

“Two wrongs don’t make a right.”

-An old saw

CHAPTER NINE

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

TREMOR

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

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

Brain hemisphere displacement: driving the internal tremor

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Atrophy: determining the location of early stage tremor

Atrophy on the Large Intestine channel

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

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

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

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

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

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

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

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

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

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

Variations on resting tremor

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

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

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

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

Amplified tremor

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

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

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

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

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

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

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

Amplified tremor while eating

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

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

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

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

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

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

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

LOSS OF COORDINATION

Left-Right integration

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

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

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

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

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

Balance

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

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

Festinating gait

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

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

Stuck foot

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

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

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

The Parkinson's gait

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

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

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

BILATERAL SYMPTOMS

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

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

A _____
B _____
C _____

B _____
A _____

Fig. 5.7
Parallel currents

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

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

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

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

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

HYPOTHESES SUMMARY

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

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

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

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

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

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

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

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

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

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

