualify for a diagnosis of basic, idiopathic Parkinson's disease, a person must have a certain number of symptoms that fall under the four categories of rigidity, poverty of movement, postural instability, and tremor.

A later chapter will help you diagnose what you have and will go into this subject in *extreme* detail: far better detail than you will find in your neurologist's pamphlet on Parkinson's or on the internet.

Diagnosing becomes even more certain if one *also* uses the electrical currents running just under the skin as another confirmation for the diagnosis. If one understands *how* these electrical schematics alter the behaviors of the underlying muscles and organs, creating the characteristic look and the internal "feel" of Parkinson's, diagnosis becomes fairly straightforward.

If think you might have Parkinson's, go through the steps in chapter xxx for confirming this syndrome.

If you confirm that you do have idiopathic Parkinson's, your next step will be determining what *type*(*s*) of Parkinson's you have. From there, you will be directed to the appropriate treatment for that type.

If you have more than one type, instructions are provided that explain the safest *sequence* for treating the different types.

More books

You might need some books other than this one. If you determine, using this book, that you are either dissociated or using self-induced dissociation, you will be directed to specific chapters in the book *Yin Tui Na* to learn appropriate recovery techniques.

If you determine that you are either stuck on pause or using self-induced pause, you will be directed to the book *Stuck on Pause* to learn appropriate recovery techniques.

If you or a friend want to learn more about the channels involved in Parkinson's or want to learn how to feel someone else's channel qi (it's nearly impossible to detect your own, especially if you're on pause) and thus make your diagnosis even more firm or track your progress in recovery, you can find this material in the book *Tracking the Dragon*.

The first two of the above mentioned books and the relevant chapters of the third are available for free download at the website of the non-profit Parkinson's Recovery Project: www.PDRecovery.org .

2. Brain damage-type parkinsonism

Another requirement for recovery is not having brain damage caused by drugs, toxins or antiparkinson's medications. The symptoms from this type of damage are referred to as drug- or toxin-induced parkinsonism.

Although these syndromes sport the name "parkinsonism," they have nothing to do with idiopathic Parkinson's disease.

The word idiopathic means "no known cause." The use of the word idiopathic to describe basic, or "classic" Parkinson's disease no longer makes sense since we now know what causes it, but I still use the word here to mean "classic" Parkinson's, as opposed to drug- and toxin-induced parkinson*ism*.

When doctors refer to a syndrome as "parkinsonism" that might mean the person has one or some symptoms *similar* to those of Parkinson's but they do not actually have idiopathic Parkinson's disease. Then again, some MDs, unsure of themselves, will give a diagnosis of parkinsonism to imply "early-stage PD." Other MDs use a diagnosis of essential tremor in the same way, to mean "early-stage PD, but I don't want to frighten you with a firm PD diagnosis."

These evasive diagnoses reflect wisdom and kindness on the part of the doctor. Many people with Parkinson's will go into an almost immediate tailspin with severe worsening of symptoms as soon as they receive the diagnosis of Parkinson's. To extend quality of life, many wise doctors will give the mild diagnosis of essential tremor or parkinsonism, thus giving the patient an extra year or two of mild symptoms before changing the diagnosis to idiopathic Parkinson's when the symptoms inevitably worsen.

For an example of what can happen to a patient upon receiving a diagnosis of Parkinson's disease, I had one middle-aged patient who, after his diagnosis with PD, went from very mild symptoms to barely able to move – over the very short period of six months. When he discovered the Parkinson's Recovery Project information on the internet and learned that Parkinson's was reversible, his symptoms *immediately* went back to approximately the mild level he'd had six months earlier. This rapid worsening of symptoms in response to a diagnosis of Parkinson's disease is *very* common.

Back in the days when there was no effective treatment for PD, giving the patient a relatively milder diagnosis such as essential tremor or parkinsonism was an intelligent and compassionate approach, because this prevented an immediate, mentally-based rapid worsening of symptoms.

Today, however, not only is there an effective treatment, but the sooner it is implemented, the easier and faster is the recovery process. There is no longer a need for doctors to hide the diagnosis for as long as possible. Just the reverse.

Channel diagnostics

If a person is diagnosed with essential tremor or parkinsonism *but* is running the electrical circuitry of pause, then he *does* in fact have idiopathic Parkinson's disease, albeit at an early stage, no matter what the diagnosing MD has declared.

In my limited experience, *every* person with idiopathic Parkinson's disease has been running most of the very distinctive electrical schematics of pause. There are four different ways to get there. But in every case, the resultant schematics will be very similar. And, although no two patients will have the exact same combination of symptoms, the symptoms that do manifest *will* all conform to what might happen when the channel qi is running in pause mode.

People with *only* drug- or toxin-induced parkinsonism do *not* have these schematics. Their symptoms do *not* have the same "look" as the symptoms of Parkinson's. They *do* have brain damage.

I do *not* have any suggestions for people with this type of brain damage.

This book does *not* address the diagnosis or treatment of drug- or toxin-induced parkinsonism.

Nor does this book address the problems of people who have taken dopamine-enhancing antiparkinson's drugs for more than a few weeks. These people might have started out with idiopathic, pause-based Parkinson's disease, but within a few weeks of using the dopamine-enhancing drugs, they might already have incurred enough brain damage to create drug-induced parkinsonism. This means they now have two problems: whatever caused the idiopathic Parkinson's *plus* drug-induced parkinsonism.

Even if they recover from Parkinson's, they might still want to take the antiparkinson's medications because of the drug-driven damage to the brain's dopamine-producing and regulating system, just like those "frozen addicts" mentioned in chapter three.

A person who is on pause is *less* susceptible to the adverse effects of the medication. At correct, manufacturer-suggested dosage levels (a rare event), the adverse effects will accumulate slowly, maybe over five to ten years.

But a person who is *not* on pause and is taking the medications in order to help with symptoms of drug-induced parkinsonism might *rapidly* develop the nightmarish adverse effects of the medication, within a few years, or a few months, or, in some cases, within a few weeks.

I have seen rapid, horrible, mental destruction and psychosis in people who took their previously safe level of dopamine-enhancing antiparkinson's medication after pause was turned off. But a person with drug-induced parkinsonism might feel he *needs* to use the medication, or might be pressured to use them by his doctors or loved ones.

In a way, being on pause acts as a brake on the adverse effects of the drugs. So if a person does have drug-induced parkinsonism from taking antiparkinson's medications, in *addition* to having idiopathic PD, he might be better off *not* trying to recover and just keeping his doses as low as possible while staying the course.

I will not work with a person who has ever taken dopamine-enhancing antiparkinson's drugs for more than three weeks. The reasons are explained in greater detail in my book *Medications of Parkinson's: Once Upon a Pill*.

Going back to the early days

None of my first six patients had ever taken antiparkinson's medications. They had recovered easily. After I'd seen these unexpected recoveries, I recruited volunteer

patients from the local Parkinson's support group. All the new patients from the support group *were* taking antiparkinson's drugs.

When these new patients slid into something similar to partial recovery, a nightmarish condition described in an upcoming chapter, my first thought was that this had something to do with the medications. And I was partly right.

All the patients in the free clinic (1998-2003) were asked to keep daily logs of their suddenly unpredictable, drug-related symptoms and their attempts to moderate their drug doses.

In patients who started showing symptoms of recovery while still taking their meds the end results were uniformly disastrous, leading either to death or super-human dyskinetic spasms and/or severe, irreversible psychosis.[1]

When it came to dose changes, I insisted that patients work with their prescribing physicians.

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[1] Please read *Awakenings*; Oliver Sacks; Duckworth & Co; London; 1973.

This book describes the almost unbelievable monstrosities and fatalities created by sustained use of L-dopa in people who did not have Parkinson's disease.

Before I read Dr. Sacks' book I saw the exact same phenomena in my patients who recovered while they were still taking L-dopa. I was actually relieved when I read his descriptions, because they confirmed what my patients and I were having trouble believing.

The results that Dr. Sacks saw in his early experiments with L-dopa were at first so charming, and then turned so shocking, so superhumanly violent, and then fatal, that many doctors declared his claims fraudulent. When he presented films of these patients at a conference, emotions ran high. The severest critic got up and left the room. Many doctors simply could not accept that any drug with a six-hour half-life could have such paradoxical and inconsistent effects over the long term.

Be sure to read the introduction, as well.

But at least, in the early days of L-dopa research, after Dr. Sacks' findings, MDs dosed with caution. Today, almost than fifty years later, the very real dangers of quickly increasing – or suddenly stopping – dopamine-enhancing medications are evidently no longer emphasized in medical schools.

For example, one of my patients was told by his neurologist, "If you want an extra boost for going to a party or something, just double your dose" [of Sinemet, the standard dopamine-enhancing drug for Parkinson's in the USA for many decades now].

The manufacturer's instructions explicitly state that a person should *never* double the dose, even if a previous dose was skipped.

Also, the manufacturers emphasize that people should be started at a low dose and *stay* at that low dose for ten weeks, by which point the full benefit of the drug will begin to manifest.

However, most of the doctors that have prescribed for my patients have irresponsibly told the patient to "increase your dose by one pill per day until you are seeing a benefit." This leads to grossly excessive dosages, with many patients taking three to six times as much L-dopa as is recommended by the manufacturer. This, in turn, leads to *much* earlier onset of the painful adverse effects and ineffectiveness of the medication.

In the 1990s, when I started my research on Parkinson's, patients typically had five to ten years before the drugs were no longer effective. Today, in 2019, because of much higher starting doses, the readily available literature reports that the average effective span is now only two to five years.

I am a licensed acupuncturist (LAc). In California, a licensed acupuncturist has at *least* a Master's degree, has put in at least four years of medical schooling including interning, and is legally considered to be a primary care medical provider, licensed to diagnose, prescribe, and treat *within* the scope-of-practice of Chinese medicine. Although we are required to study western pharmacology in school, as licensed acupuncturists we are *not* legally able to give prescriptive advice about pharmaceutical medications.

As an LAc, giving prescriptive advice or even commentary to a patient *or* to an MD regarding an *individual's* prescription medications is illegal.

Some of the patients told their doctors about their symptom changes and asked what should they do about their increased adverse effects due to now excessive medication levels. In *every* case, the doctors assured the patients with some grossly incorrect statement such as, "If you recover from Parkinson's, you can just stop taking the medications at that time."

Most of the patients' attempts to modify their suddenly over-strong drugs were tragically unsuccessful. Some patients became violently exuberant and started doubling up and then tripling their doses over a matter of days. Others became paranoid and/or psychotic, complete with wild hallucinations. These behaviors were *far* more extreme than anything I ever saw in the later years in people with mere partial recovery.

The *only* patients who died in the course of that project were the ones who followed their doctors' recommendations. Their doctors invariably advised them to come off their drugs "slowly: over the course of a few days" or "…over a week."

They died of neuroleptic malignant syndrome: sudden inability to regulate core life processes such as heart rate, breathing, and temperature regulation: the same syndrome that can kill a person going through withdrawal from certain addictive drugs.

For over fifty years it has been well known that over-fast reduction of L-dopa-based drugs can be fatal. (As noted earlier, L-dopa is the pharmaceutical counterpart of brain dopamine.) But since the early, careful days of exploration with these drugs in the 1960s, many doctors have become cavalier and now treat these mind-altering drugs that have long-term adverse effects as if they were similar to aspirin or antacids.

According to the scope of practice laws for acupuncturist, I could not say a word to my patients *or* to their doctors about their medications or dosages. Doing so would be one of the easiest ways to lose my license.

Legally, I *could* direct my patients to publicly available, written, general information about these drugs. So I wrote a booklet describing what I'd learned about the drugs and posted it on the Parkinson's Recovery Project website for free download.

Even today, anyone who asks me for advice about medications is directed to my book, *Medications of Parkinson's: Once Upon a Pill*.

The patient logs were a treasure trove. Poring through every detail of the patients' reports, I was eventually able to piece together logical answers for *why* the highly predictable patient reactions to dosage changes of L-dopa related drugs, as well as other dopamine-enhancing drugs, had long been considered "unpredictable."

One glaringly obvious reason is that researchers have wrongly assumed that the short, six-hour half-life of these drugs in the bloodstream means that the half the drug is out of the patient's system within six hours. But it's also known that these drugs only *slowly* cross the blood-brain barrier.

Based on my collected data, I hypothesized that after the drugs pass the blood-brain barrier, they then slowly *accumulate* in the brain. It appears that approximately ten weeks are required for the *brain* levels to come to equilibrium with the dose-related surges that have been going on in the *bloodstream*.

The drug manufacturers have known *something* about this right along, although they probably do not understand the full implications for dosage change. After all, many wrote in their literature that the drugs should be maintained at a low dose so the drugs can accumulate to the point that effectiveness begins or, with other drugs, be brought up to full dosage very slowly and carefully: over a period of ten weeks – or as some of them put it, two to three months.

The manufacturers of the most common antiparkinson's medication, Sinemet (levodopa/carbidopa), write in their literature that *ten weeks* at the lowest, safest dose are required to slowly and safely attain the expected benefits of the drugs.

Another of my hypotheses based on patient logs holds that the drugs also *disperse* very slowly from the brain. This hypothesis is what allowed long-term dose-related changes to be highly predictable, once I figured out the dispersal rate.

The slow dispersal rate also means that a person who decreases or stops taking his medication won't get the full result of that decrease or stoppage for nearly ten weeks.

During that time, if his body goes into failure when it crosses over the line into not enough brain dopamine to sustain core functions, usually about fifteen to twenty days after stopping or excessively decreasing the medication, it is too late to do anything about it: no matter how much dopamine you shove into the *bloodstream*, it will take more than a week to slowly restore enough dopamine in the various *brain* structures…and this one-week restoration scenario is physically impossible: the patient who started having the immediately lethal symptoms of neuroleptic malignant syndrome will have already been dead for about six days.

A second reason that the drugs have long been wrongly considered "unpredictable" is that they are hideously addictive and cause long-lasting *alterations* in the brain's dopamine processing. So a person who had enough dopamine to have stability in core brain function *prior* to starting the medications might have an altered brain…and no longer have enough dopamine stability to survive *withdrawal* from the drugs.

Thirdly, the drugs also cause lasting brain *damage* – of a type that causes drug-induced parkinsonism. So a person who has used the drugs long enough to see a benefit might also already have developed some degree of drug-induced parkinsonism, in *addition* to having idiopathic Parkinson's disease. This means that, even if such a person recovered from idiopathic Parkinson's, he might still need the medications to control his symptoms of *drug*-induced parkinsonism.

But here's the problem: the drugs are far more powerful and mind-altering in a person who no longer has Parkinson's disease. The drugs behave differently, and much more powerfully, in a person who merely has drug-induced parkinsonism.

In our free clinic, we saw that when a *moderately* medicated patient's pause-type electrical patterns suddenly turned off, that's when the drug-based psychoses and hallucinations characteristic of severe *over*-medication suddenly showed up.

We learned the hard way that a person had about three days after the backwards-flowing Stomach channel qi abruptly began flowing correctly before the severe psychoses and hallucinations began…unless they got off their medication within three days. And if,

on their MD's advice, they got off their medication in just three days, they died fifteen to twenty days later of neuroleptic malignant syndrome from over-fast drug withdrawal.

If they slowly, over a year or more, reduced their medication and finally got off of it, and only then sought treatment for Parkinson's and recovered, they might still be somewhat immobilized or tremoring due to drug-induced parkinsonism when they recovered from idiopathic Parkinson's, but at least the former syndrome doesn't necessarily have the same internal sense of doom as Parkinson's from self-induced pause. And they would have "soft" muscle immobility as opposed to immobility from painful muscle rigidity. [1]

I had one patient who slowly, over more than eighteen months, got off all his medications. He then recovered from his very advanced case of Type II Parkinson's that he'd developed over nearly twenty years. After recovering, his tremor was gone and he had a beautiful smile once again. His muscles were very weak from the drug-induced parkinsonism, to the point that he could not walk and eventually his wife, now in her seventies, moved him into a nursing home. He was also in his early 70s.

However, he was very comfortable in bed and no longer had the painful rigidity of Parkinson's. He could even move his arms and use his hands a little, which previously he had not been able to do, even with his antiparkinson's medications. He no longer had the horrible depression of Parkinson's that he'd had since twenty years earlier, prior to starting the drugs. He felt he was far better off having the mere weakness of drug-induced parkinsonism than he had been from having Parkinson's. But he was still in a nursing home, unable to walk or care for himself.

When he finally got off the last of his medications, he was shocked at how he basically had no knowledge of himself during the twenty years in which he'd been taking the medications.

His wife remarked, "His very last, once-a-week fraction of a pill of Sinemet was taken just over a week ago and yesterday, for the first time since he started taking it, he asked me how I was doing and expressed concern for my financial situation, asking if I was going to be OK given that he hadn't worked in nearly fifteen years. This was the first

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[1] Nearly twenty years after I first published my findings on rate of change of brain dopamine levels from medications, suggesting withdrawal rates that require a year to a year and a half, researchers in 2019 announced that people taking seratonin- and dopamine-enhancing anti-depressant medications should probably take a year to a year and a half to come off of them.

I know that the information I put on the internet has propagated – I have had new patients recite to me information "from the internet" explaining how a person will be safest if he never reduces his addictive drugs by more than ten percent at a time, and then waits while his body goes through the various stages of moderate withdrawal before making the next ten percent reduction – quoting exactly the information published in my book and *not* replicated in any study other than my own, that I know of. These patients have had no idea that I was the one who first put that information out there. I get a kick out of them telling me how to safely decrease dopamine-enhancing drugs in my own words. Now, twenty years later, this information, long available and evidently splashing around – unattributed – on the internet, is finally being tested and touted as new medical research.

As you can see, if there is not a significant profit motive for new information, change might come about very, very slowly in the research part of the medical world.

time he'd shown any awareness of me or my concerns since he started taking those damned drugs."

So neither he nor his wife wanted him to go back on dopamine-enhancing drugs even though they might have given him a bit of mobility…at a very high cost. They both had seen what had happened to other people in the clinic: that the psychoses and insanity from those drugs comes on a lot faster – maybe in days, not years – if the person taking the drugs has *only* drug-induced parkinsonism and doesn't have idiopathic PD.

At the end of 2002, because of the medication-related tragedies, I ended the free clinic at the acupuncture college. At the same time, I also announced that the Parkinson's Treatment team (another PD clinical project that ran from the late 1990s to 2013 with four licensed acupuncturists, including me, all doing Yin Tui Na, not acupuncture) would not work with a person who had *ever* taken dopamine-enhancing drugs for more than three weeks, total.

The next year, in 2003, I wrote a much more complete edition of the Parkinson's medication booklet. The new edition shared everything I'd learned about the medications *plus* the caveat that I would no longer work with anyone who had ever taken dopamine-enhancing medications for more than three weeks.

Again, the book is titled *Medications of Parkinson's, or Once Upon a Pill*. It is available for free download at the Parkinson's Recovery Project website: www.PDRecovery.org.

One of the most important findings explained in the book, based on all those patient logs, was that no one died or underwent *severe*, life-threatening withdrawal symptoms *if* they never reduced the dose by more than ten percent at a time and then waited up to ten weeks before making the next reduction.

This book was also written to help people understand *why*, if they have ever used antiparkinson's medications for more than three weeks, they are not necessarily considered safe candidates for recovery. It also explicitly teaches how to safely reduce antiparkinson's drugs and not die in the process.

This book has been helpful to countless people, literally countless: we don't keep a counter on the PD Recovery website. But I've gotten thank-you emails from *many* people who've successfully used the information. It explains how to make safe drug reductions from antiparkinson's drugs as well as other dopamine-enhancing drugs.

Drug reductions can sometimes reduce or completely get rid of some of the more painful and distressing adverse effects of dopamine-enhancing medications, including the dyskinesias (uncontrollable muscle movements and powerful, excruciating spasms) and the so-called "dementia of Parkinson's."

The "dementia of Parkinson's"

Prior to the introduction of dopamine-enhancing drugs for the treatment of Parkinson's, there was no such thing as the "dementia of Parkinson's." Just the opposite. Idiopathic Parkinson's was known as a syndrome in which the person, despite becoming mute and immobile, *never* lost his crisp cognitive function. Right up to the end, which was most often death from aspiration pneumonia, most people with Parkinson's retained their high intellect and alertness.

Only after the dopamine-enhancing antiparkinson's drugs were introduced did the "dementia of Parkinson's" become a recognized symptom.

The hallucinations and other mental aberrations that arise because of the medications are an absolutely logical side effect: taking these drugs is comparable to taking cocaine or methamphetamine several times a day, every day, for years. In fact, some of the MAO inhibitor drugs for Parkinson's do have methamphetamine in them. If a person takes this level of mind-altering stimulant drug(s) every day, three or more times a day, it should be no surprise that he will all too soon develop hallucinations, sleepwalking, and psychoses.

For a better understanding of how these drugs work in the brain and why they cause the well-known adverse effects, please see *Medications of Parkinson's: Once Upon a Pill*.

Please note

– If you have ever taken dopamine-enhancing antiparkinson's drugs or supplements for more than a few weeks, you might *not* be a safe candidate for recovery.

Please read the book *Medications of Parkinson's: Once Upon a Pill.* This book is available for free download at www.PDRecovery.org . This book explains which of the antiparkinson's drugs and supplements are dopamine-enhancing.

This book is only available as a download. For now, at nearly 700, 8 ½" x 11" pages, it is too massive to make it available in paperback hardcopy. I hope someday to edit it down and put it out in hardcopy.

People who are taking dopamine-enhancing drugs can have a hard time being objective about their own situation, due to the mind-altering effects of the drugs. The drugs can make a person be unrealistically, even blindly optimistic, because the drugs work by making a person *temporarily* feel overwhelmingly safe.

Therefore, I highly recommend that a person taking these drugs who is considering trying to recover ask a friend or loved one to *also* read this book. If you are taking antiparkinson's medications, you can consult with your friend or loved ones – after they've perused the literature – as to how you might most safely proceed.

I am not your prescribing physician. I legally *cannot* and I *will not* advise you about any aspect of your medications, including whether or not your drug usage is problematic in terms of recovery.

The many tragedies detailed in my book on Parkinson's medications, including deaths from neuroleptic malignant syndrome from over-fast drug withdrawal, and my inability to help ease symptoms of drug-induced parkinsonism help explain why I will *not* work with a person who has ever taken the medications for more than a few weeks.

As a gentle side note, *please* do not send me hate mail because you are already taking medications or because I have not pushed harder to make my work more quickly acknowledged. My heart goes out to you. Please know that I am working as hard and as fast I can, while being careful to be as accurate as possible with every word I write. My observations and warnings about the medications have been available online since 1999.

As for those who send me accusations that my work must be bogus because "everyone who gets a diagnosis of Parkinson's automatically takes the medications," this statement is wrong.

In much of the world, people are encouraged to stay off the medications for as long as possible, since the dopamine-enhancing drugs have a *short* period of effectiveness (two to ten years). The higher the dose, the sooner the onset of ineffectiveness and the sooner the adverse effects show up. The adverse effects, especially the dyskinetic spasms, can be worse, far more excruciatingly painful, than the symptoms of Parkinson's.

In the USA, where doctors must stay wary of lawsuit-happy patients, people with PD are *encouraged* to start on medications immediately despite the dangers and the well-proven *fact* that the drugs accelerate the worsening of PD symptoms.

Even so, many people in the USA do their research and choose to delay taking the medications for as long as possible. [1]

3. Being willing and able to turn off self-induced pause

A person who put himself into self-induced pause did so on his own, in private. In some cases, turning it off is also a deeply personal process.

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[1] "Levodopa and the Progression of Parkinson's Disease," *New England Journal of Medicine*, Janice Walton-Hadlock, Vol. 352 No. 13, March 31, 2005 p. 1380.

The above, my commentary on the Elldopa study, was the first piece by a non-MD acupuncturist ever published in the *New England Journal of Medicine*.

In addition to my own, very small four-year research project on antiparkinson's medications that gave me the information presented in *Once Upon a Pill*, research proof *abounds* that pharmaceutical dopamine is addictive and causes brain damage – brain damage of the type that accelerates symptoms of drug-induced parkinsonism.

You can do a search online for the terms "brain damage, dopamine, drugs."

These days, youth learn about brain damage from dopamine-enhancing drugs in their high school health class. They learn that a list of dopamine-enhancing drugs can include cocaine, methamphetamine, nicotine, alcohol, and the opiates, including heroin, in addition to nearly all of the antiparkinson's drugs, many anti-depression and anti-anxiety drugs, and many prescription pain-killers.

Any drug that elevates dopamine will 1) be addictive, and 2) cause brain changes, including changes that can eventually lead to drug-induced parkinsonism…no matter what your well-meaning and maybe under-informed MD or neurologist tells you. If your doctor says that the drugs have no lasting side-effects, are safe or, even, incorrectly, that the drugs *slow* the progression of Parkinson's, your doctor is wrong. Research this online.

Make sure you are visiting websites that share unbiased research reports, *not* sites supported by pharmaceutical companies or programs that *present* themselves as non-profit organizations while actually shilling or testing for drug companies. If a website or program consistently presents "research" showing that the drugs are safe or do not have dangerous side effects, and especially if the site presents "research" claiming that people with Parkinson's should start taking the medications as soon as possible after diagnosis, you might do well to investigate the source of the "research" site's funding, even if it claims to be an altruistic non-profit. Some drug-testing programs even promote themselves as non-profit research companies and do not disclose that their main "donors" are drug companies, paying them to do drug-testing on patients.

The four methods for turning off the flow of pause-type electrical currents – and thus recovering from Parkinson's – are methods that can be done either by oneself or, in cases of *only* Type II or *only* Type IV, from *only* an unhealed physical injury, with the help of a friend or family member who need not have any training whatsoever in the field of medicine.

Just as one does *not* need professional help to turn off the biological symptoms of normal pause, one does not need professional help to turn off the physical and mental sticking points that can lead to being *stuck* on pause.[1]

Therapists and psychologists are *not* taught about pause mode. They are *not* trained in the very simple steps that are necessary for coming out of biological pause if a person gets stuck in it. Nor are they trained in treating *self-induced* pause and *self-induced* dissociation.

They aren't taught anything about pause mode because, from a western medical standpoint, there is no such thing. But that's fine because professional therapy is not needed.

Many, maybe most people with Type I Parkinson's still have within themselves the mental focus and determination to do the work that turns off self-induced pause. And if a person using self-induced pause or self-induced dissociation that is causing pause-like channel qi flow *wants* to turn it off, that person *must* have within himself or herself the necessary mental focus and determination to change some of his or her thought patterns.

Then again, *if* a person has Type II or Type IV Parkinson's, he *might* be able to recover even if he *can't* understand the theory in this book. In these cases, a friend might be able to provide hands-on support by using simple Yin Tui Na. The Yin Tui Na might help the patient address the long-forgotten injury *even if* the patient doesn't remember the injury or is somewhat oblivious to the underlying treatment theory.

Please fix my grampa

Many people have contacted me because a very elderly parent or loved one has been diagnosed with Parkinson's but shows no interest in, or even any understanding of, changing his or her way of thinking by doing the self-directed mental exercises you will read about in upcoming chapters, let alone turning off the constant, wary, maybe even negative and judgmental internal monologue that has become a way of life.

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[1] People often ask if professional help for turning off pause is similar to the professional help some people get for addressing post-traumatic stress disorder, or PTSD. No.

A person with PTSD is usually considered to be stuck in "shock," meaning sympathetic mode. Biologically speaking, his body is endlessly looping through the neurological mode of fight or flight: *sympathetic* mode, *not* pause mode. A person with PTSD *might* benefit from professional help to find his way out of being stuck in this shock-related mode.

Then again, improvement is often elusive, even with professional guidance.

However, people who have a near-death injury or undergo total anesthesia (which, by the way, induces pause mode, not sleep) do *not* need professional help or counseling to come out of their temporary state of weakness and/or immobility, or even coma.

The body is designed to be able to turn off pause. The body does *not* have a specific biological sequence for overcoming excessive use of sympathetic mode, hence the need for coaching, meditation, and other aids in turning down the excess levels of fight or flight mode seen in people with PTSD.

I have received many emails saying something like, "Can you explain to my elderly father why he needs to work on recovering?"

My reply is always a regretful no.

Please do not be panicked by the idea that recovery is not for everyone. Very elderly people with recent onset Parkinson's disease who have no interest in recovering very likely will still be able to maintain a decent quality of life during the years until their passing. Even if they use the drugs, if the drugs are used very, very cautiously, the person might continue to have a high quality of life – although mentally he or she will no longer be the person you knew and loved. The drugs work by altering mental perceptions and behaviors, after all.

I recommend reading *Medications of Parkinson's: Once Upon a Pill* in order to understand what is meant by the word "cautiously" in the previous paragraph, and to learn how to determine if a person is over- or under-medicated, and what dose changes your loved one might need to make in order to have the highest possible quality of life.

Your MD will almost certainly not be informed enough to safely help you with this.

Heal my husband

A local woman brought to my office her husband with relatively early-stage Parkinson's. She was adamant that I needed to "fix" him.

She had heard of my work through the local grapevine. Neither of them had read the free online materials despite having been requested to do so when she booked the appointment.

I did a quick assessment and agreed with his doctor's diagnosis of Parkinson's. I explained a little bit about what was involved in recovering and asked him to please go home and read the online material to see if he was interested in recovering.

He very calmly replied, "No. I am the only remaining person in my family-line. Everyone in my family dies by age seventy-five. I'm already seventy-nine. My parents have died, my siblings have died. I should have died already.

I don't want to change. I'm ready to die. My wife made me come, but I'm fine with having Parkinson's."

His wife became furious…at me.

She demanded that I start treating him then and there.

The wife clearly did not understand anything I had said about him needing to do the work himself. The man, on the other hand, seemed to understand perfectly. He simply wasn't interested.

The wife then changed her tune, explaining that *she* needed for him to not have Parkinson's.

I assured the man that I understood completely, and if he ever had any questions he could give me a call.

I ushered them out. I did not charge them for the visit.

Many times I have heard from distressed people that a loved one has looked over the PDRecovery.org website and quickly announced something like, "I was exposed to bad chemicals many years ago. I probably have toxin-induced parkinsonism. I know I can't recover so I don't want to waste my time on this."

Please be aware that living on pause can eventually make a person wary and fearful of everything, including change.

Also, some people have told me that they would prefer to be the innocent victim of a cruel and random syndrome such as toxin-induced parkinsonism than consider the possibility that their own mental actions, however understandable and even justifiable, might have played a part in their syndrome, or than to even consider that in their own case Parkinson's disease might have a mental component.

I have been told, in anger, that I have ruined people's lives with my hypotheses: "Parkinson's used to be a respectable illness. People felt sorry for me because I had Parkinson's for no reason at all. Now, you make it seem like it's all my fault for having Parkinson's because it's a mental disorder. My spouse is now blaming me for having caused my own Parkinson's disease."

I've also received angry emails from health practitioners stating that my work "blames the victim."

And then there are the people who let me know, after a few question-packed emails, that they've decided they don't want to try to recover. They plan to take medications instead because they need to hide their symptoms in a hurry. "It sounds like recovery might take too long. I can't let anyone at work know that I have a degenerative illness."

Sometimes they add something like, "If people know I have a degenerative illness, they

"…won't trust me anymore."

"…won't believe in me."

"…won't admire me."

These might be wrong assumptions, based on a lifetime of wariness and the false idea that a person has to be perfect in order to stay safe or be considered a good person. Then again, if a person knows he will lose his job if the boss suspects a degenerative illness, then the person must do what is best for him.

Again, I will never pressure anyone to attempt recovery.

If a person with Parkinson's disease from self-induced pause is not interested in or not able to work steadily on creating some new, very specific thought habits, habits that will directly stimulate the striatum and thalamus and eventually lead to turning off pause, he will probably *not* be able to recover from Type I Parkinson's.

As for recovering from the other three types, *so long as* the possibility of Type I PD has been ruled out, it should not hurt to work with a person who doesn't understand the theory. You might try some Yin Tui Na on the feet or any other location where bones seem to be displaced or the person recalls a significant injury, or in the case of Type III, a bit of medical Qi Gong, if the patient is capable of it.

Chapter five

Searching for answers

To answer some of the next-most common questions, this chapter will share the approach I took, the questions *I* had, and the hypotheses I developed in response to what happened after my first few patients recovered easily but the next group of patients seemed to be more "stubborn."

After I'd seen a few dozen Parkinson's patients, my own biggest question was, "Why have *some* people recovered from Parkinson's in response to a simple, supportive holding therapy for old, unhealed injuries, while *most* of my patients with Parkinson's became distinctly *altered*, both mentally and physically, in response to the same therapy, but have *not* recovered?"

Let me back up a bit.

The late 1990s

Shortly after my first Parkinson's patient unexpectedly recovered, two more PD patients recovered in response to the same, very simple, anyone-can-do-it treatment I'd used on my first recovered patient. I had used Yin Tui Na, a Chinese medicine holding treatment that should be used for a dissociated, unhealed injury. [1]

At the time, I didn't suspect that there were four types of Parkinson's.

As it turns out, this type of treatment works for people with *only* Type II and *might* be part of the treatment for people with Type IV Parkinson's. I had no idea, at that time, that my patients who recovered so easily were either Type II or Type IV. As mentioned earlier, most people with Parkinson's have *both* Type I and Type II.

After I'd seen three people recover from a supposedly incurable illness, I was completely baffled and uneasy. Had *all* three of these PD patients been misdiagnosed? If not, was I morally responsible for announcing I'd found an effective treatment for Parkinson's disease…even though I didn't know how or why the treatment worked?

By chance and by word of mouth, several more people with very early Parkinson's happened to contact me for appointments. After I'd seen these people also recover from Parkinson's, my distress level soared. They couldn't *all* have been misdiagnosed. I had to do something with this information.

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[1] Most acupuncturists do *not* learn Yin Tui Na in school. Tui Na is usually a non-required, elective class. Not only that, but most students study the Yang-style Tui Na, a strong-arm type of physical manipulation that is nearly the opposite of Yin-type Tui Na.

For example, Yang Tui Na is used for forcefully returning a dislocated arm bone to its shoulder socket. Yin Tui Na is very subtle. It is used for injuries that are "old, forgotten, and painless." It might consist of firmly holding a displaced joint or injured body part until the body relaxes in the area so that the damaged or displaced bits can nestle back in or realign themselves.

In my limited experience, I've seen that many acupuncturists from China are disinclined to do any sort of Tui Na or hands-on therapy even if they studied it briefly in school. In China, acupuncture is often considered more highbrow than the lowly hands-on techniques.

I was, at that time, a painfully private person. I abhorred controversy. Still, I forced myself to attend the local Parkinson's support group, where I made a very short announcement of what I'd seen. I offered the people with PD a few free weekly treatments in exchange for letting me examine their feet and do Chinese holding therapy if it was appropriate.

Twelve people from the support group volunteered. When I talked with them and then physically examined their feet, they all had evidence of a significant foot injury. Many only remembered the incident, or details of the incident, following some Yin Tui Na treatments.

Their degree of dissociation was astonishing. For example, I might be able to feel a severe bone displacement in the left foot. If I asked, "Was it the *left* foot that was hurt in the railroad tie incident?"

The reply might be, "I don't know."

The rest of the dialogue might go like this:

"Was it the *right* foot?"

"No!"

"Was it the *left* foot?"

"I don't know."

Was it the right foot?"

No, I'm certain it wasn't the *right* foot."

"So was it the left foot?"

"I told you, I have no idea which foot it was."

The above response is typical of dissociation. This is *not* a problem of intelligence. Most people with idiopathic Parkinson's disease are disproportionately high on the intelligence scale. I'll include more on this facet of PD later on.

Around this same time some colleagues and friends helped me set up the non-profit Parkinson's Recovery Project, the website of which is www.PDRecovery.org. The website was originally a place to share my observations with other practitioners of Chinese medicine and ask if they could replicate my findings.

A few could. Most could not. Again, I had no idea at this time that there were four types of Parkinson's, each of which has to be treated in a specific way.

I also had to post instructions for the fairly obscure art of Yin Tui Na on the website. This was the earliest version of what is now a book: *Yin Tui Na: hands-on therapy for traumatic injury*.

Since the beginning of my research, all my writing related to recovering from Parkinson's is available for free download at this website.

A call for replication

I've been asked many times if I have any background in "real" medicine, if my research is "scientific," and/or do I know what constitutes legitimate research. I have also been asked if acupuncturists need to attend a class or two before they start poking people with needles. Good questions, all.

My writing is directed mostly towards people with Parkinson's, including those with not much background in science/research. Therefore, I'm going to include some bits

of information on the subject of research and the "scientific method" over the next few chapters, to answer questions I've received.

My "call for replication" is an important part of *qualitative* research: what happens in response to a treatment? Can the result be replicated? Can we isolate the variables? This research phase must precede the *quantitative* studies: big studies that focus on how *many* subjects are examined or treated.

For example, an individual might claim thousands of successful outcomes with some medical therapy, but if *no one else* can replicate his results, his research is worthless, or even suspect.

In the early stages of any paradigm-shifting research, the most important question after a seeming medical cure is found for an "incurable illness" is: Can another person replicate this result?

Ideally, the "cleanest" replication occurs in a separate setting, even a different country, and with the replicating researchers working only from printed instructions without ever having met or spoken with the original researcher and without having a financial or vested interest in the success of the research.

Single-blind and double-blind studies

A double-blind is a study in which neither the patients nor the doctors know who is getting the real therapy and who is getting a sham treatment or a placebo. Double-blind studies are often used for testing drug safety and drug effectiveness. Some people feel that the highest level of qualitative scientific proof for all medical research has to be the double-blind study.

Many studies in which sham surgeries or other sham procedures are performed are single-blind: the patient doesn't know what's happening, but some doctor within the system has to know. For example, the doctor who did the procedure knows what he did. Sometimes, the doctor doing the surgery is not the same doctor who is doing the assessment, making the process closer to a double-blind. But even in these cases there might not be a perfect double-blind.

Cohort studies

Some people assume that cohort studies (studies with hundreds or even thousands of subjects) are of the greatest value.

Many very useful cohort studies do *not* necessarily show that everyone gets the same result. For example, in the cohort studies for some cancer drugs, an improvement in outcome for even a very small percent of the subjects might be enough to qualify a drug to be approved as useful.

But for any given individual, what matters most is his own outcome. The fact that a benefit appears in five percent of people in a cohort, or in ninety-five percent, doesn't necessarily mean anything predictive for any one individual.

In syndromes such as headaches, diabetes, or Parkinson's, where the researchers often fail to recognize that different people can have different underlying causes, cohort studies can be extremely misleading.

Psychological and physical therapies

Sometimes, double-blind and even single-blind studies are not possible. With psychological retraining therapies or hands-on therapies such as Tui Na, both the doctor or therapist and the patient need to be cognizant of what's happening.

This means that double-blind studies cannot be used in testing the effectiveness of a patient-participation psychological therapy: a person cannot perform mental exercises that help change the mindset without knowing that he's working on doing those exercises. A person cannot receive hands-on therapies without knowing it.

And merely substituting a "sham" location for physical therapies doesn't work. Sometimes, the benefit of physical therapy might have as much to do with the psychological changes that come about through being touched, period, as it has to do with the location of the touch.

The same goes for sham acupuncture treatments, with needles inserted at locations other than the "correct" ones: insertion of a needle almost *anywhere* in the body causes a surge of current to flow throughout the channel qi. While a sham acupoint might not be the most efficient place to put a needle, it will still cause an electrical surge that *might* very well correct the problem being treated.

Likewise, a doctor or therapist cannot teach a person new cognitive skills without knowing whether or not he's teaching cognitive skills.

Although a final evaluator might not know who was taught the therapies and who was not, the patient knows what he's done to himself, or not.

In general, double- and single-blind studies are not used for syndromes with a predominantly mental component that is being altered via cognitive therapy. Self-induced pause, dissociation, and self-induced dissociation all qualify as mental components.

One model that is now widely accepted for psychological research is the "single-case study."

A later chapter will discuss the growing importance and acceptance of the single-case study for developing non-drug treatment strategies for problems that have a mental component.

Getting back to the subject of my budding research project, I started the website as a "call for replication": the first step for the type of research I was getting into.

Doing more research

In the late 1990s I also started a small qualitative research project at the local acupuncture college where I teach. Free, weekly Yin Tui Na treatments were provided for people with Parkinson's by students working towards a Master's degree in Chinese medicine.

Soon, word of mouth and the website of the Parkinson's Recovery Project brought more people with Parkinson's to my office. By now, I thought I was onto something, and was charging my newest patients for their treatments. I was still keeping my prices as low as possible. My original twelve recruits and the patients at the acupuncture college's clinic were still being treated for free. My world was filling up with Parkinson's.

Nearly all of the patients *changed* in response to the Yin Tui Na treatments. But for most of them, the strange, even bizarre changes in symptoms and personality were not complete recovery, not by a long shot.

Becoming obsessed

I spent *hours* interviewing every Parkinson's patient I worked with. I didn't just dwell on their symptoms.

At every weekly, one-hour session, while treating them, I continued my inquiries about their childhoods, their hobbies, professions, food preferences, amusements, attitudes towards life, and spiritual paths, if any. I dug deeply into anything that seemed a little outside the norm. I kept noticing uncanny similarities in most of their personalities and in their *type* of intelligence.

These similarities squared with the observations of other twentieth century doctors dating back to the 1930s, observations that suggested that most people with Parkinson's are different, somehow, from the "ordinary Joe" or the hypothetical "man on the street."

Many studies through the years have examined and tried to define what western Parkinson's researchers refer to as the "Parkinson's personality." The list of traits varies from one theorist to the next and no consensus exists even as to whether or not the personality is related to the underlying *cause* of Parkinson's or is the *result* of the PD.

I also noticed many inexplicable mental behaviors. For example, most of my patients responded to mind-calming therapies, therapies that reduce the use of sympathetic mode, with the exact opposite response from what I expected: these therapies made my patients more adept at sliding into a state of deep numbness, not the expected joy or ease of parasympathetic mode.

For example, even though what seemed like a disproportionate number of my patients had spent years practicing meditation, yoga, or Qi Gong, nearly all of them said that they never felt joy from it. It was more like a job or a duty, or something to help them feel "still" (meaning deathly still or numb) inside. But the vaunted joy and/or radiant inner peace of meditation or various other spiritual/energy practices had never made an appearance.

While most of these patients usually felt that the deeply numb state was a good thing, and very often it did temporarily slow or stop their tremoring, it was obvious that the numbness and *temporary* reduction of symptoms, if any, wasn't related to any long-term improvement. In fact, as people got better at turning off sympathetic mode (the typical goal for most self-calming therapies), they got better at going *deeper* into pause.

As it turns out, in a *deeper* degree of pause, moving closer to the state of coma or sleep, the tremor is stilled. After all, it's only when the body is saying, "I'm *alert* and stable now on the inside and therefore ready to come out of pause" does it ask the question, via the tremor, "So is the outside vicinity safe?"

Only when a person is alert does his tremor become or resume being overt. Although a very few people tremor even during sleep, for most of my patients, when their bodies slide into sleep or deep numbness, the question-provoking tremor backs off. Then, even though the electrical agitation in the sacrum and thalamus continues, the physical tremor is usually not visible.

As an aside, according to their spouses, a very small percentage of my patients with tremor *did* tremor in their sleep, usually while dreaming. But most did not.

Wrong assumptions

Early on I observed that all my Parkinson's patients – and none of the other, non-Parkinson's patients in my all-purpose acupuncture practice – had channel qi running in a weird, *backwards* direction in their Stomach channels.

In Chinese medicine, backwards-flowing channels, though uncommon, are not unheard of. They are referred to as flowing "Rebelliously," and this condition is considered to be incorrect and unhealthy. Except of course, in pause mode, when it *is* correct. But we don't learn about pause mode in school.

This backwards electrical flow pattern in the Stomach channel that I observed in my PD patients actually *explained* many of the classic *symptoms* of Parkinson's. And if, in response to foot therapy, a person's channel qi started running the correct way, running in the path I'd learned in school, the Parkinson's stopped and never returned. At least that's what happened in my first Parkinson's patients.

After seeing the first few recoveries from Parkinson's, I assumed that the trick to recovering from Parkinson's was correcting the backwards flow of these channels – flow that I referred to back in the early days as an aberration.

Certainly, in all my patients who completely recovered, their channel qi had resumed running normally. I didn't suspect that, for most cases of PD, I was reversing cause and effect. As it turns out, backwards channel qi flow does indeed cause symptoms of PD *but* the channel qi is not behaving aberrantly: in most people with Parkinson's, being on pause is what *correctly* causes the backwards flow. For most patients, the problem is being in a neurological mode that isn't supposed to be used long term. The backwards channel qi flow and the resultant symptoms of Parkinson's are merely side-effects of the long-term use of pause mode-type circuitry.

When *most* of my newer, recruited patients failed to recover in the same quick manner as my first six Parkinson's patients, I assumed it was because the new patients were all taking antiparkinson's medications. None of my earliest patients had ever taken the drugs.

In 2003, after I stopped working with patients who had ever taken antiparkinson's medications for reasons mentioned in the previous chapter, I assumed that any new, un-medicated patients would recover in the same simple manner that my earliest Parkinson's patients had done.

But even after that, most of my new patients did *not* completely recover. In response to healing from their foot injuries, most of them experienced distinct changes in their personalities and symptoms, as so many of the medicated patients had done. Some of the new symptoms and personality changes were bizarre. Weirdest of all, after the foot injury healed, the channel qi was still running backwards in their legs…sometimes. When a person has backwards-running currents because of a channel-blocking foot injury, the channel qi should run backwards *all* the time.

Overly optimistic, I named this baffling set of changes "partial recovery."

In people with now-healed foot injuries, people who clearly were not recovered from Parkinson's, people in what I called "partial recovery," I found I could usually make their channel qi go one way or the other by something as innocuous as telling a joke. While telling the joke, their Stomach channel qi might run correctly. When I finished the joke, if I said something even mildly negative, such as "Uh oh…", the channel qi would immediately run backwards.

From a Chinese medicine perspective, this quick variability in channel Qi flow in response to thoughts should only occur if the syndrome has a mental component: "*Qi* Stagnation" as opposed to "*Blood* Stagnation": primarily a psychological problem as opposed to primarily a tangible, physical or chemical one.

Most of my patients with Parkinson's tended to think of themselves as extremely logical, and often considered themselves *not* susceptible to suggestion. So despite the channel qi fluctuations that suggested a channel-influencing mental component, the problem just *couldn't* be psychological. Or so I thought.

Since those early days, I've collected research articles that show that most people with Parkinson's are *extremely* susceptible to suggestion and to the placebo effect. But of course I didn't know that at the time. [1]

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[1] *Suggestible You*; Eric Vance; Penguin RandomHouse; USA; 2016. This book on suggestibility and the placebo effect leans heavily on studies that used people with Parkinson's.

For decades now, people with Parkinson's disease have been used in studies about placebos because its so easy to tell if they are affected by placebos or not: if they are, they can go from rigid and immobile to moving somewhat normally after taking a mere sugar-pill: a pill that they have been *told* is a dopamine-enhancing drug. So the observers don't have to ask the patient's opinion or guess as to whether or not the placebo is working. They can *easily* see and *objectively* measure the response to the placebo.

If and *only* if a person with Parkinson's has already become accustomed to taking dopamine-enhancing medications and gets a predictable response from them, *and* if he thinks he is receiving his usual dopamine-enhancing dose of medication, he can usually move as if medicated until he assumes that the medication is starting to wear off, as per usual… even though he was only given a sugar pill, a placebo.

As an aside, it's important to note that, within a few days, the placebo benefit ceases because the person starts down the dangerous road of drug withdrawal and possible death.

One placebo study showed that the effect of the placebo was even stronger in people with Parkinson's who were falsely told they were receiving a new, far more *expensive* brand of dopamine than the other patients in the research study. They were still only receiving sugar pills, but their "On" time lasted longer than it did with what they thought were "cheaper" dopamine pills.

As another indication of how susceptible people with PD are to the placebo effect, the first test groups for Deep-Brain Stimulating (DBS) implants included people who got sham surgery and people who got actual implants. The sham surgery recipients got an incision in the scalp that was sewn back over, and were then *told* that they'd received a DBS implant, even though they hadn't.

The people who got the implants did well. The people who got the *sham* surgeries did even *better*. All of the participants were taking antiparkinson's medications at the time and continued to do so following the surgery. One qualifier for the experiment was that people had to have such advanced Parkinson's that their medications were no longer effective enough.

A year after the test surgeries, participants in the study were told whether or not they'd had the implants or had the sham surgery. The people who'd done the very best, had even resumed skiing or doing their other favorite sports, had a hard time believing that they'd only had the sham surgery.

And why did the people with sham surgery do better? Maybe all the people in the study felt extra lucky and therefore *safe* because they had been selected for this study – a mental component.

Possibly this positive feeling translated into a conviction that they'd had the real implants. *And* people with only sham surgery didn't have the same brain trauma and tissue damage as the

As it turns out, in people in partial recovery, which is to say in people who previously had Type I *and* Type II but who, following foot treatment, have only Type I, the channel qi in the Stomach channel runs backwards and other pause-like channel behaviors kick in when the person is wary. When a person in partial recovery is wary, the Parkinson's symptoms might be drastically worse than they were *before* receiving treatments for the foot injury.

And yet, in partial recovery, after the foot injury has healed up, *if* the person is feeling temporarily safe, the channel qi *might* run normally and the symptoms of Parkinson's might subside or temporarily stop.

As an aside, even if the *visible* tremor stops in response to feeling temporarily safe or "deadened," the internal tremor does not. The stopping of the *internal* tremor is permanent – and stunning. When it finally happens, it's an event so profound that people sometimes think that they must have died, even though they are still breathing.

The come-and-go of partial recovery

As an example of the come-and-go-symptoms of partial recovery, I had one Parkinson's patient who successfully re-associated with and healed from his childhood foot injury. Shortly after that, he bragged that, on one recent "good" day, he was once again able to chase his wife around the dining room table.

He also told me that on the following day, a day that featured an appointment with the dentist, he spent the day curled on the living room floor in a fetal position, *painfully* rigid, tremoring, drooling, unable to speak or move…until his wife cancelled the appointment.

Prior to his receiving successful Yin Tui Na treatment for his dissociated foot injury, his Parkinson's symptoms were fairly mild. He could always walk and take care of

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people who'd gotten the actual implants. Maybe that's why the people with the sham surgeries had the best results in terms of physical improvement.

Many of those who'd only had sham surgeries did not believe the doctors when they learned that they'd had the sham. And you can see why: if the sham surgery had given them such improved quality of life, the obvious implication was that a significant aspect of their Parkinson's was psychological. And no one with PD, in my experience, has ever wanted to hear that, whether out loud or implied.

Placebo studies using subjects with PD in the book *Suggestible You* are examples of research being done in western medicine that has powerful applications for *understanding* what is going on in people with Parkinson's. For example, high levels of suggestibility and high levels of expectation-dependant behaviors could be related to a high level of susceptibility to self-commands – including self-induced brain behaviors. But no one seems to be looking for these applications. In this case, researchers were only interested in learning about the placebo effect, based on the wrong idea that all people, with all syndromes, will respond to placebos in the same manner.

In fact, the effectiveness of placebos very much depends on whether or not a health problem is expectation dependant – which is to say, is dopamine-reliant. Dopamine release for motor function is inherently expectation dependant. But the researchers never asked what these studies might mean regarding Parkinson's itself – they just assumed – incorrectly – that a placebo works the same in all health scenarios. They often use people with Parkinson's for these studies because the results – whether or not the person can go from *not* moving to moving – are so easy to see. But they wrongly extrapolate, concluding that the placebo effects they see in research are universal.

himself, although his movements were becoming a bit stiff and slow. In partial recovery, in his *bad* hours or bad days, his symptoms were utterly debilitating: full-blown pause mode. This patient no longer had Type II PD, but he still had a come-and-go variation of Type I.

The wrong modes

From the start, I mistakenly assumed that my patients with Parkinson's, since they obviously were not in a significant degree of parasympathetic mode (relaxed and joyful), *must* be primarily in sympathetic mode. Based on my western schooling, including my BA degree in biology, I incorrectly assumed that these were the only two possible modes. I was wrong.

My patients were in neither.

I was confused. I had a lot of questions. I also had a lot of varied resources thanks to my interests in many seemingly unrelated fields.

A bit about my academic background

I am often asked, "What books or articles did you read that led you to your conclusions?"

So I'll mention here that, while getting my BA in biology at the University of California, I also studied history of western science, ancient and modern. This field of study quickly dispels the notion that scientific breakthroughs flow out of the so-called "scientific method."

The scientific method is a way to bolster the status quo. The scientific method *cannot* create a paradigm shift. In fact, due to the premise that one cannot prove a negative, the scientific method often cannot even be used to *conclusively* prove that a given hypothesis is *wrong*: only that another theory might be *more right*.

None of the great paradigm shifts in science, since the days of the ancient Greeks up to the present day Nobel prize winners in science, has come about via the scientific method. Rather the opposite: when we come up against something where the outliers and the exceptions start to predominate over a current theory, people on the leading edge of research have to throw away the theory.

Very often, it's a dream or a hunch from out of left field that provides new solutions and also accounts for the outliers. But despite the better fit of the new hypothesis, overcoming the comfort of the previous, though inadequate, theory is often a slog.

As Mark Twain, a beloved 19th century American humorist noted, there are three stages in the acceptance of a new idea: First, it is mocked. Then, it is despised. Finally, it is accepted as self-evident.

One of my first advisors on my Parkinson's project, a former professor of medical research theory, helped me understand that my work was *not* going to use the so-called "scientific method" to build on work that had gone on before, at least not western medical work in the field of Parkinson's.

He explained that, when a new concept was as far off the accepted norm as mine was, a researcher had to "bootstrap himself up." This means getting small "proposal" and "new hypotheses" articles published in top ranked, peer-reviewed journals.

I wasn't going to start out with a large, quantitative study. Instead, I would start with publishing short articles that people in my own profession could agree with even if they said to themselves something like, "I hadn't thought of it that way before, but it makes sense. And it gives answers to questions that were inexplicable using the previous paradigm."

Then, when I had published enough articles, I would be able to write bolder articles, and then books, and support my bolder statements with citations from articles I had already published in peer-reviewed journals. This is one way to grow research that is 1) based on radically new ideas and which 2) has no potential for significant profit.

Getting back to fielding questions about my academic background, I will add that after getting my BA in biology in 1974 I continued to voraciously study western medicine on my own.

In the 1980s, I got a master's degree in traditional Chinese medicine. In 2009, I earned a doctoral degree in acupuncture and oriental medicine (DAOM).

Since 1998, I am a professor of Chinese medical theory. I also am a professor of western psychology and counseling.

This combination of studying western medicine, Chinese medicine, and academic psychology, plus a life-long interest in quantum physics and history of science, with decades of study of ancient physics and metaphysics, all contributed to my unexpected findings regarding Parkinson's disease and to my other researches in Chinese medicine.

Chinese medicine theory helped me figure out that my patients were in pause mode, not in sympathetic mode. That freed me from the prison of having to fit my findings into the incorrect, western framework.

I don't remember exactly when it happened, but after at least ten, maybe even fifteen years of struggling with the limitations and implications of the western two-mode system for understanding Parkinson's, I came across pause mode while poring over a baffling English translation of a revered Chinese medical text, as mentioned in chapter one.

Only at that point did I began to suspect that backwards-flowing channel qi was not a channel aberration that *causes* Parkinson's – although when it ran non-stop, it accounted for *all* the *symptoms* of Parkinson's.

Instead, being on pause *correctly* causes the channel qi to flow backwards in the Stomach and Large Intestine channels and thus triggers the physiology of Parkinson's.

From a research standpoint, this changed everything. For people with Type I or with Type IV Parkinson's, the channel qi *wasn't* aberrant; the long-term use of pause mode in a non-emergency situation *was*.

I still didn't know that there were four different ways to get stuck on pause, that there were four kinds of idiopathic Parkinson's disease. That understanding was still several years in the future. When it came, it would explain why different people with Parkinson's responded differently to various treatments.

As I was later to figure out, all of my first six patients had either Type II or Type IV Parkinson's, the easiest types to treat: the types that respond to Yin Tui Na or that respond to the steps for coming out of normal, biological pause.

Fortunately, right along, while I was busy being confused by what was happening to my patients who slid into partial recovery, new research on brain behaviors from a wide

spectrum of western medical fields steadily poured in – research that was going to help me enormously.

Research examples

For example, new research in the field of hypnosis showed that a hypnotic command to inhibit motor function works by inhibiting the ability to *imagine* motor function. [1]

This inhibition occurs in the *exact same* brain regions in which people with Parkinson's are inhibited. [2]

Most of my patients with Parkinson's have assured me that they cannot visualize, in general, let alone imagine the sensations of themselves performing motor functions. To a large degree, most of them use silent, *word*-based thoughts and even commands to initiate movement.

Learning of this similarity of mechanism in hypnotic immobility and in Parkinson's immobility didn't help me at the time, but I remembered it when I discovered the phenomenon of self-induced pause. Then I had to wonder, could the powerful commands to "feel no pain" have served as self-hypnotic suggestions?

For another example, I discovered in Dr. Temple Grandin's book *Animals in Translation*, a book on animal behavior, research showing that people with a logic and word-based intellect ("left-brained") as opposed to sensory-dominant ("right-brained") were far less likely to develop post-traumatic stress disorder (PTSD) in response to a horrible trauma.

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[1] "The Brain under Self-control: Modulation of Inhibitory and Monitoring Cortical Networks during Hypnotic Paralysis"; Cojan, Waber, Schwartz et al; *Neuron*, Vol 62, iss 6;862-875, 25 June, 2009.

The gist of this article is that, following successful hypnotic suggestions for left-side paralysis, they saw in brain scans that "…Preparatory activation arose in right motor cortex despite left hypnotic paralysis, indicating preserved motor intentions, but with concomitant increases in precuneus regions that normally meditate [inhibit] imagery and self-awareness [proprioception].

In other words, the brain *tried* to activate movement but was stymied because it was unable to *imagine* the arm moving. "Imagine" in this context can mean "visualize or imagine the sensations of." Imagining is an important step in what is called "automatic" (dopamine-based) movement, as opposed to command-based (norepinephrine-based) movement.

[2] "A dissociation between real and simulated movements in Parkinson's disease"; Cohen, Pourcher; *Neuroreport*; June 28, 2004; 15(9):1489-92.

Based on brain scan results, "…in individuals with Parkinson's disease motor imagery is impaired…execution of overt movements is spared."

In other words, the ability to move is there, but it is blocked because the person cannot mentally imagine the movement.

Also, from a few years earlier, "Motor imagery in normal subject and in asymmetrical Parkinson's disease"; Thoois, Dominey, Decety, et al; *Neurology*; Oct 10, 2000; 996-1002. This article reports, "Previous work in PD has shown that bradykinesia (motor slowness) is associated with slowness of motor imagery."…"Conclusions: In patients with PD, brain activation during motor imagery is abnormal."

Instead, people who were word-dominant often created a mental story line that put the trauma into a controllable mental compartment.[1]

Most of my PD patients were extremely left-brained, at least in their own opinions and in the opinions of their friends and spouses. Most of them had some powerful physical or emotional trauma in their past. They had dealt with it either by staying dissociated from the specific injury or event *or* by commanding themselves to feel no pain in general.

These two *very* different mental processes are both left-brain processes.

This mental ability to control and/or not experience the results of trauma seemed to dovetail with some of the elements of the Parkinson's personality, as well as the idea of being stuck in a wrong neurological mode.

I collected stacks of research articles that seemed related to some of the behaviors I'd seem in my patients, even if they weren't *overtly* linked to Parkinson's. As demonstrated in the above two examples and in the placebo-research footnote, the new research was usually *not* directed at Parkinson's, specifically, but very often had *implications* for Parkinson's disease, eventually contributing support for my paradigm-shifting hypotheses.

I am *not* a born paradigm shifter. At the time I started my Parkinson's research I was extremely shy and my live-by rule was "Don't make waves."

No one would have been happier than me if I could have found a way to turn off Parkinson's disease while staying in the comfortable confines of western medicine theory or even modern Chinese medicine. But even while western research was frequently if indirectly providing proofs for my hypotheses, I was moving steadily further away from the western medicine dead-dopamine cell model for Parkinson's disease and the formulaic, one-size-fits-all protocols of modern, Chinese government-approved Chinese medicine, and moving closer to the fringe realm of ancient, channel Qi-based Chinese medicine and ancient Vedic (from India) theory.

But even while so many unrelated fields in medicine and psychology were doing research that seemed to have *glaring* significance for the subject of Parkinson's, no one seemed to be connecting the dots.

Slowly putting together ancient eastern medical theory and modern western research, I kept connecting widely scattered dots until a picture emerged. For most of my patients, their emotional traits and intellect, as well as their symptoms and their inexplicable recoveries – or failures to recover – all began to make sense…but only if I was willing to consider a psychological component.

Eventually, hesitatingly, grudgingly, based on the western research as well as the older eastern theories, I headed off into a radically different hypothesis for what causes Parkinson's disease.

Meanwhile, on Parkinson's from dissociation, not pause

At the same time, the methods I was developing to help other, non-PD patients self-diagnose and heal from dissociation from injured body parts were also making it clear that a small percent of my *Parkinson's* patients were *not* exhibiting the *mental* behaviors

[1] "The Nature of Traumatic Memories: A 4-T fMRI Functional Connectivity Analysis"; Ruth Lanius et al; *American Journal of Psychiatry* 161, no. 1 (January 2004); 36-44.

associated with pause – but they *were* manifesting symptoms of dissociation and only the *physical* symptoms of pause.

These patients appeared to have merely dissociated from ankle or foot injuries, which had then failed to fully heal. Due to the location of their injuries, failure to heal had, over decades, caused electrical aberrations in the leg, including backwards flow in the Stomach channel. Eventually, the domino effect of these aberrations was an electrical flow pattern that resembled very closely that of pause...and which therefore caused the physical, but not the mental, symptoms of pause.

I realized in retrospect, years later, that the PD patients who had recovered easily in response to Yin Tui Na treatments had only had dissociated injuries – they had *never* told themselves to feel no pain or embrace numbness. Looking back at my notes about their self-described attitudes and personality traits helped confirm that they had *not* mentally or emotionally been using pause mode. Instead, their electrical circuits were running in the pause patterns because of injury *only*, causing the physical symptoms of pause but with no or few signs of the "Parkinson's personality."[1]

Yin Tui Na causing partial recovery

In people with *only* Type II PD, whose channels were flowing in the pause patterns *only* because of a dissociated, unhealed foot injury, the healing of the injury via Yin Tui Na caused the Parkinson's to cease.

In people with self-induced pause *and* a foot or ankle injury, Yin Tui Na therapy *did* allow their foot injuries to heal.

In people with self-induced pause *and* a foot or ankle injury, Yin Tui Na therapy did *not* turn off pause. The treatment helped heal the foot and only the foot. But because they were still using pause mode, the foot healing often triggered unexpected, even bizarre emotional and mental behaviors while their channel qi continued to run backwards *when* they were feeling wary. Which was most of the time.

The reason that most people with self-induced pause also have unhealed foot injuries was discussed earlier. You will recall that it has to do with inhibition of non-

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[1] The disproportionate number of people who recovered quickly and easily in my first few years and who in retrospect had only Type II PD might be explained by the fact that, in the early years of my acupuncture practice, many if not most of my clients were in the music department at the local university or were their friends and fellow musicians around the bay area.

At one point, I even hypothesized that maybe people who are classical music performers have an advantage in recovering from Parkinson's.

I suspect now that I was confusing cause and effect. A person who is on pause might not be able to become a top level, expressive musician because being on pause inhibits the ability to feel, let alone convey, one's heart-feelings via "voluntary" as opposed to command-based movements. A person on pause might be able to cerebrally compose, or produce electric melodies. A person on pause might find it increasingly hard to perform empathetically heart-stirring music.

Therefore, my patients who were older, professional, classical musicians were unlikely to be on self-induced pause. This lucky coincidence meant that I first worked with people who had easy-to-treat Type II and Type IV PD.

If I had started out with people with Type I, I would *never* have seen recoveries. I would never have obsessively embarked on what became the Parkinson's Recovery Project.

crucial healing when on pause…and nearly everyone hurts the foot or turns the ankle at some time or another.

For an example of what I mean by "bizarre" physical and emotional behaviors of people in partial recovery, I already shared the case study of the person who could merrily chase his wife on a good day and was immobilized by fear of the dentist on a bad day. Here is another example:

I had been working with a patient whose foot injury had healed up over the last few weeks. Her Parkinson's symptoms had suddenly become greatly magnified in response to negative thoughts. Oppositely, they now might cease altogether for hours at a time when she felt safe or calm.

During a subsequent session with her, I asked her to try using positive affirmations, a type of medical qi gong, during the coming week. I offered a few examples: "I am safe enough to relax all the time, I am safe enough to be calm all the time." I repeated this several times. She repeated them with me.

When she was leaving, with her hand on the doorknob, I asked, "You remember the affirmations?"

She replied, with a strange glaze over her eyes, "Yes. I cannot be safe enough to relax, ever. I cannot be safe enough to be calm, ever." And then her mouth smiled at me in a strange, mechanical manner…nothing like the genuine smiles that had already replaced her previously "masked" facial expression.

Although the look in her eye gave me the creeps, I wasn't too surprised at her response. Partial recovery symptoms often included bouts of mental incomprehension, twisting of reality, and/or even short windows of amnesia. And these symptoms *only* started up after the old foot injuries started to heal.

I wasn't the only one noticing these bizarre changes. Spouses and friends also observed, in response to my patients' foot injuries healing up, the new, drastic come-and-go changes in symptoms, as well as the appearance of glazed eyes, periods of severe illogic and even amnesia lasting up to an hour or so following events such as a gentle inquiry as to whether or not the person's symptoms might have a psychological component.

The name "partial recovery" didn't do justice to these inexplicable symptom changes and the eerie, even creepy mental changes that occurred in response to healing of the foot, but I had to call it something.

At the time, because the foot injury had healed and the channel qi was now able to flow correctly *some* of the time, I sincerely thought that these people had merely hit a bump in the road. The bump seemed psychological. I assumed that, with time, they would get over their psychological bumps and join the others in full recovery.

In a way, I was right: these people had come up against psychological bumps. In a way, I was wrong: time alone would not help them to overcome those bumps.

As it turned out, nothing was going to *lastingly* change for the better unless they made some very specific changes in their mental habits, changes that would stimulate their long-dormant striata and thalami.

Chapter six

Worrisome observations

Didn't want to go there

I *really* did not want to find a psychological component. I wanted a straightforward biological explanation for what causes Parkinson's and a logical, corresponding cure.

I thought I'd found one, but it only seemed to apply to about five percent of my patients with Parkinson's.

I also knew that most people with Parkinson's did *not* want a mental component to be contributing to their symptoms. Not At All.

Nearly all of my Parkinson's patients had told me, so many times, in so much detail, how their symptoms increased or decreased in response to various stresses or relaxation methods, or even the *anticipation* of stress or relaxation, and yet most of them were *adamant* that their syndrome was purely physical: not mental.

For an example of the mental component being dominant in many of my PD patients, one of them was astonished to learn from me that some people have PD symptoms even after they come home from work. "Why would they have symptoms once they're done for the day?"

He wasn't even in partial recovery. From the *start*, he had *never* had symptoms after six in the evening. As soon as he sat down in his lounge chair at 6:00 p.m., he was symptom free for the rest of the evening. He refused to consider that there might be a mental component to his Parkinson's.

As for my patients who were now in partial recovery, my team members, the patients' spouses and friends, and I were all seeing new, mood dependent extreme come-and-go Parkinson's symptoms and personality swings. As for the selective amnesia and glazed eyes in response to suggestions about positive affirmations or to casual remarks about the symptoms appearing to have psychological triggers, that looked a heck of a lot like the behaviors of a person using two personalities.

As a professor of psychology, I know that *most* psychologists consider multiple personality disorder (now known officially as dissociative identity disorder) to be incurable. The *others* say that it doesn't exist.

Many big names in psychology feel that this is the realm of freaks and frauds. I did *not* want to consider the possibility of multiple personalities.

Then again, why not? Was I afraid of the "messiness" of a psychological component such as multiple personality disorder? Was I afraid of the "incurability" label? Parkinson's was considered incurable and yet it clearly was not – for some people. So why should an "incurable" label worry me?

Was I also concerned about the research credibility aspect?

When a syndrome has a psychological component, it is much harder to isolate any research variables. Double-blind studies often don't make sense for such syndromes. The big cohort studies with thousands of people who are all similar or who all have a similar syndrome are much harder to put together if the problem is mind-based: for example, if

the problem is a highly individualized mental construct designed in secret to keep the person from feeling pain, it might be nearly impossible to design a meaningful cohort study.

The single-case study

For many reasons, in the field of psychology, the single-case study, meaning a case report with just one patient, is considered a valid and important type of research.[1]

In the field of psychology, ever since the 1970s, the recognition of and significance of single case studies has been growing. Because each person responds to psychological therapies in a somewhat unique way, journal write-ups of *individual* treatments and *individual* results have become a respectable source of data for researchers and for people trying to find their way out of pathologies that have a mental component.

Then, further down the research road, by building on single case studies and observing how the methods used with one person get results, or not, when used with another person, it is possible to build up enough data to show that, *in general*, a certain percentage of people *do* or do *not* tend to have a particular response to a given therapy.

Cognitive behavioral therapy: an example

Since being developed in the 1970s and '80s, the original type of cognitive behavioral therapy has been tested on thousands of people with depression. It has been shown to be effective in a majority of cases, and to be more effective than drug-based therapies.

The researchers who developed cognitive behavioral therapy, a therapy now recognized as a powerful tool in abating depression, started with single-case studies. Over time, compilations of single-case studies showed that, while not perfect, cognitive behavioral therapy had a far higher success rate in treating depression than did the pharmaceutical approach.

As cognitive behavioral therapy became more widely accepted, further research studies were done to investigate whether people could use the methods effectively by working on their own, from a book. It turned out that the percent of people that recovered from depression via working from a book on cognitive behavioral therapy was as high or higher than the number of people who recovered by working with a trained therapist. And both methods had a higher success rate than treatments using anti-depressant medications.[2]

The book-based approach, called "bibliotherapy," is now considered a valid therapy track. In its early years, bibliotherapy was scorned by many practitioners of psychological talk therapies such as Freudian analysis – therapies now proven to *increase* negative thought patterns.

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[1] *Single Case Experimental Designs: Strategies for Studying Behavioral Change*; Herson, Michel, PhD, Barlow, David PhD; Pergamon Press; Elmsford, NY; 1976; ISBN 0-08-0019512-1.

[2] *Feeling Good: the new mood therapy*; David Burns, MD; HarperCollins Publishers; 1980; p. xxviii. ISBN 978-0-380-81033-8.

Compiled research that builds upon single-case studies is almost the opposite of a double-blind study: often, everyone involved knows who is doing what process.

Single-case studies are also considered highly valuable when dealing with medical problems that are incurable. Even if only one person in fifty in a very small study recovers from an "incurable illness," that research is considered to be highly significant…*especially* if those unexpected results, however skimpy, can be replicated by others.

At the time cognitive behavioral therapy was being developed, severe, disabling depression was considered to be more or less incurable. Therefore, even single case studies in which people recovered were of value.

After many years and many single-case studies, when cognitive behavioral therapy was firmly accepted by a number of researchers in the field, then the larger, quantitative studies were done. These studies verified that the therapy was, in fact, helpful for a large percent of people.

This slow approach to confirmation takes years. Along the way, the founders of this therapy were, of course, despised by the people who promoted the pharmaceutical approach to what they were then calling "chemical depression." Some pro-drugs people even claimed that cognitive behavioral therapy, by changing the patient's attitude, amounted to "blaming the victim."

When I started on the Parkinson's Recovery Project, I did not know any of the current standards for research in the field of psychology. Fortunately, one of my earliest advisors on my research, Dr. Fred Jones, PhD, had a background in teaching both medical research and psychology research. He suggested that I focus on single case studies in the beginning.

Readers of the first few editions of this book will remember that those early books featured primarily single case studies, and reflected much less *technical* understanding of what was physiologically actually happening in the people who recovered from Parkinson's.

The single case studies were hugely important over the long run. They provoked in me and in others a curiosity and even a passion to find the common electrical, physiological, and mental denominators in people with Parkinson's, so as to make sense of those intriguing single case studies.

Didn't want a psychological component

I really didn't want to find a mental component in people with Parkinson's disease, but there clearly was. The weird behaviors that looked a lot like a second personality – one that didn't always know what the other personality was doing – was the strongest proof yet that something psychological was going on.

I needed a name for the mental process driving the glazed eyed, briefly, or intermittently, amnesiac personality. I settled on "the Blocker."

Then I described the Blocker-driven behaviors in *Stuck on Pause,* a book that I posted online even though the book was not completely finished. Many people in partial recovery who were not my personal patients but who were following my work online sent me emails after reading *Stuck on Pause*. They described their own interactions with their very real and sometimes scary Blocker when they tried doing positive thinking or positive

affirmations. Nobody disagreed with the name I had given to this strange force that seemed to be living in the brain and resisting all attempts to alter the present course.

Some people said the Blocker manifested as a voice. Many people with religious tendencies said the Blocker was a devil. A patient from southeast Asia said it was a green ghost, (a traditional form of demon from that part of the world).

In some patients, the Blocker was silent, but manifested as threatening signs or cue cards. For example, when one patient tried to therapeutically imagine feeling the chest expansion characteristic of parasympathetic mode, his saw in his mind's eye that his heart had little signs all over it saying, "Stop!" "Don't do this!" and "No!"

Another, trying to imagine that her heart and head were filled with bright and happy light, saw that the inside of her mind was a cave with barbed wire and a big "Keep out" sign around the opening of the cave.

As an aside, although inhibition of *positive* visualizations is normal during pause, visualization of *negative* scenarios is usually effortless. This makes good biological sense: when a person is motionless following an almost deadly attack, that is not the time for him to start imagining rainbows and jolly dance moves. Being preoccupied with dangerous outcomes might even help the injured person remain curled up and wary.

Happily, when people recover from Parkinson's, they often find that they can once again see fanciful images in the clouds or see "faces" in the bark or the light-layered leaves of a tree.

But getting back to the Blocker, it was a common problem for many people in partial recovery who were trying to do mind- and mood-changing self-help and/or cognitive behavioral therapies in order to turn off their come-and-go pathological degree of wariness, wariness that brought on the PD symptoms.

As a licensed acupuncturist, psychological counseling is not within my scope of practice. However, I am legally allowed to instruct a patient in self-change therapies so long as they fit under the description of medical Qi Gong.

Medical Qi Gong is the art of using visualization, affirmations, the sensations of energy, and sometimes physical movements, in order to bring about changes in the way a person uses or perceives his physical and mental energy.

I encouraged my patients in partial recovery to try various self-help therapies, both common and fringe, including Qi Gong and bibliotherapy-type cognitive behavioral therapy, but none of the therapies proved helpful. Some of my patients saw therapists or hypnotherapists. Nothing seemed to help.

Worse, repeated attempts at positive mental changes, even short affirmations, seemed to strengthen and embolden the Blocker.

Sometimes, recovery was spurned

In a few cases, I had patients who had been in partial recovery call me on the phone to say that their Parkinson's was now completely gone, and they were moving normally, even dancing wildly, and that they were going to enjoy it because in a few days or in a specific number of hours, the Parkinson's would return and would *never* leave again. This self-fulfilling prophesy would play out, just as predicted.

I would plead on the phone with these people saying, "If it's gone it doesn't *need* to come back!" They would tell me that they *couldn't* permanently recover for various

reasons. The single *most* common reason was something along the lines of "I don't deserve to recover."

They would often add something like, "I'm being given one last taste of freedom as a gift and then I'll have Parkinson's forever, so I won't be working with you anymore. I don't want to be wasting your time."

One patient in partial recovery, after playing tennis again with several girlfriends after years of not being able to because of the muscle rigidity and immobility of advanced Parkinson's, was one of the many who declared to me that she didn't "deserve" to recover. She was never able to play tennis again and even said, "I shouldn't have been able to play tennis like that."

She was a highly respected child psychologist with a history of early childhood sexual abuse from her father who, "was a very good man…I shouldn't have such negative memories about him."

After her day of playing excellent tennis, she very quickly became unable to walk, move her arms or torso or even feed herself. She was much worse off than she had been before her Yin Tui Na-induced respite from Parkinson's.

However, her husband thought I should know that, after he put her into bed each night, she could move *perfectly normally* while she adjusted her pillow and got herself comfortable.

She interjected, snappishly, "I don't *have* to have Parkinson's once I've gotten into bed. I only need to have it as a punishment when I'm trying to show off by having mobility." She was adamant that her symptoms did not have a psychological component.

I repeat, she had a doctoral degree in psychology and was a highly respected psychotherapist.

Weirdest of all

Moving even further into the realm of the unexpected, *several* people with Parkinson's recovered almost instantly, within minutes, in response to an epiphany.[1]

One of them had been in partial recovery for several years and was finally bedridden most of the time due to the post-Yin Tui Na, rapidly increased severity of her Parkinson's symptoms. Despite the severity of her symptoms, she recovered almost instantly after her epiphany.

In each of these recovery-by-epiphany cases, the person had finally given up on a lifetime of trying to be loving to others while staying strong, even stoic with regard to himself or herself: maintaining an extremely high degree of noble self-control. He or she had exploded in an unplanned conversation, either silent or aloud, either angry or surrendering gratefully, with God, with some other higher authority, or with a deceased loved one who had previously been silently spoken to *dutifully*, but kept at an emotional arm's length.

At some point in the harangue, the person suddenly was overwhelmed with an internal feeling of love and safety, burst into tears, took deep gulping breaths, felt shivers going down the spine, and the Parkinson's was gone. Permanently.

[1] Epiphany definition: (1) a sudden perception of the essential nature or meaning of something. (2) an intuitive grasp of reality through something (such as an event) usually simple and striking. (3) an illuminating discovery, realization, or disclosure.

Just like when a person who has been in mild shock gives a strong shiver or two to "shake it off" and then snaps out of it, these people had an epiphany that they were safe, or loved, or OK, after all, and then their Parkinson's was gone.

As noted before, following that almost *instant* change to *not* having Parkinson's, a person will still have to heal from decades of nerve inhibition, muscle rigidity, and in other areas, muscle atrophy. But the *driver* behind the Parkinson's will be clearly gone...in an instant. And it's glaringly obvious.

But instant recovery via epiphany made this syndrome seem even more like a mental disorder. You can see why my conviction of a mental component was getting stronger.

Even so, if I wasn't happy about introducing the idea of a mental component, let alone the drama of multiple personality disorder, I was flat out determined *not* to go down the path of faith healing or epiphanies.

This was hypocritical of me, as my own recovery from Parkinson's disease had been of the epiphany type.

One of my medical advisors for the Parkinson's Recovery Project told me early on that regardless of *how* I recovered, my own case should *not* be of particular importance in my overall research. I could use it for yet another set of data points, but I should be careful to *not* play too heavily on the fact that I too had recovered. My own data, my single-case study, was only important for what it contributed to the larger picture.

Because the spontaneous-epiphany style of recovery was fairly uncommon, and because it did not *seem* – at the time – to contribute to a logical understanding of Parkinson's, I minimized my own recovery in my earliest writing and kept looking for *logical* answers.

What I failed to connect, for years, was what led up to each person's epiphany: a vigorous, even bitingly honest and profoundly intimate communication with someone or something loving, invisible, and outside of and separate from oneself: behavior that can stimulate the striatum, a brain area that is inhibited in people with Parkinson's disease.

I didn't learn about that connection until I stumbled across some radically new brain research in 2015. This research showed the relationship between the thalamus and striatum and a person's ability to *feel* connected with something outside of himself and larger than himself, or mentally *communicate* with something outside of and larger than himself, respectively.

After reading about this new brain research, I went back over my notes and realized that every person with an epiphany-type recovery also had a very *long* history of regularly talking to someone outside of himself, either a spiritual figure or a departed loved one. During the epiphany, they had just taken the relationship to a higher degree of trust or let go of some mental distancing.

As I mentioned earlier, I had taken copious notes about as many aspects of my patients' lives as possible. I was able to confirm by my notes that these epiphany-recovery patients had long maintained some mental contact such as a spiritual practice of almost constantly, dutifully, talking to some aspect of the Divine or having a regular conversation with a departed mother or friend, or even a beloved deceased pet.

The epiphanies occurred – and the Parkinson's turned off – when the conversation was, for some reason, taken to a deeper, more passionate and intimate level than before so that the sense of being mutually connected was enhanced.

This surge of connection evidently stimulated the thalamus and striatum, and also allowed the person with PD to mentally let go of his ferocious, pause-amplified sense of isolation and need for self-control as he mentally allowed himself to be consoled and/or comforted by the communicant.

When this occurred, the self-induced pause immediately turned off – as pause is *supposed* to do when a person begins to 1) feel stable inside and 2) confirms that he is now safe enough to come back to life.

Suddenly, given the new striatum and thalamus research, those epiphany-type recoveries didn't seem as random and unrelated to my research as I had supposed.

For the curious

In the late 1990s, I was diagnosed by a close friend and colleague while describing to him the symptoms of Parkinson's disease.

"What are you trying to tell me?" he asked.

"I'm just telling you about how all the symptoms of Parkinson's fit with backwards-flowing channel qi on the Stomach and Large Intestine channel," I replied.

"What are you trying to tell me?" He asked again, more slowly.

"What do you mean?"

"You've just described every symptom with reference to yourself and symptoms that you say you've had increasingly for some years now. Even your tapping with your index finger when it's idle is getting more frequent."

"Oh." I followed this with a long silence. "Well, I don't have Parkinson's. Don't be ridiculous." I abruptly walked away.

I was dismayed by his diagnosis, with which I eventually had to agree.

I hadn't been able to explain to myself a recent episode with festinating gait, in which I had fallen, spread-eagled, on nasty gravel when my legs suddenly moved in slow motion and couldn't keep up with my torso during a six-mile run when I had temporarily dropped my mental focus on my legs in order to look at a passing cement truck. For the last few years, it had been getting harder and harder to turn over in bed.

I was only in my late forties, but I often needed a hand up from family members to get up off the sofa if I sat down for "too long," even though I was an avid runner.

Lately, when strolling around town, I often caught myself mirrored in the shop windows. My body was increasingly hunched over, elbows bent at a ninety-degree angle. My posture was positively distorted. I would straighten up my spine and relax my arms. But continuing my stroll, I would see in the next shop window that I was hunched and bent again.

I could no longer coordinate my hands enough to play classical piano. I fell back on playing only ragtime – where the timing didn't matter so much.

For about the past two years, my face in the morning mirror was expressionless. I would pull my mouth up into a smile to start my day. Increasingly, people on the street said in passing, "Hey, it can't be that bad…smile!" or something similar. In decades previous, total strangers had been wont to say, "You've got a beautiful smile!"

People were starting to have trouble reading my handwriting.

I made a list of my Parkinson's symptoms. I had, though to a modest degree, *all* of the classic symptoms of Parkinson's, and more than two *dozen* of the more obscure ones such as the inability to separate the second and third toes (the terminus of the

Stomach channel) on the more affected side and an oppressive feeling of pressure against the upper chest while raising the arms above the head. This pressure is due to the rigidity of the pectoral muscles – which lie on the path of the Stomach channel.

Another example of an "obscure" symptom is the very specific way in which most people with Parkinson's turn over in bed at night – while they are still able to. While laying on the back or side, one brings the knees up as far as possible towards the chest. Next, the head and shoulders are pulled forward, bringing the body as close as possible to a tight fetal position. Then, using a powerful sideways jerk, the whole body is hoicked over as a unit towards the desired side.

In comparison, a healthy person rolls over languorously, leading with the head, the shoulders, or the hip and leg and then letting the rest of the body smoothly follow.

For a few years prior to my diagnosis, I had been rolling over by using the fetal-position jerk.

I had also trained myself, over the years, to produce a very slight, constant exhalation whenever food was in my mouth, in order to prevent aspiration of food particles and the choking and throat spasms that accompany the aspiration. I had even seen a doctor, nearly fifteen years earlier, because of my frequent, spontaneous "choking on my own saliva."

The MD had assured me, "You can't choke on your own saliva. It's physically impossible."

But since starting the Parkinson's project, I had already learned from my patients that many of them had trained themselves to practice this cautionary, faint but steady exhalation while chewing. And even with this precaution, they often had spontaneous choking, "choking on their own saliva."

A person who is on pause has a very poor swallow reflex, and can easily choke on his own saliva whenever saliva slips down into the windpipe instead of into the stomach tube.

Most of the more obscure symptoms are not known to neurologists, but a thorough intake of even just a few dozen people with Parkinson's will create a long list of symptoms that are not common in the general population and very common in people with Parkinson's disease. I had nearly all of these "obscure" symptoms.

More significantly, at the time my friend offered his diagnosis, I had already seen several people recover from Parkinson's.

I had *seen* people recover. And yet, my first thought was, "I won't be like them…I won't be one of the lucky ones who can recover."

This thought startled me, and them made me furious at myself. This attitude that "I won't be one of the lucky ones" had already been stated by *most* of my new recruits from the PD support group…and had never been stated by my earlier, what turned out to be Type II or Type IV patients.

Then again, a *few* people who were stuck in self-induced pause did have a powerful conviction, based on their self-perceived ability for "mind over matter," that they would be one of the "lucky ones"…but weren't able to turn off pause despite their determination. As it turns out, an ability to emotionally surrender is a better predictor of recovery than a determination to "succeed."

In fact, the Type IV patient had laughed about the possibility of recovery: "Ha ha! Wouldn't it be funny if I recovered?!"

This breezy denial of hope in the new recruits had always filled me with resentment. After all, I had seen people recover from this syndrome. Why were all the new people being so negative? And now, here I was displaying the exact same, uncharacteristic (for me) negativity with regard to my own possibility of recovering.

Adding to the negativity, many of the new recruits wanted to meet and talk with someone who had recovered in order to combat their cynicism. In the very early days of the Parkinson's project I set up meetings so that people with PD could meet one or two of the people who had recovered.

Instead of being inspired or hopeful, these meetings *always* had the opposite effect. *Everyone* with PD said, after meeting with a recovered person, "*I'm* not like that person. *I'm* different. So *I* won't be able to recover even if *everyone else* does."

I also heard a lot of, "That person [who recovered] looks perfectly normal. Their Parkinson's couldn't have been very bad. Mine is more advanced, so *I* won't be able to recover."

I really resented this negative attitude and yet, for several days, I couldn't help myself. I sulked around, certain that I was doomed. I was different from those people who had recovered. Recovery couldn't happen to me. Rats. And just when the arc of my life had finally started looking higher and brighter than it ever had.

Then again, my diagnosis helped me understand why I had always felt as if I could finish the sentences of my patients when they were trying to describe what their symptoms felt like from the inside.

And maybe my immediate conclusion that I could not be one of the lucky ones wasn't *me*. Maybe it was part of the Parkinson's personality!

After a few days, I abruptly decided that I didn't have Parkinson's after all. All I had was doctor's disease: the sudden manifestation of symptoms in a person who is *studying* a syndrome. So I sat myself down and wrote a more thorough list of all the somewhat idiosyncratic physical and *behavioral* traits I'd noticed in myself that might *possibly* be related to Parkinson's, and the approximate date of onset.

Some of my earlier symptoms went back fifteen years or more. The classic internal pre-PD brain-spin event, triggered when the Stomach channel first short-circuits from ST-8 into GB-14 and GB-7 (see page 5), had occurred when I was seventeen years old. Some people remember this event. Others do not. I hypothesize that, if the event occurs while sleeping, one will not remember it.

One symptom went back to around age seven or eight. I secretly, now and then, pulled off the toenail on my right-side smallest toe all the way to the root because for the next few days the throbbing sensation gave me a rare awareness of my increasingly numb foot, an awareness that I found strangely reassuring.

The day after making my new, longer list, I started asking my Parkinson's volunteers if they had ever experienced some of the symptoms on my new list.

I asked my first patient of the day, "Did you ever pull off your little toenail all the way to the root because the throbbing sensation was a relief, somehow?"

His wife exclaimed, "No! That's gross!"

The patient gaped. Several seconds passed before he whispered, "How did you know?"

The commonality of that symptom turned out to be *very* low. Most of my patients with PD had *not* done this. It was only a happy chance that my first patient of the day had

done the same thing as me, for the same reason. Still, by the end of the week, I had learned that many of my idiosyncratic behaviors were *not* so unusual in people with Parkinson's. Many of my symptoms and behaviors went back at least ten years, if not all the way back into childhood.

I had only been doing the Parkinson's project for less than two years. So my symptoms weren't doctor's disease.

I had Parkinson's.

I decided that I might be able to take advantage of having Parkinson's to better help my patients. After all, the way the western medical theories described this syndrome did *not* fit with how we all – I had to say "we" now – *felt* inside.

But I was still feeling depressed, uncharacteristically bitter towards God, and certain that I would not recover. I was ashamed of all three.

As a medical professional, I had always seen that the patients who do the best are the ones who find some way to look on the bright side, some way to be grateful in spite of the way things are playing out. I wanted to be like those people.

And so, early one afternoon, more than a week after my friend's diagnosing me with Parkinson's, I forced myself go into my meditation room for a long talk with God. I told myself I couldn't leave the room until I was genuinely grateful. I didn't know or care what I would be grateful for, specifically, but I did know that when inexplicable difficulties arise, I should always seek the kindest possible explanation, in order to be closest to the truth.

I started talking to God.

A few hours later, joy radiating from my tear-swollen face, I staggered from the room. I was indescribably tired. I was also spilling over with gratitude for the many, many positive things in my life and especially for the utterly unexpected foreign-feeling waves of joy that had surged within me, swamping me and repeatedly overwhelming me during my hours-long one-way "conversation" with God.

It was mid-afternoon, but I went to bed and slept deeply.

When I woke up from my atypical mid-day nap, I realized that something inside me was gone. The profound stillness (lack of internal tremor) made me think that I must have died in my sleep.

Then I inhaled. So I wasn't dead. But I was still just as motionless inside as before I'd taken the breath. I breathed again, just to test the waters.

Since I was breathing, I concluded that I wasn't dead, even though the internal stillness felt utterly unfamiliar, as if I was in an altered mental state.

The passing idea that "I must be dead" is fairly common in people with PD when their *internal* tremor ceases, although others have far less dramatic thoughts, such as, "The washing machine inside me just turned off."

I could sense energy moving over my right foot, from St-42 over to the big toe along the Spleen channel – a healthy pathway and one that had been numb in me probably since my foot was smashed in a car door when I was around five years old. After my initial scream of shock when the car door shut, I had not cried from the pain, knowing that if I made a fuss, my sister who'd closed the door and maybe I, too, would be beaten by my mother.

I had taught myself to be numb to pain long before that foot injury.

Now, still lying in bed and *not* feeling agitation inside (!), I felt physically light inside and filled with inexplicable joy. I suspected that the thing that was gone was the thing that had driven the Parkinson's symptoms. I was filled with new energy and radiance and my muscles were strangely soft. I *knew* I no longer had Parkinson's disease.

But what was going to happen to my research? I had already decided that one of the many reasons to be grateful for having Parkinson's was because it could help others via my research project.

I marched back into the meditation room. I told God I didn't want to recover unless everyone else could.

I sensed silent words that seemed to come from all around me. "Everyone can. Most people do not want to."

Suddenly much less confident, I told the voice that I didn't believe him.

And I set out to prove him wrong.

Years later, when the Blockers showed up, and second personalities, and I saw how many people were determined that they didn't deserve to recover or couldn't recover even if everyone else did, I had to wonder, "Is this what that Voice was talking about when it said that most people do not want to recover?"

I now understand that, while most people do want to recover from the symptoms of Parkinson's disease, most people do *not* especially want to change the personality traits that they think are keeping themselves free from harm. They do not want to get rid of the personality they created that is stronger, faster, and somewhat impervious to pain: the underlying *cause* of most cases of Parkinson's disease.

But getting back to the problem of the epiphanies, the epiphany model didn't really help further my research...right away. I did try, for a few years, to encourage people to talk vigorously to God in hopes of triggering an epiphany, but no one was able to do this just by mentally reciting words to the silent walls.

As it turns out, constantly, silently talking to the right kind of invisible friend in a loving and casual way *does* help lead to recovery because it stimulates the striatum. As you will read in the next chapter, this is *one* of the two techniques that, used together, can turn off self-induced pause.

How long does it take?

Once in a while, self-induced pause turns off in a matter of moments after starting to talk silently to a loved one. But most of my patients have spent months, sometimes years, before these techniques become habitual and strong enough to be effective.

But whether the response is quick or takes a few years these techniques that involve working with an invisible, trusted loved one – one with whom the patient can laugh – *can* eventually turn off self-induced pause. Talking to a distant God who is unknown, emotionally, will *not* do the trick.

That finding was years in the future, and until I understood the brain biology at work when silently talking to a trusted friend, one with whom the patient could *laugh* and be utterly honest, I didn't understand how to *teach* people how to have either a healing epiphany *or* have what should be a most natural, effortless type of silent conversation with someone or something that's invisible, loving, and located outside of oneself.

"Not going to die…"

I've wandered far off the main point of this chapter. Returning to the subject of my growing bafflement around partial recovery symptoms, I also didn't know what to make of some symptoms that occurred when people *did* recover.

The *immediate* behaviors following recovery were similar in nearly all patients, and in a most unexpected way. People who had been on pause mode or in partial recovery, when they suddenly, fully recovered often laughingly, light-heartedly, said something like, "Ha ha! I was doing this to myself!"

To appreciate this, you need to know how hard it is for most of the people I've known with Parkinson's to un-self-consciously laugh at themselves for having made a mistake. For example, many of my patients, when asked, admitted that, if they were walking down the street and suddenly remembered that they had to turn around and go back the other way, would not pivot, but would continue walking straight, turn at the corner, and go all the way around the block so that none of the strangers around them would perceive them as having made a mistake or as "behaving strangely" by turning around in mid-stride.

During the week that followed the recovery event, in response to some situation that previously would have been a *really* big deal, even a *shameful* really big deal, such as arriving a few minutes late to an appointment, many people said to me lightly, and I quote, "So what. It's not like someone's going to die, after all."

A post-recovery statement about "not going to die" was so common that, in one week, I heard it *twice*. Two people who, a week earlier, would never have made that statement because *everything* was crucial and everything should be done *right*, said laughingly something like, "It's not as if anyone's going to *die*…"

This type of statement was *so* common that, for a short while, I asked people who were stuck in partial recovery to say this phrase or one like it as often as possible, in case it might trigger turning off the Parkinson's. It didn't, of course.

As an example of how utterly uncharacteristic this phrase was for these people, one of them laughingly told me that she'd talked to an old friend on the phone the evening before. She hadn't seen her friend in a very long time and the friend had no idea about the Parkinson's diagnosis.

Her friend had asked what her plans were for the upcoming season, and my newly recovered patient had replied, "Oh, I don't know, I'll see what I want to do when the time comes."

A long pause had followed, and then her friend had asked carefully, "Are you OK?"

My patient laughed again and told me, "I've always been the Great Planner. I always know exactly what I'm going to do and when I'm going to do it. When I told her I didn't know just what I'd be doing next season, she was shocked. She must have thought I was sick or dying. Ha, ha, ha!"

I'm trying to get across just how freaky it was to have this type of person suddenly *not* worrying about the future or *not* dreading the reveal of some character flaw, and instead casually telling me, "Ah, so what. I screwed up. (Ha ha!) It's not like anyone's going to *die,* after all…"

I eventually began to wonder if maybe these people had been using a personality that was constantly reminding them that they were on the cusp of death, and only vigilance was protecting them from dying. And keeping alive while on the cusp of death is the whole point of pause mode.

When they recovered, when pause turned off, it was as if that personality disappeared. They cheerfully *laughed* at themselves and their shortcomings and said, "It's not like someone's going to die…"

Those patients who, in retrospect, had probably been using self-induced pause, instantly underwent a glorious personality shift when they recovered. They were able to laugh at themselves, to be spontaneous in speech and action, and *feel* the expansion of their chest in response to things beautiful or poignant: they could *feel* joy again.

But sadly, most of the people in partial recovery, both my patients and my email correspondents, stayed there. By most measures, because of the increased severity of symptoms when wary – a problem that spiraled in intensity as they learned to be wary of wariness itself – they were far worse off than they had been prior to receiving the foot therapy.

I eventually came to understand that, in order to help them, I would need to find a consistent method for turning *off* their weird, often paranoid, second personalities if that's what they were – thus preventing the descent into partial recovery. Relying on epiphanies wasn't going to cut it.

But neither Chinese theory nor western research in medicine *or* psychology told me how a person could go about turning *off* what seemed to be a deeply imbedded instruction to feel no pain. And in case you're wondering, hypnosis didn't help, nor did any of the many, *many* modern techniques that my patients worked at, including techniques for changing brain behavior. Neither did the purchased gimmicks and gizmos that promised to increase inner calm or heighten energy.

The helpful Blocker

In the end, the realization that *some* people had a Blocker was actually helpful. It *forced* me to venture past the overly simplistic ideas that 1) everyone with Parkinson's had the same underlying cause and 2) the problem was purely a physical one with no psychological component.

Also, the idea that a person in partial recovery might have two personalities, an idea backed up by symptoms observed by not just me but by so many spouses and friends, and even some circumspect patients themselves, was alarming but enormously helpful in the long run.

Ordinarily, a person who is using pause mode *cannot* access parasympathetic mode at *all*. It's a biological *law*.

The possibility of two personalities, one of whom could clearly be in parasympathetic mode when conditions were "safe," provided an explanation, at least an hypothesis, for why these people were *sometimes*, temporarily, able to feel joy or able to have greatly diminished symptoms or almost none at all.

At some point it occurred to me that possibly, when *originally* commanding himself to be numb, the person had *not* actually put himself into pause mode. Instead, maybe the instruction had inadvertently created a *personality* that was on pause mode. That somewhat numb but physically powerful and intensely focused personality might

have been so helpful, especially considering some of the childhood settings that I learned about from many of my patients, that it became the *dominant* personality.

But theses patients' new, post-Yin Tui Na capacity for sensory awareness and the joy, yes the joy of having a foot that they could *feel*, that joy and feeling could only manifest in the personality that could do parasympathetic mode: the non-pause personality.

This might explain why so many people in *partial* recovery now behaved as if they had two personalities for going through life. The default personality was the one stuck on pause. This is the one that developed a Blocker if and when the chronic use of pause mode was being suspected by others.

The healthy, full-range personality was able to use parasympathetic mode, but this personality had increasingly been snoozing in the background as the pause personality had steadily gained in strength.

It might be worth mentioning that, of all my patients who *had* always remembered commanding themselves to not feel pain, including myself, not one had ever told *anyone* what they had done. The action had always been a very private event. For some it was even a shameful secret…until I came along with my nosy intake questions.

As for why people in partial recovery were now so much worse off when they needed to use their protective personality, the one that the Blocker worked for, I hypothesized it was due to the re-association with the *foot* injury while the rest of the body remained numb. That healed foot, complete with restored sensory perception, might have served as a threat to the numbing protection system that had long ago been put in place.

Biological laws, for good reason, prevent a person or animal on pause from going into parasympathetic mode until the brain signals the all-clear.

By re-associating with their old foot injuries, had people who'd been using *self-induced* pause opened the door to their healthy personality that had been waiting in the wings? Had this then threatened their body-wide game plans for staying impervious to pain? Or created some kind of brain confusion?

In response to Yin Tui Na, the foot or ankle injury(s) had re-associated – a condition that is *not supposed to happen* so long as a person is on pause. Maybe the foot part of their consciousness had opened a door to their healthy, normal, real personality, a personality that could use parasympathetic mode, and thus move normally. Which *isn't* supposed to happen if you are on pause and at risk of imminent death.

Whatever the reason, when there was any hint of risk, the people now in partial recovery certainly fell back on the pause-using personality to even a much stronger degree than they had before. And in some cases, it was now re-enforced by the Blocker.

I also hypothesized that the re-association with and healing of the old injury was, for some reason, disabling the use of the emergency override with norepinephrine, which had previously been used for motor function instead of dopamine.

You'll recall that norepinephrine, or something like it, enables a person to override the motor inhibition of pause mode. Maybe, with the foot or ankle injury now re-associated and healed, the pause-related emergency norepinephrine override was no longer as readily available, if at all. But the motor *inhibition* from pause mode was still available, and could be activated when deemed necessary.

Or maybe summoning up the emergency norepinephrine override was no longer possible because being in partial recovery meant there was no more actual emergency any more…only the *habit* of using pause.

In writing the above, I'm just pondering and proposing why some, only some, people in partial recovery don't seem to be able to activate their norepinephrine override any longer.

But *if* a person could no longer access the norepinephrine override, he might, when using the paused personality, suddenly be *horribly* immobilized, far more than before. Which is what we were seeing in some people who were in partial recovery.

And at the same time, when these people were using the healthy personality, the one that was only allowed to manifest when everything seemed completely safe – and every patient had a different idea of what constituted safe – he might move almost normally.

Meanwhile

During these years, still more people, a small percentage, completely recovered from Parkinson's in response to receiving Yin Tui Na treatments from spouses or friends or even by doing the treatments on themselves.

In people with Type II Parkinson's, this type of treatment, by helping the patient re-associate and heal from a foot or ankle injury, *does* turn off the electrical behaviors that mimic those of pause. I often learned of their recoveries via emails.

Most people with Parkinson's who tried to recover did not. They slid into partial recovery. I heard from many of them as well, also via email.

By 2011, I was semi-retired from my acupuncture practice. I was working steadily on finishing a set of textbooks for acupuncture students. When I had been a student in acupuncture college, all we had were poorly translated, confusing books that had been approved by the Chinese government. I was writing what I hoped might become replacements for, or at least additions to, these terrible texts. I was still teaching one class per semester and following closely the new research and trends in neurology and psychology. I was still working with patients two days a week and seeing a few people with Parkinson's, but no longer taking new patients.

Nearly all of the patients who'd gone into partial recovery had drifted away or lost touch. Which makes sense: I had nothing more to offer them. Some started taking antiparkinson's medications and had horrible, horrible adverse effects very quickly. If a person is in partial recovery, the medications seem to be much more powerful and addictive than if a person has body-wide idiopathic Parkinson's.

By this time, I didn't want to work with more patients until I understood what was happening in partial recovery.

In May of 2013, the Parkinson's Treatment Team agreed that we should disband because there was clearly something that we didn't understand.

Considering that, in many ways, partial recovery was worse than Parkinson's, I did NOT want to provide foot-injury treatments that made people slide into partial recovery, or make them more susceptible to the dangers of the medications *if* they decided to start taking the medications sometime down the road.

I kept the website up and running, so that the small percentage of people who were able to recover in response to Yin Tui Na might do so. But until I understood who

was a good candidate for that and who would instead end up in partial recovery, I was no longer willing to work with new patients.

I made my new position clear to the patients I was still working with. A few of them wanted to keep working with me anyway, or to stay in touch via email.

To one of these patients in particular we are all indebted. She continued to visit me once a week through the next six years, her condition steadily worsening but her determination not to use the medications remaining strong. She had seen other people going through hell with the adverse effects of the meds and was not tempted to go that route.

She felt strongly that she was supposed to keep working with me. So she did.

After her childhood foot, leg, arm, and head injuries had healed via Yin Tui Na, I mostly worked on her horribly twisted neck. She'd had various injuries to her head and neck, and week after week I did Yin Tui Na on these areas. After each session, she'd have a little less pain, for a while. Over the years, the neck slowly, slowly was able to straighten and grow stronger in a healthier position.

She called herself my guinea pig. When I had some new idea for turning off what I was now calling pause mode, she would throw herself into practicing it, and give me feedback, week after week. She was never afraid for what the future might hold, even when she had to stop working and her husband became her full-time caregiver.

I also had a few other patients that I saw less often, and I had the email brigade, people who were willing to test my treatment ideas that I sent via emails. Since, at this point, these people had recovered from their foot injuries, if any, and the remaining problem seemed to be psychological, there was no need for me to meet with these people in person.

So I was still working with some people with Parkinson's, most of them via long-distance. I had much less confidence that a cure for *everyone* with Parkinson's might come about in my lifetime.

Meanwhile, though now semi-retired, I was extremely busy. I hoped to record for posterity what I had learned so far about Parkinson's, pause mode, Yin Tui Na, and Chinese medicine in general. I had boxes of patients' file folders filled with notes written in my scrawling, near-incomprehensible shorthand plus all the information in my head that I had never written up about my patients with Parkinson's.

I wanted to get as much information into shareable form as possible. Even if I hadn't figured out how to help *most* people with Parkinson's, I hoped my observations might give the *next* researcher a leg up.

By this point, I strongly suspected that there were four ways to induce pause-type flow of channel qi but until I could come up with a way to systematically, predictably turn *off* what I had recently named self-induced pause, I couldn't even be sure that there were *only* four ways to trigger Parkinson's. Until I could figure out an effective treatment for curing all of the types of PD, I couldn't even be *sure* how many types of PD there were.

2015

The very last part of the puzzle, how to turn off self-induced pause, the activator behind Type I Parkinson's, the most common type of PD, was solved with information based on brain scan findings in the very new field of neurotheology.

The answers were found in the types of behavior that can activate the brain's thalamus and striatum, two brain areas involved with dopamine storage and release.

In 2015, I was stunned by the implications of the work of a leading neuroscientist, Andrew Newberg, MD. [1]

Using brain scans, his research showed which areas of the brain manifested an increase in activity when people of various ages and religions were asked to "think about God."

Different areas of the brain were activated in different people. The increased-activity area depended on what type of God one had.

If a person's God was a critical or vengeful God, then thinking about God would bring about increased activity in the amygdala (fear and rage center). If a person's God was knowable through word-based learning, then thinking about God increased activity in certain parietal (sides of the brain) areas.

Here's what jumped out at me: if God was feel-able and/or something one could physically resonate with, thinking about God increased activity in the *thalamus*.

If God was someone or something with whom one could communicate, then thinking of God increased activity in the *striatum*.

In Parkinson's disease and in pause mode, the thalamus has erratic, insufficient electrical signals and the striatum is inhibited.

This new information started a revolution in my brain.

Based on these findings, and working with my remaining patients, I developed two simple mental exercises that I hoped could re-awaken healthy activation in these two brain areas.

The new exercises

In the first of the two exercises, the person should treat all thoughts and spoken words throughout the day as if they were part of a silent two-way conversation with a "friend": a deceased beloved friend or relative, a "higher power," some saint or sage, or even a beloved, deceased pet.

And when I say "two-way" I mean *potentially* two-way. Grandma might be silent, but the assumption had to be that she was listening and *could* reply if and when she felt like it.

The friend had to be someone you could *laugh* with, someone who loved you in an easy-going, honest manner. It could *not* be a stern or spiritually "superior" critic.

The thoughts needed to become *conversation* and not the usual, non-stop, even obsessive, silent monologue that is characteristic of most people with Parkinson's. This

[1] *How God Changes Your Brain*, Andrew Newberg, M.D., Ballantine Books, 2010, chapter 3.

chatty duologue, I hypothesized, would stimulate the striatum, the brain area that exhibits increased activation while thinking of a God with whom you could converse.

The second exercise could begin when, in response to the first exercise, the constant communicant began to seem *real* and trustworthy enough. This sense of a real relationship took some people *years* to attain.

Once attained, a person could do the second exercise: regularly ask the communicant to feel more physically present.

For example, a patient could ask the invisible friend to palpably hold him or her, or manifest as some sort of feeling or sensation either inside or on the periphery of the patient's body. The expectation is to feel as if the friend is giving physical comfort or gentle stimulation to some part of the body that wanted support, or maybe feel that the same loving energy that is present in the friend could manifest inside the patient: a *feeling* inside.

If a person had some part of the body that was particularly vulnerable or that he really didn't like to be touched by others, he might ask his constant friend to "help me feel your presence" or "…your loving energy" in that body part.

I hoped that this might stimulate the thalamus, the brain area that exhibits increased activation if your God is something subtly energetic that you can *feel* or resonate with.

Unlike every previous attempt at imposing a psychological change upon the mind, this method did *not* seem like it would significantly challenge or try to change the self-protection behaviors of a person with self-induced pause. Therefore, I hoped, it *might* not activate or stimulate the Blocker, if any.

I had reason to think that this technique might work to jump-start the process that turns off pause. My thinking was this: if a person could *directly* stimulate his own thalamus and striatum, this should eventually create electrical brain behaviors that mimic those that occur following sensory confirmation of external safety: step two of coming out of pause.

As it turned out, I was sort of wrong about the Blocker.

As it turned out, the Blocker *might* try to prevent a person from doing these exercises. Even so, the Blocker's influence was *less* powerful against these techniques than it was with other cognitive behavioral therapies we'd tried: The Blocker was hesitant, rather than outright resistant.

But as it also turned out, I was correct about the benefits of brain stimulation from these exercises.

I asked the Parkinson's patients I was still working with to try these techniques. I also sent out short write-ups describing these techniques to the people with Parkinson's with whom I was still exchanging semi-regular emails.

Over the next few months, I was completely gratified by the slow but definite changes that some people started to experience. I was also not too surprised that some of my patients were at first completely baffled by the extremely simple suggestions. Some did not even want to risk doing something that seemed so crazy to them and/or to their Blockers.

Many needed to pose a seemingly infinite number of questions that had to be answered by me before they dared to attempt something as simple as, "Imagine your thoughts are a silent conversation with someone who you trust, who can make you laugh,

and who is not in a physical body. It might be your beloved, late grandparent. All day long, direct your thoughts towards him or her, *not* towards yourself."

But the ones who trusted me enough to do this "risky" exercise eventually noticed a difference.

The epiphany people, again

You will recall that all the people who recovered during an epiphany had spent decades or more regularly talking to a higher power or a loved one. When they spontaneously, maybe from desperation, took this long-established relationship to a deeper level, possibly in response to the new safety they felt while having their foot injuries treated, they recovered.

But the people I was working with now had *no* experience doing this kind of sincere talking to someone outside of themselves. Many didn't think it was normal. Some thought it bordered on insanity.

One of my patients was a long-time monk, a lama. He had constantly practiced talking perfunctorily to his departed guru, but never actually felt there was anyone listening or, if he was listening, was listening in a critical and superior way. In this monk's mind, he felt the guru was not a personal friend with whom a laugh could be shared. Maybe the guru was a laughing, loving friend to *other* people, but not to him. It turned out, just having a mechanical habit of talking wasn't enough. A loving, *fun* relationship with the communicant was needed.

Risky behavior

When I came up with this idea, most of my patients and correspondents with Parkinson's told me that it's not normal to talk to an invisible someone. Most of my patients with Parkinson's had, as long as they could remember, always talked in a non-stop, *internal*, fairly circular monologue, a judgmental, constantly-assessing monologue that ran on even while outwardly conversing with other people, while listening to music, while going through all the motions of life. The moments of their lives weren't *experienced* so much as *analyzed*.

To many, the idea of directing their silent, secret thoughts to someone else seemed unsafe, even alarming: the behavior of a crazy person.

I was even told by some patients that only religious people could talk to an invisible friend. (Clearly forgetting that lots of young children even talk out loud to what we call "imaginary friends.")

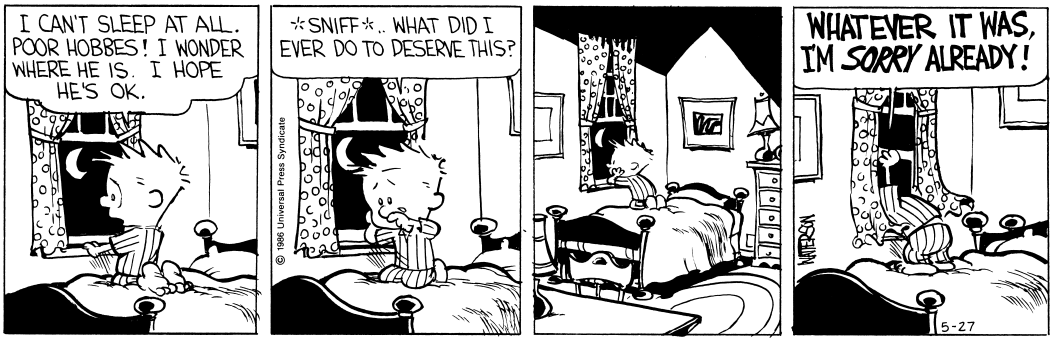
Some people with Parkinson's, completely missing the point, even asked why atheists don't all have Parkinson's if, in fact, talking to an externalized someone is normal, as if, correspondingly, *failure* to talk to someone outside the self causes Parkinson's.

This question is packed with fallacies. It assumes that only religious people talk to the universe. This isn't the case, of course.

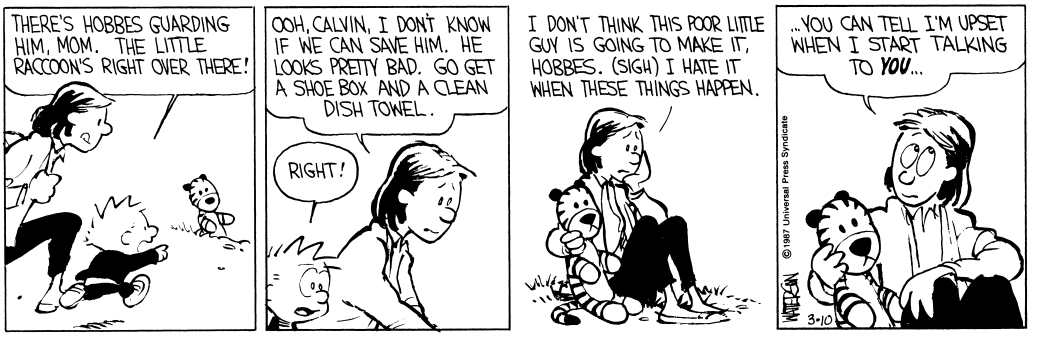
So I purchased the right to use two cartoons from Bill Watterson's famous comic strip, "Calvin and Hobbes," for this book. I thought the cartoons would serve to show that, in healthy people, this type of conversation is, in fact, the norm. I hoped that my patients would see these two cartoons and say, "Oh! I see what you mean. Talk sincerely to something outside yourself. How simple!"

A couple of cartoons

In the strip below, Calvin is desperate. Hobbes, his best friend, a stuffed tiger toy, has been missing for a week after having been run away with by a stray dog.[1]



In this next strip, Calvin is relying on his mom to save a dying baby raccoon that had been hit by a car. The mom seeks comfort in talking to Hobbes.[2]



But they *didn't* read the cartoons and say, "Oh, I understand."

Instead, most of my Parkinson's patients said things like, "So who is Calvin talking to in the first cartoon…the reader?" and "Why is Calvin's mother talking to the stuffed tiger?"

So many of my highly intelligent Parkinson's patients couldn't even understand what these characters in a comic strip were doing, or why!

One of my patients even asked me, "Are you trying to imply that the boy in the first cartoon is religious"?

No. Calvin is a self-centered six-year old. I don't think most of the comic strip readers would assume that Calvin was a particularly religious or spiritual child. However,

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[1] CALVIN AND HOBBES© 1986 Watterson. Reprinted with permission of ANDREWS MCMEEL SYNDICATION. All rights reserved.

[2] CALVIN AND HOBBES© 1987 Watterson. Reprinted with permission of ANDREWS MCMEEL SYNDICATION. All rights reserved.

when he is desperate, he instinctively screams out the window to someone or something that he knows is listening.

For decades, this beloved comic strip resonated deeply with millions of readers because the strip almost always drew on core human behaviors, behaviors that nearly all of us can relate to. The above two strips derive their poignant humor from the inherent human need to call out to or talk intimately, trustingly, to someone other than the self when seeking comfort.

When most people see this strip, they chuckle to themselves because they have found themselves in a similar position – distraught and therefore unself-consciously talking to an unknown, despite the seeming illogic of it.

But seeking comfort from others is something most of my Type I Parkinson's patients are very hesitant to do. Some have told me they would rather die than be "reduced" to asking for help.

The Parkinson's support group

Here's an illustrative vignette.

When I attended the local Parkinson's support group to recruit my first group of volunteers, I took a chair while people seated in a circle took turns telling how they were doing.

One of the women with PD said, "This week, I was at the grocery store and I needed something from a top shelf. I just couldn't reach it. Though I didn't want to, I asked a young man if he could reach it for me. He did. He was so nice. But what I want to share is that he seemed to actually be happy because he could help me. It never occurred to me that asking for help might make another person feel happy. So maybe it's OK to ask for help sometimes."

I was amazed that this most human of interactions had been eye-opening to her. I'm quite short, and asking for things on high shelves is normal for me. Then again, I too had been a long-time member of the I'd Rather Do It Myself club, so I understood her humiliation and chagrin.

The other members of the group nodded in silent accord when she said "so maybe it's OK to ask for help." But I could just tell, somehow, that everyone one of them was thinking, "Yeah, but I hope I never have to…"

While I'm on the subject of the support group, I want to share another observation from that meeting. The first time I walked into the church's community room where the Parkinson's support group met, I was instantly overwhelmed with a strange, pleasant feeling of what I now think of as resonance.

I was usually uncomfortable in large meetings of people, especially if they were strangers. But I felt physically OK, safe, even. I felt as if, for the first time in my life, I was in a roomful of people most of whom I could relate to as fellow humans.

Although I hadn't ever before used what I thought of then as a "corny" expression, I found myself thinking, "I have found my tribe."

However, I still felt that about *half* of the people sitting in the circle weren't especially resonant and safe. They were just regular people.

This isn't to say that I'm usually paranoid, but I'd always felt a *physical* discomfort, almost an agitation, in the presence of large groups of people, and in this group of people with PD, that discomfort was far less than I expected.

Shortly after the meeting started, the head honcho said, "So now, the caregivers will go over to room C, down the hall. The people with Parkinson's will stay here."

And all of the "ordinary" people, the people with whom I did *not* feel this new resonance, got up and left the room! I was now in a room full of people that felt *real*, that felt safe. For the first time in my life, I *physically* felt like I fit in. At the time, I just assumed it was because I had some sort of morally superior compassion that made me feel good about being in a room full of people fighting a powerful illness.

After I recovered and found myself losing my shyness and my discomfort around large groups of people, I concluded that the weird electrical configurations of pause had long made me feel what I now refer to as an electrical dissonance with nearly everyone else in the world except for people who had the same, pause-type electrical configurations that I had. My feelings of agitation and electrical dissonance with others ceased as soon as pause turned off.

Prior to my recovering, sometimes, in large groups of people or when given a friendly hug by others, including loved ones, the feeling of dissonance would be physically painful.

But the first time I was in a room full of people who, unknown to me, were all running the exact same electrical currents that I was, I felt myself relax. After all the non-Parkinson's people got up and left for room C, another platitude appeared in my mind: "I've come home."

Which takes me further afield, to a class I taught in Reading, England, in which about thirty people with Parkinson's were sitting in a circle going around introducing ourselves and each saying a few words about their thoughts regarding their battles with Parkinson's.

Two-thirds of the way around the circle, a woman in her thirties said, "I don't belong here. I'm not like any of you. You're all so clever and, well, you're all *different* from me. I'm just a *regular* person."

She then told us how, after injuring her shoulder at work, the next day she couldn't move her right arm. She saw an MD and was immediately diagnosed with Parkinson's. She had no other symptoms of Parkinson's other than "lack of automatic arm swing." I popped her arm bone back in the shoulder socket and she was immediately able to swing her arm for the first time in three years. She had been *inexcusably* misdiagnosed. Her mother, who had come with her, burst into tears.

But the point is, this woman was absolutely correct. Electrically, she was *not* like the other people in the room. She knew it intellectually and I suspect she physically *felt* it.

I wonder if, by being in a room filled with people with Parkinson's, she was feeling the same discomfort that I had always felt in a room full of people who *didn't* have the electric circuitry of pause.

Whether or not our bodies are resonant with those around us is an extremely important factor in how connected and how safe we feel.

Waxing philosophical, one's life goal might even be to learn to be resonant with something far larger than the mere individuals around us. When we are resonant with all the love in the universe, we are less likely to be thrown off course when in the vicinity of some individual whose currents are running a bit differently from our own.

But I digress.

Getting back to the previous point, which was the idea of conversing with someone unseen outside of oneself, people with Type I PD are very often reluctant to do this, let alone ask for help, cry out for help, or physically reach out for a comforting hand – normal human behaviors.

Many of my patients with Type I Parkinson's, by commanding themselves to feel no pain – often for *very* good reason – have inadvertently shut the door on this type of *instinctive* behavior of talking to an invisible friend or reaching out: behavior that should be *automatic* during times of stress or emotional suffering.

The command that triggers self-induced pause also serves to deaden a person's innate ability to somatically *feel* and to have a sense of being connected with something outside of oneself. It also inhibits the ability to have a sense of communication with someone/something outside of oneself.

Biologically speaking, the command to not feel and the resulting activation of pause seems to inhibit the thalamus and striatum, two of the main dopamine-related centers in the brain…areas that are stimulated when a person feels that he can have a somatic (physical, inside the body) connection or *resonance* with something outside of himself, or feels that he can *communicate* with something outside of himself.

Looking ahead, in terms of channel theory, pause mode inhibits the flow of energy through the midline of the brain, along which the striatum and thalamus are located. In pause, the amperage in the current flowing through the midbrain is greatly decreased. The amperage levels of the electrical currents on the left and rights sides of the brain, including the left and right side amygdala (fear and rage centers), are correspondingly increased.

And now, getting back to 2015, when I shared my new ideas for self-stimulation of the thalamus and striatum with my patients, most of them were eventually willing to give it a try even though it made no sense or even seemed a bit dangerous – or even bordered on the insane.

Within just a few months, for some of them, they noticed that their social and mental behaviors were slowly changing…and their symptoms of Parkinson's were a bit less constant, and a bit less powerful.

An instant recovery

One new patient with very early Parkinson's symptoms unexpectedly went through the behaviors that turn off pause within *minutes* of starting to talk silently with his lately deceased grandmother.

Most people, after turning off pause, don't have recovery symptoms for a few weeks or even a few months. But this patient started having recovery symptoms almost immediately. He exhibited a bout of the infant-like facial spasms within twenty minutes of turning off pause, while he was still there in my office. He was alarmed, and said, "Those things [recovery symptoms] are actually going to happen? I didn't believe you!

Within just a few days he found himself grabbing his burning, aching biceps to try and prevent the automatic arm swings that were now occurring thousands of times a day, one healthy arm swing with every footfall.

I should note that this person was very young: in his late twenties. He also had long forced himself to keep a positive, trusting outlook even as he found himself

increasingly inclined to be wary and non-trusting. He had constantly, actively, rejected the wary voice in his mind.

For others, becoming aware of internal changes after starting to talk to an invisible friend took much, much longer: years.

But those who stuck with it, teaching themselves to constantly direct their thoughts and questions to a loving someone outside of themselves, slowly found themselves feeling safer, eventually, over the months and in some cases years. They were learning to keep themselves constantly – and I mean constantly – as if in the presence of someone or something that loved them and with whom they could laugh and be themselves, warts and all.

This felt drastically new to all of them.

For many, it felt ridiculous, at first. They were embarrassed to be "talking to nothing." And yet, if they stuck with it until their friend seemed real and then eventually added the thalamus-stimulating request of "let me *feel* your presence" or "let me *feel* your joy and love inside of me," their long-running use of pause mode turned itself off.

In a few cases, some aberrant electrical currents were still running from mere habit, but these were easily corrected. Learning how to track down and make the simple corrections to these residual stuck channel patterns will be discussed later.

It seemed as if, when they finally felt internally stable and safe enough to come back to life, pause turned off.

The Parkinson's ceased.

Recovery symptoms began.

Chapter eight

The biology of pause

Setting aside, for now, the unfinished discussion of recovery-related questions and the chronicle of my research project, this chapter presents a more extensive discussion of the electrical behaviors of pause mode.

This chapter was lifted, with a few modifications [and bracketed additions for this Parkinson's book], from chapter fourteen of my book *Tracking the Dragon*. *Tracking the Dragon*, in addition to teaching one how to feel channel qi, describes the channel schematics for each of the four neurological modes and explains, in everyday English, many of the theories of Chinese medicine.

Extracts from Tracking the Dragon, chapter fourteen

Pause mode is the correct, healthy neurological response to severe blood loss or other near-death traumas such as concussion or potentially mortal injuries. Pause might also be triggered by excessive perforation of the skin, which can include self-cutting and, to a mild degree, excessive acupuncture needling.

Pause is not an all-or-nothing mode. Like the other three modes – parasympathetic, sympathetic, and sleep – pause can manifest on a continuum from mild to strong and in combination with sleep or sympathetic modes. At the mild end, symptoms of pause might manifest as slightly altered consciousness and the release of pain-reducing endorphins. At the extreme end, immobility and/or coma might occur.

Physiological changes during pause

During pause one might experience, to varying degrees, a *decrease* in heart rate, shallower or slowed breathing, and a *drop* in blood pressure. Blood might leave the skin and muscles and shunt deeply interiorly to the spine and brain – *not* to the heart and lungs. The skin might be clammy. Temperature regulation might be poor, especially in the extremities.

These alterations in physiology might be life saving. For example, the inhibition of heart-force *greatly* slows the rate and strength at which blood is pumped. If too many blood vessels have been broken, this inhibition of heart rate and strength might help reduce further blood loss.

The release of endorphins and a concomitant decrease in awareness of pain is common.

The body might curl into a fetal position. This fetal positioning occurs because, in the parts of the body where channel qi suddenly flows *backwards*, the muscles become rigid. Where channel qi is *cut off*, muscles become weak. The backwards and the turned-off channel qi flow over paired, oppositional muscle-sets can cause muscle rigidity and relaxation in opposing muscles. This can cause the pulling up of the legs and maybe a curling in of the torso and pulling forward of the neck: the fetal-esque position observed in some people who are on pause.

The voice, if any, might be faint.

The senses of taste and smell might diminish greatly or become altered. The senses of hearing and vision might be *heightened* while on pause, although the *type* of hearing and vision is more analytical and behaves more as it does in sympathetic mode.

For example, in sympathetic or pause mode, a person is *less* able to see fanciful images in the clouds or in tree bark. The ability to visualize positive images or playfully imagine shapes and faces while gazing at something is a feature of *parasympathetic* mode. The ability to do this type of playful imagining diminishes as a person slides into a higher degree of sympathetic mode or lurches into pause.

Sound perception also shifts during sympathetic mode and pause. Hearing becomes more attuned to sounds of possible danger. Background sounds can become a source of agitation instead of pleasure.

For example, if a person gets lost while driving and becomes concerned, one of the *first* things he does is turn off the music in the car. As the driver transitions from parasympathetic-dominant to sympathetic-dominant, the music transitions from pleasant to distracting, even annoying.

[Many of my PD patients have told me something like they used to enjoy listening to music, but that now it distracts them. "Distracts you from what?" I reply. "From the things I need to be thinking [or worrying] about," is the most common response.]

In parasympathetic mode, a sudden burst of bird song might evoke laughter, or at least a smile. In a high degree of sympathetic or pause mode, the same sudden song might elicit a startle response or a sharp intake of the breath.

Other pause-specific behaviors also occur, but this should be enough get you started – to give you a mental picture of a person who is on pause, and let you recognize that this is *not* a variant of sympathetic mode. It also is *not* the pre-sympathetic hesitation that we refer to as "deer in the headlights."

A deer in the headlights is surprised and has not yet activated the appropriate behaviors for making a dash for safety – but he certainly isn't almost dead.

A person on pause, in preparation for possible death, is switching over into suspended animation, feeling no pain, and most likely perceiving himself from outside his body. Observing oneself as if outside the body is referred to in western medicine as dissociation.

Dissociation

Unfortunately, the term "dissociation" has been adopted by several fields of scientific inquiry, each with a very different meaning of the word.

In the field of medical *biology*, the word dissociation refers to perceiving oneself from outside the physical body, a change in perception that often occurs during pause.

In the field of *psychology*, the term refers to a separation or compartmentalization of some mental data away from normal consciousness. You might describe this type of dissociation as mentally blocking awareness of some event or body part to the point where the person has no memory or a distorted memory of the event or body part. This is the most common meaning of the word dissociation for today's general public.

While on pause, a person might experience *biological* dissociation. He might *observe* his own body from outside of it, while having a diminished level, or even none, of the usual physical sensations of being inside his own body. He might lose both somatic

awareness (*how* the body feels inside) and loss of proprioception (awareness of *where* his body parts are if he can't see them).

While the idea of a person perceiving himself as if outside his body might stretch credulity for some readers, *many* people who have had a concussion or severe, life-threatening injury describe the strange experience of a shift in self-awareness, so that they are somewhat numbly *looking* at themselves from outside of their body instead of *feeling* as if they are inside their body. The *skin* isn't necessarily numb. The internal, *somatic* self-awareness feelings might be missing. [1]

Anesthesia

Full medical anesthesia puts a person into a high degree of pause mode. It does *not* put a person to *sleep*, despite the popular euphemism. The phenomenon of "watching my surgery from the ceiling" is *not* unusual during lengthy surgeries, and is even somewhat *common* in heart-related surgeries. [2]

Shifts in channel qi

During pause, very specific shifts in channel qi flow kick in. The following is a brief description.

Changes in the Stomach channel schematics

In pause mode, the channel qi in the Stomach channels runs backwards from St-42, on the foot, to, depending on severity, the leg, torso, or even the head. (See illustrations on pages 4 and 5xxx.)

[This backwards flow of the channel qi can be felt by hand. The art of feeling channel qi is taught in chapter one of *Tracking the Dragon*.]

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[1] In my earliest writing on this subject, I decided to refer to some strange aspects of the Parkinson's personality, "aspects" that I now know to be characteristics of pause mode, as "dissociation" because so many of my patients with Parkinson's perceived themselves as if outside of their bodies. Using the word dissociation was a bad choice. The many meanings for the word dissociation led to lots of confusion.

I have since changed the name of this mode to pause, but some of those early editions that refer to a "dissociated mode" are still out there on pirated web pages, keeping the confusion alive.

I am constantly writing up-dates. Please, do not try to be helpful by posting my quickly out-dated writings on your website. Instead, if you want to share this material, please consider making a link to the Parkinson's Recovery Project website so that your readers can always get the most up-to-date information. Thank you.

[2] While *western* medicine has no explanation for the normal outside-the-body dissociation that is a common feature of being on pause, it is simply the re-centering of one's Source waves, one's Yuan Qi, to a location outside the body.

Normally, electromagnetic waves created by this energy, a type of light-wave based energy associated in ancient Asian scriptures with the immortal part of one's soul, is centered in the pericardium, the conductive tissue around the heart, but it can actually be centered anywhere. This aspect leaves the body after death. During pause, it may relocate to outside the body but usually stays in the vicinity.

When you feel the Stomach channel qi of a person on pause, you might detect a sensation of it flowing *up* the leg (flowing upstream), or making a "standing wave" (rapidly vibrating back and forth), or even seeming to disappear.

If backwards-flowing Stomach channel qi makes it all the way up to the jaw, it is shunted from ST-6 to ST-8, on the forehead. From ST-8, it can flow into the Gallbladder channel at adjacent GB-14, on the forehead, and thence into GB-7.

When on pause, Stomach channel qi does *not* flow from ST-42, on the top of the mid-foot, down to the toes (the parasympathetic pattern) *nor* does it flow from ST-42 over to SP-3 (the sympathetic pattern).

On pause, in addition to flowing backwards from ST-42 towards the ankle and on up the leg or higher, some channel qi in this area might seem to vibrate back and forth between the top of the foot, at ST-42, and the bottom of the foot, at KI-1.

Stomach channel qi can flow backwards in response to situations other than pause. A severe blockage at any point along the path of the Stomach channel might cause the Stomach channel qi to run backwards from that point, also variously known as running Rebelliously, Retrograde, or Counterflow.

A water analogy

Electricity and water both always flow in the path of least resistance. If a blockage inhibits the flow of electrons or of water, the path of the electrons or water will either back up or get deflected in some other direction.

In clinic, I have seen this backwards Stomach channel pattern set in motion by C-section scars and by childhood foot injuries from which the bones were still displaced, to name just a couple of *non*-pause examples.

Any channel will flow backwards when doing so is the path of least resistance.

Changes in the Du channel schematics on pause

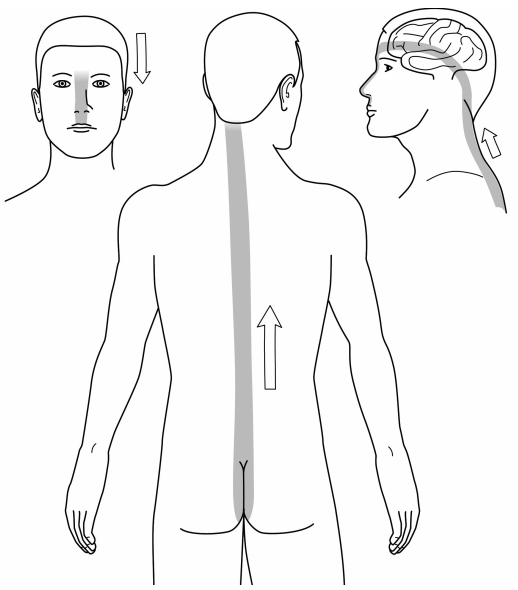


Fig. 8.1 The path of the Du channel when awake

[The Du channel begins at the anus. Du channel qi flows up the back, just over the spine. At the neck, it flows into the head and through the midbrain. After emerging at the forehead, it flows down to the upper lip. It then flows into the mouth, down through the gut, and out the anus.

From the anus, it again flows up the back, keeping a continuous loop going.

During sleep, the through-the-brain portion of the channel is greatly diminished. While sleeping, most of the already diminished Du channel qi is redirected and runs over the top of the head. This shift greatly diminishes activity in the midbrain regions and allows for the drop off in consciousness that accompanies sleep.]

When on pause, the torso portion of the Du channel qi becomes a standing wave. It stays between the sacrum and the base of the neck.

During pause, the Du channel does *not* noticeably flow up the neck and into the head in the parasympathetic pattern *nor* does it flow over the top of the head in the sleep pattern.

When feeling the Du channel qi of a person on pause, The Du channel might feel as if it stops at the base of the neck, stopping somewhere lower down the spine, or missing altogether. The Du channel might also feel as if the channel qi is making tiny back-and-forth, up-and-down movements, rather than flowing steadily in an upward direction.

As a gross generalization, the *midbrain* areas regulate the brain functions that are dominant during parasympathetic mode, and the bilateral brain areas (left and right sides) regulate brain functions that are ego-driven (sympathetic) or sleep-based. Sleep behaviors can include consolidation of memories – side-of-the-brain processes. Ego-based behaviors are more active during sympathetic mode and pause mode.

When a person is in a high degree of parasympathetic mode, he is predominantly oriented towards enjoying his sense of resonance with others or with nature. His Du channel runs at full strength through the midline of the brain.

In pause, when most of the Du channel qi stops at the base of the neck, the brain is primarily served by channels on the sides of the head, especially the Gallbladder channel.

The side-of-the-head channels influence activities in the brain areas that regulate speech, risk assessment, fear, and rage, and that originate command-based motor function, to name just a few.

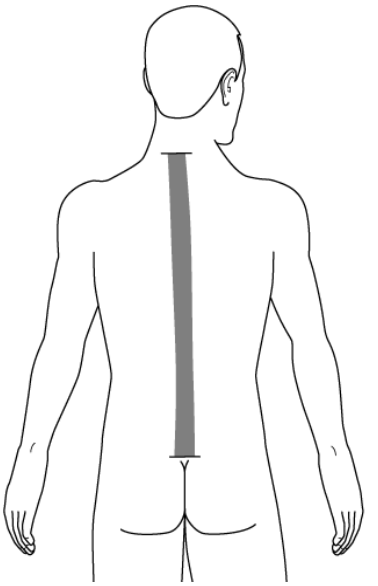


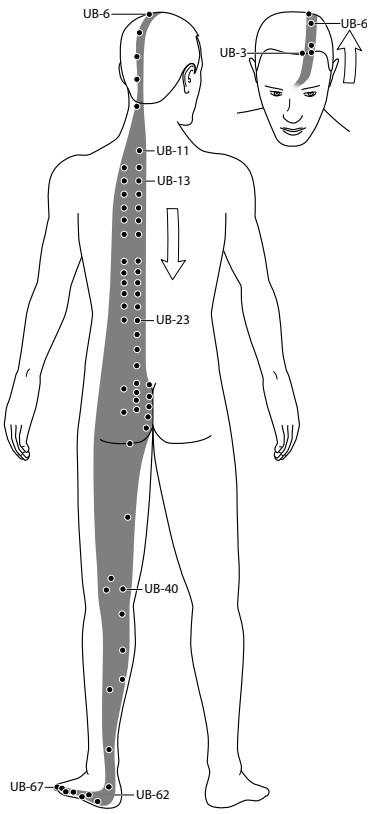
Fig. 8.2 The path of the Du channel while on pause

When on pause, with the brain portion of the Du channel somewhat inhibited, the energy level in the midbrain – where the striatum and thalamus are located – is correspondingly inhibited.

Instead, when a person is on pause, his brain functions are predominantly oriented towards self-preservation: side-of-the-brain behaviors.

The Urinary Bladder (UB) channel on pause

The UB channel normally runs from the eyebrow, up over the head (directly over the amygdala), down the neck, down the sides of the spine (directly over the connections where the spinal nerves meet the spine), and all the way down to the littlest toe.



During pause, the same electrical obstruction that causes the Du channel to stop at the base of the neck causes the UB channel to shunt off into space at the base of the neck, just above UB-11, flowing out into the air. You can feel this electrical "divergence" with your hand.

This UB channel change causes two shifts:

1) an *increase* in the speed (voltage differential) of the UB channel qi that flows through the head and over the amygdala – the fear and rage center of the brain.

This increases the degree of terror or rage that a person feels.

2) a *decrease* in the normal stimulation of the spinal nerves. The spinal nerves ordinarily stimulate the *sympathetic* nerve responses in all the torso organs. (The parasympathetic nerve responses are regulated by the vagus nerve, a cranial nerve that emerges from the neck, up near the skull.)

The decrease in activation of the spinal nerves inhibits the sympathetic nerve impulses that go to the internal organs *and* inhibits pain signals that are heading towards the brain from the torso and limbs.

Fig. 8.3 The path of the UB channel in parasympathetic mode

The absence of UB channel qi flowing over the roots of the spinal nerves emerging from the spinal vertebrae also turns *on* the release of endorphins at these locations. Endorphins are neurotransmitters that inhibit transmission of pain signals.

This particular shift in channel qi flow, the flow of the UB channel into space from just above UB-11 – instead of staying just under the skin where it usually flows – may be related to the feeling of "being outside of the body" that is a common feature of being on pause.

This might also be the reason that ancient Chinese tradition holds that the UB channel is the "closest channel to the exterior," meaning "closest to the air and/or surface of the skin," even though most of the other channels are equally close to the surface of the skin in terms of physical distance.

This particular shift is also one of the places that people are likely to get "stuck" while coming out of pause. The rest of the body's schematics might shift back into the usual blend of parasympathetic and sympathetic electrical flows, but it is not unusual for the UB channel to continue flowing into the nearby air at the base of the neck. This pattern can cause a person to stay mentally agitated and feel somewhat "out of his body" even though most of his physiology has reverted back to a healthier set of behaviors.

In recovering from Parkinson's, some people must work at visualizing this current returning to its correct location – staying *under* the skin and flowing down the torso – in order to turn off a continuing elevated amygdala response and help turn off the tremor.

Until this UB channel returns to *under* the skin, it can be very hard, if not impossible, for the person to perform the "shimmy" that travels down the spine when turning off pause. It is possible that the actual cause of the shimmy that people experience when coming out of pause or sometimes coming out of a high degree of sympathetic mode is a physical response to the return of the UB channel qi coming back into the body.

In most cases, if a person cannot bring himself to let a shudder or shimmy travel down his spine, it may well be because the UB channel is still flowing out into space. Simple visualization, together with the other steps for turning off pause, will eventually retrain this channel back into its correct path.

Changes in the Ren channel schematics

[When a person is calm and contented, the Ren channel qi flows from the anus to the front of the torso, up the midline of the torso, and up to the lower lip.

From the lower lip, the Ren channel qi flows into the mouth, down through the gut, and out the anus. At the anus, it flows up the front of the torso again.

Both the Du and Ren channels begin at the anus, flow up the back and front of the body, respectively, flow into the mouth, down through the gut, and out the anus. These huge, powerful currents are present in the embryo before the twelve primary channels begin to take shape. In the embryo, the Du and Ren channels pave the way for the digestive tract. The Du channel also provides the energy for the area that will become the brain.

The Du and Ren channels drive all the other channels, as well as providing much of the energy that moves the digestive tract.

Together, the Du and Ren channels are the strongest (largest amperage) channels on the body. All of the primary channels (bilateral channels named in honor of organs) are derived from the energy that flows in the Du and Ren channels.

"Du" means "governor," and refers to the subordinate relationship that the brain and sympathetic mode should have with the heart – "the King" – and with parasympathetic mode.

"Ren" means "humanity."

The Ren channel qi provides back-up support for any channel that is needing a boost. For example, during sympathetic mode, some amount of Ren channel qi in the vicinity of the chest is diverted deep inside to the heart, to help with the need for increased heart rate.]

During pause, when blood is diverted deeply inside to possibly prevent further loss of blood, the Ren channel qi also diverts deeply inside the body, towards the spine.

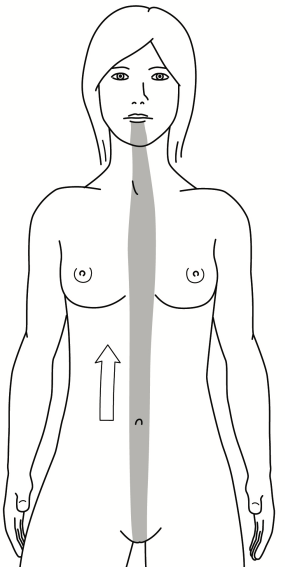


Fig. 8.4 The Ren channel in parasympathetic mode

When a person is on pause, the Ren's channel qi leaves the skin level and dives *deep* within, becoming undetectable by hand. The location from which it dives deep is variable, ranging from just below the pubic bone to the bottom of the sternum. [The sternum is the "chest" bone. It connects to the front ends of the upper ribs.] On pause, the Ren channel qi only rarely gets as far up the torso as the mid-sternum.

The alteration in flow of both the Du and the Ren during pause greatly diminishes the amount of channel qi flowing into the mouth and gut. When a person is on pause, his appetite and digestion is severely inhibited. Under these conditions, constipation is normal. Sometimes, in the absence of adequate energy flowing down through the digestive tract and in combination with the *backwards* flow of Stomach channel qi, the digestive tract might even move backwards, causing low level nausea or even vomiting.

The other channels

The alterations in the Stomach, Du, and Ren channels *might* lead to further changes in channel qi flow. The possibilities include backwards or inhibited flow in the Large Intestine channel and/or diminished power or other altered behavior in the Heart, Pericardium, Urinary Bladder, Gallbladder, Liver and Triple Burner channels.

Confused qi

In translations from classic Chinese medicine, the pause-related shifts in channel qi schematics are customarily referred to in English translations as "Confused [channel] Qi."

There is *nothing* "confused" about these shifts. The axiom we are taught, "Terror Confuses the Qi," is a poorly expressed reference to the *logical* and even *life-saving* channel qi changes that occur during pause.

Pause does *not* trigger an *incorrect* flow of channel qi, as the word Confused implies. The changes of pause are *healthy* and *correct* responses to severe, life-threatening damage. Pause can be a life-saving mechanism.

However, getting channel qi *stuck* in some pause-related pattern after the crisis has ended might sooner or later lead to serious health problems down the road.

The joy of inner stillness is not the same as pause

Many people have asked me if pause mode is the same as the deep stillness and joy of meditation. No.

The stillness and joy attained by long practice of single-focus meditation during which breathing and heart function might become slower or greatly diminished is *not* pause. Just the opposite: this type of joy is a manifestation of pure parasympathetic mode: "In tune with the Divine"; resonant with Love.

Holding the body motionless while practicing single-focus types of meditation trains a person to bring all the life force energy deep within the spine and from there to specific locations such as the heart or the point between the eyebrows. In response, *physiological* functions might diminish or even cease. But superconsciousness, awareness of the heart's feelings of joyful expansion, and awareness of subtle, light-based energy in the body are easier to maintain or can even increase.

In certain types of single-focus meditation, a person might be able to straighten out the brain portion of his Du channel: keeping the Du channel energy in the thin line right up the center of the spine and through the center of the brain, as opposed to it constantly zigzagging to the left and right – especially in the sacrum and head.

With this narrow "antenna" of current flowing in a straight and narrow path up the spine and through the brain to the forehead at the location of the "third eye," sometimes called the single eye, one can literally tune in with the energy and joy of one's own soul. As Jesus is quoted as saying, "Straight is the gate, and narrow is the way." [1]

The delusion of being *defined* by the body, a delusion sustained by activities in the right and left sides of the brain, is thus destroyed. Tuning in, radio-like, with the soul and/or the frequencies or wave vibrations of Universal love via this spinal antenna is then possible.

This is *very* different, almost the opposite, from the *ego-identified* sense of self that *usually* perceives itself as distinctly *apart* from everything else in the universe. During pause, a person might feel as if his consciousness has been expelled from his physical body and that he is teetering on the verge of dreaded and permanent annihilation.

While on pause, it is *extremely* difficult to sense the electromagnetic wave variations and amplifications in the pericardium [the connective tissue around the heart], sometimes called "heart-feeling," that are associated with joy. On pause, awareness of heart-feeling sensations are *greatly* inhibited: the opposite of joy.

On pause, a person or animal is struggling to stay alive so that he can get back inside his precious, unique body. If he is conscious, the side structures in his brain, especially the risk-assessment area, are *extremely* active. Assessment of the situation's risk is elevated until the risk level comes down, physical stability is attained, and pause is turned off – or until the person or animal dies.

Oppositely, in a high degree of parasympathetic mode, a person realizes that his real nature is waves of loving, consciousness-bearing vibrations...not a physical, ever-changing body. In this state, distracting neural activity on the *sides* of the brain is deeply stilled.

Research using brain scans of highly experienced meditators, including some Hindu and Buddhist monks, shows them having this type of narrowed, highly focused brain behavior. A person in this nearly pure parasympathetic condition can know his body as a temporary residence in which and from which he might lovingly experience various sensory events without having ego-based attachment to his observations.

Oppositely, a person on pause, with his Du channel stopped at the base of his neck, has a greatly diminished amount of current flowing through his midbrain and a greatly diminished ability to resonate with joy. Instead, he might have an elevated level of ego-driven desire to protect and preserve his precious body. (The word "ego" is used here to mean "sense of identity that imagines oneself to be apart from the rest of the universe.")

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[1] Matthew 7:14. Another scriptural quotation states, "When thine eye is single (all the attention is focused at the third eye), thy whole body will be full of light": attuned to the wave frequencies of light that create the illusion of the solid body via rivers and glaciers of electrons and protons.

The almost dead mouse

Many of us have seen an animal in pause mode. A very common example of pause occurs when a cat catches a mouse. As the claws of the cat sink into the mouse (excessive perforation of the skin), the mouse immediately goes into pause.

The mouse becomes rigid. His body starts to curl up into a fetal position. His skin becomes cold. Breathing is minimal and nearly silent. He might appear to be dead.

If the cat was hunting merely to amuse himself, and not to satisfy hunger, the cat might biff the now-rigid mouse around a few times, seeking a response. If the mouse remains cold and rigid, the cat will soon lose interest, and go off in search of livelier sport.

After a few minutes, when the mouse is no longer bleeding at the clawed sites and its blood pressure has stabilized, the mouse's sacrum and brain will begin producing a small electrical signal that causes a sense of internal tremor. This tremor, in turn, stimulates the risk assessment area of the mouse's brain to shift into a higher gear.

When the mouse no longer sees the cat, hears the cat, smells the cat, or feels the cat, allowing his risk assessment area to determine that he is now safe, he will take a deep breath, wobble his head slightly to turn back on his vagus nerve at the base of his skull, allow a visible shiver to run down his spine activating his spinal nerves, and then he'll scamper off to safety.

The mouse was not "playing dead." A mouse does not have the intellectual capacity or self-control to play dead. The mouse entered into an involuntary condition of pause brought about by perforation of his skin by the cat's claws. This collapse into the inert immobility of a high degree of pause renders the mouse cold, rigid and corpse-like, and may well save the life of the mouse.

Many, many people have seen this "dead mouse" behavior. Sometimes, people imagine the mouse is just "playing possum," pretending to be dead.[1]

Of course, this type of *full-blown* pause mode, a condition in which a person or animal is motionless, is not usually seen in an acupuncture clinic. However, patients with a modified degree of pause or a situation in which some *portion* of channel qi has become stuck in the channel qi schematics of pause might very well come to your office. Long-term use of even a portion of the pause-type channel qi pattern can cause significant health problems, often causing syndromes of "no known cause," or "incurable" syndromes and health problems.

Getting back to the changes in the Stomach channel's basic path

Perhaps the most important Stomach channel shift during pause is the shunt to St-8, on the forehead (Fig. 8.5). As mentioned earlier, when Stomach channel qi runs backwards, it is prevented from going backwards through Yin Tang and *backwards* into

[1] A possum's pause response to being startled is not a trick. A possum has a hair-trigger, *involuntary*, full-body pause response when startled. The possum isn't making a decision: he is hard-wired to go into pause in response to just about any startle event.

"Penguins," the 2019 Disney full-length nature movie, has footage in which an Adèlie penguin goes into pause after being attacked by a seal – and survives when the seal loses interest.

Again, this is very different from the frozen "deer in the headlights" phenomenon, which is a *pre*-action part of the sympathetic mode response.

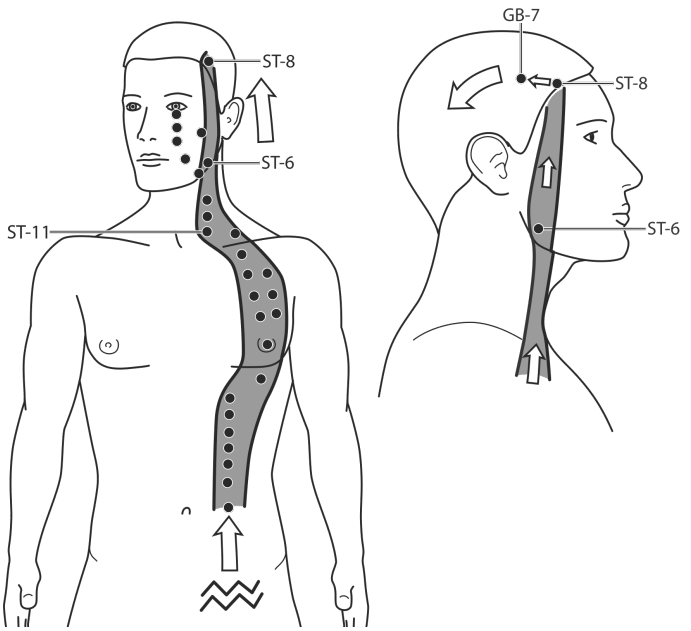
the brain – a routing that might prove deadly. St-8 is *not* on the Primary route of the Stomach channel. Stomach channel qi is *only* routed through this point when the Stomach channel is running *backwards* up as far as the head.

When Stomach channel qi is flowing *backwards*, when it gets to St-6, at the back of the jaw, it is shunted up to St-8, on the forehead. There, it either builds up pressure, causing a headache, or it short circuits into the nearby Gallbladder channel.

If the backwards flow is mild, qi might merely accumulate at St-6 or St-8. Tooth or jaw pain at St-6 or a pressure-type headache at St-8 might develop, brought on by the localized channel qi build-up.

The electrical system at St-8 behaves somewhat like an electrical capacitor: a place where charge can build up. If channel qi build ups at St-8, it *might* cause a headache. But if *enough* channel qi builds up at St-8, the channel qi might surge out of St-8 and short circuit into the nearby Gallbladder channel, causing an increase in amperage of Gallbladder channel qi. The person, if awake, might experience a sensation as if the inside of his head is spinning, or as if he has temporarily experienced an altered consciousness or even a momentary loss of consciousness.

If the backwards flow becomes *chronic*, a new, *constantly open* path into the GB channel might develop.



In this diagram, Stomach channel qi is flowing backwards due to a blockage. The blockage is shown by zigzag lines on the lower torso. This location was selected at random. The blockage might just as easily have been drawn on the neck part of the Stomach channel or on the foot part of the Stomach channel.

Although the Gallbladder channel is not shown, a large arrow on the side of the head demonstrates the direction of flow in the head portion of the Gallbladder channel qi.

The addition of Stomach channel qi (the small arrow) to the channel qi already flowing in the Gallbladder channel increases the overall amount of channel qi in the Gallbladder channel path.

Fig. 8.5 St-6 to St-8, the safety shunt for backwards *flowing Stomach channel qi.*

[As an aside, sleep mode is activated by a night-time circadian surge of energy in the Gallbladder channel. When a surge of increased current runs through the head portion of the Gallbladder channel, which runs from the front of the head to the back, there will be

a corresponding *decrease* in the amount of channel qi running in the opposite direction: a decrease in amperage in the head part of the Du channel.

To learn more about the electrical schematics of sleep mode, please read chapter 13 in *Tracking the Dragon*.]

During pause, backwards flow shunting from St-8 might flow into the Gallbladder channel, thus *increasing* the power in the GB channel. As the power in the GB channel *increases*, the power in the Du channel qi *decreases*. If the Du decreases in power enough, sleep or even coma might ensue.

It is easy for the backwards Stomach channel qi to flow into the Gallbladder channel: the Gallbladder channel is very wide across the forehead, and offers little resistance.

If traumatic, life-threatening damage occurs and pause mode kicks in, the sudden surge of backwards-flowing Stomach channel qi might activate the St-6 shunt (on the back of the jaw) and send a *surge* of channel qi up to St-8. This might then send a surge into the Gallbladder channel. This surging *increase* in the amount of Gallbladder channel qi in the head might *greatly* decrease the amount of Du channel qi in the head – so much so that the person or animal might pass out and stay passed out until the body stabilizes and pause turns off.

Then again, sometimes the amount of backwards Stomach channel qi flowing over into the GB channel is small enough that the traumatized person might merely feel woozy or go in and out of alertness.

As soon as pause mode turns off or the Stomach channel *blockage* is broken up, the Stomach channel qi will *usually* resume flowing in the correct direction automatically: any previous build-up of charge at St-8 will disperse by flowing back down to St-6, and thence to the foot.

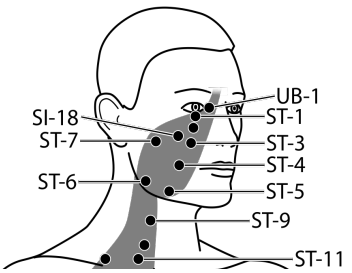


Fig. 8. Head portion of the Stomach channel's parasympathetic path

Turning off pause

Turning off pause requires going through a very specific set of feelings and movements. These are not *attitudinal* thought processes, but very specific sensory feelings and observations that *start* with the resonance that occurs when physiological trauma ceases and the body's crucial life systems such as blood pressure regulation and heart rate are stabilized.

The next biological step is sensory (eyes, ears, nose, touch) confirmation that the immediate danger and risk of imminent death has now passed.

After confirmation, the body should then automatically takes a deep, audible inhalation. The vagus and spinal nerves that have been turned down or off are physically turned back on with specific neck and spinal movements.

But, as with any channel qi variation, it's possible for the channel qi flow to get *stuck* in all or some of the schematics of pause.

Self-induced pause

The channel schematics of pause can also be set in motion in response to a person's forceful command to himself to "Feel no pain." Very often, this command refers to emotional pain as well as physical pain.

The body does have a mode in which pain is minimized: pause mode. Many of my patients who've been stuck in pause mode schematics got that way by telling themselves to "don't show pain," "don't feel pain," or something along the lines of "I don't want to be a part of this world."

The usual steps for turning off pause will not work for this type of pause. Instead, the numbness to somatic *feeling* that was triggered by the mental instruction must be ended. A person doesn't need to remember the specifics of the instruction, but he must learn how to create the feeling of biological stability and safety. Only then can he go on to turn off the neurological behaviors that he unwittingly set in motion with his self-instruction.

Pseudo pause: the slow accumulation of pause-like channel qi flow

Pause-type schematics might develop slowly, over years, in response to an unhealed injury that, at first, only partially blocks a channel. In my limited experience, it's usually been an injury along the Stomach channel. This is not true pause: the patient hasn't lapsed into a state of decreased mobility or lowered blood pressure – at least not at first.

Rather, this unhealed injury creates a condition in which channel qi behaviors might eventually *resemble* those of pause. In the case of a displaced bone or soft tissue in the foot, the pause-type channel qi behaviors might come on gradually.

Then again, in the case of a broken femur (upper leg bone), the Stomach channel might become immediately blocked: pause-type channel qi flow might begin almost immediately.

In the early years of, for example, a dissociated and therefore somewhat painless unhealed foot injury featuring slightly displaced soft tissue and bone(s), the Stomach channel qi might be only partially blocked on the foot. Due to increased resistance in the channel qi on the foot, *some* amount of the channel qi might back up or diverge on the side of the leg, upstream from the blockage, maybe going sideways from the middle of the lower leg over into the nearby Gallbladder channel at GB-35.

This not unusual divergence might cause a weird, unsettled feeling or maybe tension in the side of the leg, but there will be no backwards-flowing Stomach channel qi getting up to the forehead, and no significant symptoms of pause...yet.

If the electrical confusion at the site of the blockage snowballs in size over time, the backwards Stomach channel qi might back up a little higher up the leg or even into the torso, where it might then flow sideways into any nearby channel. If it flows backwards as

high up as the neck, it might intersect with the Large Intestine channel qi and cause the Large Intestine channel qi to start flowing backwards.

After another decade or so, as the chaotic channel qi in the vicinity of the injury continues to spread out, at some point almost *no* Stomach channel qi can get past the injury site. With almost no channel qi in, and diminishing somatic awareness of, the area, fungus might start to grow on the second and third toenails. The toes might become easily cold or even numb.

Meanwhile, the force of the backwards-flowing channel qi might gradually become severe enough that Stomach channel qi flows backward all the way to St-6 on the back corner of the jaw and thence to St-8, on the forehead.

When the build-up at St-8 is large enough, the channel qi will shunt into and augment the Gallbladder channel, which flows from the front of the head to the back. This contributes to inhibition of the Du channel, which flows from the back of the head to the front. This diminishing of the Du channel leads to diminishing of or inhibition of certain midbrain behaviors such as dopamine release for motor function.

When aberrations in the flow of channel qi remain uncorrected for years, all bets are off with regard to how the channel qi flow might change, over what time frame, and what health problems might arise.

To turn off this type of injury-based pause-type channel qi flow, the injury must begin to heal.

Yin Tui Na is the most effective way that I have found for treating a long-ignored or forgotten, painless, or unhealed injury.

Whether from pseudo-pause due to a long-unhealed injury or pause-like channel qi flow stuck in parts of the body from an old, pause-inducing trauma – one that the patient had either forgotten or had assumed was long over – pause-type channel qi behavior in other channels can become very unpredictable if the electrical system becomes *stuck* in the electrical patterns of pause. Once electrical currents start flowing in an unhealthy direction for the long term or become unable to perform a healthy self-correction, the only certainty is that the channel qi will always flow in the path of least resistance.[1]

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[1] Here's a plumbing analogy: blocked plumbing in a house causes the wastewater to flow somewhat unpredictably as it follows the path of least resistance through the pipes that are hidden in the walls.

Sometimes a mild blockage will cause bubbling noises in the toilet and water moving transversely into other lines until it slowly drains, but a severe blockage beneath the house might result in raw sewage from upstairs spewing up from the downstairs shower drains. When plumbing flows backwards, it can *seem* unpredictable as to direction because you can't see what's happening inside the walls. But rest assured, the sewage will *always* flow in the path of least resistance.

Water is impelled by gravity.

Channel Qi is impelled by voltage differentials (analogous to water's relationship with gravity) and conductivity. These factors can be influenced by physical damage and scar tissues, as well as the distinct brain signals associated with each of the four neurological modes. Other influences on channel qi flow include wave signals that come from thought waves, including the subconscious waves, habit, *and* the electromagnetic signals defined by cellular DNA.

But with both water and channel Qi, their movements, if blocked, can build up and flow sideways or even flow *backwards* from their usual routes.

Backwards in the head: danger!

In terms of life-saving physiology, the design for shunting Rebellious or Counterflow (backwards) channel qi up to the corner of the forehead is simple and elegant.[1]

If the Rebellious Stomach channel qi were allowed to flow backwards all the way to Yin Tang, it could then flow backwards into the brain portion of the Du channel.

Backwards flowing channel qi in the midbrain portion of the Du channel can quickly cause loss of consciousness and even death. The shunt at St-6 prevents that.

When Du channel qi sedation occurs in response to Rebellious Stomach channel qi surging into the GB channel, the tiredness, wooziness, sleep, or even coma can last for as long as the Stomach channel qi is strongly flowing backwards.

These symptoms of lassitude, ranging from relaxation to coma, can be an important factor in healing.

The physical stillness might allow the injured person to rest while the body navigates its way back to stability. In some cases, profound stillness can also serve to induce the dead mouse effect, which can sometimes save the life of an animal being attacked by a non-hungry predator.

Thanks to the St-6 shunt, the brain portion of the Du channel does not take a *direct* hit from backwards flowing Stomach channel qi. Instead, the Du is merely *influenced* by an increase in posterior-flowing Gallbladder channel qi – a much weaker force. The elegance of this Du channel protection mechanism never ceases to amaze me.

Pause-based alterations in the brain supply of channel qi

When the Du channel is blocked at the base of the neck, the other currents flowing over and through the head (Urinary Bladder and Gallbladder channels) can still provide support and direction to the currents inside the brain.

As noted earlier, when the head portion of the Du channel is greatly inhibited or blocked, as it is in pause, channel qi flowing through the midline of the brain is inhibited. This causes inhibition of some of the brain structures that straddle the midline such as the thalamus and striatum (grossly generalized here as "feel-good areas").

At the same time, the brain experiences increased activity in the sides of the brain: the locations for fear-based risk assessment, hyper-analysis (anxiety), and increased amygdala (fear-and-rage center) activity – areas located directly under the Urinary Bladder (UB) and Gallbladder (GB) channels.

It is likely that the channels that flow front-to-back over the *sides* of the brain, the UB and the GB, become more influential on thought behaviors while the Du channel is

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[1] In some British publications, *Rebellious* channel Qi is referred to as Counterflow or Retrograde channel Qi. I prefer the term Rebellious, a more appropriate translation from the Chinese. For thousands of years, the Chinese political system has considered rebellion to be *the* single greatest threat to civic peace and harmony.

Using the word Rebellious to refer to channel Qi running backwards suggests how *very* dire this condition can be. Rebellious channel Qi is not just preventing correct physiology in the vicinity of the backwards flow: it can be a deadly threat to the entire system.

blocked, stimulating the various ego-based fear centers to a higher degree, while the midbrain's dopamine-regulating (feel-good) areas are simultaneously inhibited.

In summary

During pause, many specific channel behaviors are set in motion as well as some side effect channel behaviors. While on pause, aside from the backwards flow in Stomach channel, stoppage in the Du at the base of the neck, and disappearance of the Ren, all other channel bets are off.

Making diagnosis trickier, *sometimes* the Stomach channel qi runs backwards *not* because of pause, but because of scarring or injury blocking the channel's path or a long-forgotten, dissociated (and hence unhealed) foot or ankle injury that has slowly turned into a complete blockage of the Stomach channel. A significant leg injury that didn't set pause in motion at the time might eventually result in the Stomach channel qi getting stuck partially or fully in a backwards flow pattern.

In order to diagnose pause using the channels, it's best to assess not only the left and right Stomach channels, but also the Du channel flow – especially the behavior at the base of the neck.

In addition to feeling the Du channel with your hand, you might also want to ask the patient to do some visualization of the Du channel flow. Then have him tell you what he "sees" or feels. If he says he can't visualize at all, he might be stuck on pause.

You might also want to ask the patient how he perceives sensations if he imagines himself walking down the street on a lovely day, listening to the birds and enjoying the blue sky. Does he imagine he's inside his body, feeling his sensory experiences? Or does he imagine he is outside of his body, *looking* at his own body. If he's outside his body, he may well be stuck on pause.

My book, *Stuck on Pause*, has more details on diagnosing pause and diagnosing what type of pause a person is using.

Pause mode is supposed to turn off *automatically* when a trauma comes to an end and the body negotiates its way back to stability. Sometimes it doesn't.

Becoming stuck on pause or having even a small segment of channel qi stuck in a pause pattern can lead to a variety of chronic, "incurable" health problems.

As for treatment, neither acupuncture needles nor herbs will turn off *stuck* channel qi patterns of pause. Specific steps must be taken: steps that, for whatever reason, were never taken when your patient's original trauma ended. These steps include:

1) feeling physically re-stabilized, 2) strongly confirming that one is no longer at risk of imminent death (it's often helpful to have a second party also confirm it), 3) taking an audible deep breath, 4) bobbling the head, and 5) moving as if a shimmy is traveling down the spine.

If the UB channel is still flowing out of the body into the air at the base of the neck, it will be nearly impossible for the person to perform a natural shimmy or shiver. Physical attempts at inducing a shiver might be helpful at inducing the UB channel to go back into the body. Oppositely, attempts to induce a physical shiver might make a person more determined that he cannot or should not allow himself to make this movement.

If this channel behavior has become "stuck," information in chapter 6 of the book Yin Tui Na on destroying a lodged brain habit might be useful.

If the person has *self-induced* pause, he must go through a very *different* set of specific steps that will allow him to turn off the command he gave himself and which he may well have long-forgotten: a command such as "Feel no pain" or "Rise above the pain."

In cases of self-induced pause, so long as the thalamus and striatum are inhibited due to the brain's channel qi flowing in the pause pattern, self-induced pause mode will continue to be active no matter what physical or *superficial* mental steps the doctor or the patient might take.

The patient must practice and master techniques that can activate the striatum and thalamus in spite of his instruction. Only then will he be able to turn off pause. He will very likely also need to mentally restore the UB channel to its flow pattern down the neck and into the torso.