

“Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forward; and to pass from a walking to a running pace: the senses and intellects being uninjured.”

– the opening definition from *An Essay on the Shaking Palsy*, by James Parkinson, member of the Royal College of Surgeons, 1817.

CHAPTER FOURTEEN

THE WESTERN UNDERSTANDING OF PARKINSON'S DISEASE

This chapter will explain idiopathic Parkinson's from the current western perspective: symptom descriptions, diagnosing, current treatments, and where the western research might be heading.

It may become very important for a PDer who starts recovering to have the clearest possible understanding of the classic symptoms of Parkinson's. Only then can he tell the difference between Parkinson's symptoms and counterintuitive, often unpleasant symptoms that can occur during recovery.

Therefore, this chapter will start with a quick overview of the syndrome and then follow up with more detailed descriptions of PD symptoms. These descriptions are based on lectures, articles, various medical journals, Medscape's neurology weekly reports, books, the Internet and that medical standard, the Merck Manual. After that comes a section about diagnosing Parkinson's, then one on the current western treatments for Parkinson's disease: antiparkinson's drugs; brain implants; and stem cell research.

Please note – the symptoms listed throughout this chapter are those of unmedicated Parkinson's disease. Those patients who are taking antiparkinson's medication may exhibit a wide range of symptoms including ticcing, spasming, personality or mood change, and even psychotic behavior, all of which may be referred to by the patient – and even the uninformed doctor – as Parkinson's symptoms, but which are, in fact, short- and long-term effects of the medication.

OVERVIEW

In 1998, when I started my investigation, the following were accepted explanations of Parkinson's disease:

“Parkinson's disease (PD) is named for an Englishman, James Parkinson, who first wrote up a description of the syndrome. Parkinson's is the second most common neurological disorder in the world after Alzheimer's.”¹

¹ James Tetrud, MD, director of the Parkinson's Institute of Sunnyvale, California. From a lecture at the Santa Cruz Parkinson's Support Group, March 1998.

From the Merck Manual, I read the following: “[Parkinson’s disease] is an idiopathic (cause: unknown), slowly progressive, degenerative central nervous system disorder with four cardinal features: resting tremor, slowness and poverty of movement, muscular rigidity and postural instability. Although symptoms initially develop on one side of the body, they eventually become bilateral. In the advanced stage, the patient can suffer complete rigidity and immobility. Dementia occurs in about 50% of patients.¹ Depression is also common.

“The disease is characterized by loss of the pigmented neurons involved in controlling movement, which are located in the substantia nigra, locus ceruleus and other brainstem dopaminergic cell groups. This loss of neurons results in depletion of the neurotransmitter dopamine. The cause of the neuron loss is unknown, but it now appears not to be genetic, but rather induced through an as yet unknown external factor.”²

¹ The numbers on dementia in this reference are misleading. Dementia most often occurs in patients who are diagnosed with Parkinson’s late in life. The dementia usually begins about ten years after diagnosis. In other words, the dementia of Parkinson’s may well be the same, and seen in the same frequency, as the dementia of very old age. Part of the misunderstanding about the frequency of dementia, a symptom that strikes fear into the hearts of the recently diagnosed, is due to the way that all psychoses are sometimes lumped together under the heading “dementia.”

As noted in the *Parkinson Report*, Fall 2000, in an article “Hallucination and Psychosis in Parkinson’s disease,” Goetz, MD, “Although there were rare reports of hallucinations and delusions in medication-free Parkinson’s disease (PD) patients prior to the advent of effective drug therapy, these cases are exceptionally rare.” He goes on to point out that these problems arise in PDers in response to dopaminergic therapy (dopamine-enhancing drugs). He adds that, in some cases, psychosis can occur from complications of infection, dehydration, or drug toxicity. In other words, the psychoses are not due to Parkinson’s disease, per se.

For specific numbers on dementia, I quote from *Parkinson’s Disease, Questions and Answers*, by Hauser and Zesiewicz, Merit Publishing, Florida, 2000, p. 29: “Reported prevalence rates [of dementia] range from 10% to 80%, but actual rates are probably closer to 15% to 30%. In a series of 155 PD patients, 8% had severe dementia... In one sample of 139 PD patients in Norway, at least one “psychiatric” symptom was reported in 61% of patients. The most common psychiatric manifestations are depression (38%) and hallucinations (27%).”

From the above, the reader can see that dementia is often lumped in with other symptoms, many of which are drug related. This suggests that the true numbers for dementia are much, much lower than the 50% mentioned in the Merck Manual. James Parkinson, who admittedly worked with a much smaller sample size, was careful to make the point that “the senses and intellect remain uninjured.” I personally remember reading in the 1960s, before dopamine-enhancing medications were the norm, that the tragedy of Parkinson’s was having a completely alert mind trapped in an inert body.

² Berkow R (ed): *The Merck Manual*, Merck Sharp & Dohme Research Laboratories. Rahway NJ, 1992, pp. 495-500.

The illness is not genetic, except in the very rare familial form of a Parkinson’s-like disorder in the Contursi family. As for research looking for a genetic connection in Parkinson’s, a large, nationwide identical twin study of PD patients in the late 1990s came to the rare conclusion that if one identical twin has PD, the other is less likely than the national average to have PD. This indicates that PD is not genetic. More interestingly, it suggests some protective benefit from having a twin.

Considering that PD is often seen to run in families, we can make this hypothesis: the attitudes of “stiff upper lip” and stoicism that can be passed along from one generation to the next as family values or behaviors can contribute to the occurrence of Parkinson’s disease.

However, in an identical twin situation, one twin is usually more dominant, more protective of the other. In such a situation, even if the family tendency is towards a cold disdain of showing pain or emotion, the intimate relationship of the identicals may provide a safe haven, at least for the subordinate twin, from the emotionally rigid behaviors of the rest of the family. If the young subordinate identical is injured, he may hide it from the rest of his family, but he may avail himself of the sympathetic ear, maybe even a hug, from his closest sibling. The dominant identical may provide succor for his womb-mate in a family dynamic that otherwise would be hostile to the expressing of emotions. (Continued on next page.)

Signs and Symptoms

“A patient with PD may present with from three to all four of the variants of the symptoms below at the time of diagnosis. As symptoms progress, a patient may become wheelchair bound. PD is not fatal, but increased mortality occurs because of debility, aspiration pneumonia, and infections.”¹

The following are the four categories of Parkinson’s disease symptoms. I call them the Big Four.

1) Resting Tremor: The tremor is seen in 50% to 80%² of patients. The Parkinsonian tremor, in the early stage, is a "resting tremor," occurring when the affected body part is inactive: at rest.³

2) Poverty of Movement: “Bradykinesia [slowness and poverty of movement], akinesia [difficulty in initiating movement], and a reduction in automatic movements such as alternate arm swinging while walking”⁴ are characteristic symptoms.

3) Muscular Rigidity: Rigidity at the wrists, ankles, shoulders, and hips prevents smooth flow of movement; attempts at rotating the wrists and ankles result in jerky, "cogwheel" motions.⁵ The rigidity and slowness of movement combine to create a shuffling, labored gait.

4) Postural Instability: The increasing rigidity in the legs, loss of balance and coordination, and imbalance between left and right, combined with the postural forward stoop, lead to a tendency to fall forward.

Therefore, this genetics study, considered to be a failure in that it did not find a genetic connection, may in fact be an important lead. Certainly, the unheard of statistic that a person with an identical twin with an illness is less likely than average to have his sib’s syndrome suggests that something sociological is going on in Parkinson’s.

¹ The phrase “from three to all four” means that a person may possibly be diagnosed with PD even if all he has is one symptom from at least three of the four categories. As the disease progresses, he may have many symptoms from each category, but he must still have symptoms in three of the four categories for it to be diagnosed as Parkinson’s. The quote is from *Cecil’s Textbook of Medicine*, Wyngaard JB, Smith Jr LH, WB Saunders Co., Philadelphia, 1988, p. 2144.

² The percent of PDers that have tremor varies depending on which medical books or articles you read. There is no consensus.

³Tierney LM Jr, McPhee ST, Papadakis MA: *Current Medical Diagnosis and Treatment* (ed 35). Appleton & Lange, Stamford CT, 1996, pp 885-887.

⁴ Ibid.

⁵ Whoever named the uneven rotational movement of PDers’ wrists and ankles got his nomenclature wrong. A “cogwheel” is a smoothly-turning toothed gear. The word the writer was probably looking for was “camwheel.” A cam is a wheel that has one irregular lump on its circumference. As the turning camwheel rotates over the lump, it causes a pause and a thud in the circular motion of the wheel. This corresponds to the pause and skip that may occur when a PDer rotates the ankle or wrist.

Despite the original error, this uneven movement in PDers is now officially referred to as “cogwheeling.”

The above was the essence of understanding Parkinson's disease when I started the Little Project in 1998, and still is. What I call "The Big Four," the four categories of symptoms above, were derived from the work of James Parkinson and are still used today to determine a diagnosis of PD.

However, there are more details about symptoms that can help flesh out the description. These details are usually listed as being "in addition to" the Big Four, but most western-recognized symptoms are, in fact, derivatives of the Big Four.

Example of a Big Four derivative

For example, I found the following in a book on PD, after it listed the Big Four: "Another symptom of PD can be 'foot drop'." This symptom is actually not a separate symptom, but a derivative of poverty of movement. However, because the generalized Big Four does not always include many details, sometimes lists of details are included in books about PD. Often, these details about the symptoms make it appear as if these detailed symptoms are separate from the Big Four. However, no matter how many details are added in, it turns out, upon close examination, that these detailed symptoms for the most part are still derivatives of the Big Four: tremor, poverty of movement, rigidity, and poor balance.

Foot drop is an aspect of "poverty of movement"

Let's look more closely at the Parkinson's symptom called foot drop to see how it fits into the Big Four category system. During normal walking, the ball or toes of the affected foot may intermittently fail to be lifted clear of the floor during a normal stride – it can feel as if the foot sticks to the floor. The "stuck" foot causes the forward-moving patient to shuffle, or even to trip, falling face first. The falling is a symptom in the category of "postural instability," also known as "losing one's balance."

Patients may attempt to recover balance after a foot drop, but, now and then, a randomly occurring, utter inability to initiate large (normal) strides may result in a desperate attempt at walking that uses a multitude of tiny, labored, rapid steps, also known as "baby steps" or "festinating gait."

The tiny rapid shuffling steps of festinating gait are a (usually hopeless) attempt by the feet to catch up with the torso, which is still hurtling forward at the previously established speed. The torso is being propelled forward by momentum and downwards by gravity. When the slow-moving, baby-stepping feet fail to keep up with the forward moving torso, the upper body plummets to the ground or crashes into nearby objects, such as the wall. This forward and downward movement of the torso, accompanied by frantic, hurried-but-tiny footsteps that fail to catch up to the upper body, is called a festinating gait.

Though they may be listed as additional symptoms, foot drop and festinating gait are members of the Big Four of tremor, poverty of movement, rigidity, and postural instability: because of the falling and stumbling involved with the festinating gait, this problem fits into the postural instability category. Because this problem includes slowness of the footfall, inability to lift the foot off the floor (foot sticking) and slowness in making small subconscious movements that are needed to correct for imbalance, the problems of foot drop, small steps, and losing balance also fit the poverty of movement category.

This example simply shows that, although many "extra" or "additional" symptoms are sometimes listed in addition to the Big Four, these extra symptoms are actually Big Four derivatives.

The need to be informed

It is important that a person setting out to recover from Parkinson's disease has a very good understanding of what defines Parkinson's. If one understands the extent to which the recognized symptoms of Parkinson's are variations on the Big Four, one needn't memorize lists of seemingly unrelated symptoms, but need only understand the principles involved. Since every individual's Parkinson's disease manifests slightly differently, it makes more sense to understand the principles rather than a long list of symptoms which may or may not apply to any given PDer.

Recovery misunderstandings based on a TV show

As an example of how one's misunderstanding of the symptoms can lead to unnecessary worries during recovery, let me include part of a recent case study: I had one patient who, during recovery, was frightened by new sensations in her arm and the slow, rhythmic muscle extensions and flexions that spontaneously moved her bicep muscles when energy started returning to her left side. She became certain that tingling in her arms from increased sensation and improved range of movement were new symptoms of Parkinson's disease!

She was further convinced that her new ability to move easily was sign of worsening Parkinson's after seeing Michael J. Fox speaking to congress: in a televised program, Michael J. Fox stated for the cameras that his wildly flailing, dyskinetic arms (symptoms set in motion by *overmedication*) was what happens with his Parkinson's when the medications don't work. This statement may have been intentionally misleading. This was filmed during his successful attempt to show congress that more research money was needed for Parkinson's disease.¹

Misunderstandings based on observing overmedicated PDers

Others misunderstand the true nature of Parkinson's because they know someone who is medicated who "moves like a crazy person and doesn't know what he's doing half the time." Because of the rampant misunderstanding of the true nature of Parkinson's, based on uninformed people's experiences with *overmedicated* PDers, it is extremely important that I drive home the idea that Parkinson's is a syndrome marked by *decrease* in motor function: *less* movement, not more; *rigidity* over most of the neck, torso and legs, not limpness; *hesitancy and stiffness*, not rapid performance and increasing range of movement.

When the strange symptoms of recovery begin, it will be extremely important for morale that the ex-PDer has a good grasp of what constitutes symptoms of Parkinson's and what does not. Only in this way can one understand that the recovery symptoms, though annoying, sometimes painful, and even bizarre, are the exact opposite of Parkinson's symptoms. So now, back to the symptoms of PD.

¹ Why on earth, you may be asking, should Mr. Fox present symptoms of *overmedication* as if they were symptoms of Parkinson's? One reason might be that it makes for much better drama: a person whose medications aren't working is most likely to be hunched, drooling, not moving, and possibly even incapable of speech: not very dramatic or romantic. A charming TV actor might not want the public to see such a pathetic image.

Therefore, the TV presentation was possibly calculated to show something highly alarming: the wild, uncontrolled movements that can occur when a person's medications are grossly excessive. Mr. Fox's statement, in order to be correct, should have been "this is what happens when the medications don't work correctly, *due to excess dosage*."

However, the harm has been done. Millions of Americans now believe that Parkinson's disease is a disease of spontaneous, uncontrolled muscle thrashing.

More details about the Big Four

Again, while many of the symptoms listed in these headings may be listed separately in some books, one attains a greater understanding of the illness if one sees the relationship between the many symptoms and the four basic categories.

1. Poverty of Movement

Slowness, also called bradykinesia, and difficulty in initiating movement (akinesia) and a reduction in automatic movements such as alternate arm swinging while walking are characteristic PD symptoms.¹ While the arm swing may be consciously forced, temporarily, the arm will stop swinging as soon as the conscious effort ceases. Other symptoms include lack of coordination between arm swing (if any) and stride. Slow, shuffling steps, slow hand and/or arm movements, slow, muffled speech, and slowness in performing coordinated finger movements such as cutting up food, doing up buttons and picking up coins are forms of bradykinesia.

Micrographia, the extremely small, slow, and labored handwriting, is a form of poverty of movement, and can be characteristic of PD.

Unblinking eyes, sagging or useless facial muscles, inability to smile, poor swallow reflex/excess salivation, and inability to move the middle toes are all symptoms of poverty of movement.

2. Rigidity

There are two primary lines of rigidity. One starts at the back of the jaw, then goes down the front edge of the neck's sternocleidomastoid muscle, over the mammary line down the torso, crossing from the abdomen to the outside of the hip, continuing downward along the front (anterior) – outside (lateral) aspect of the legs down to the ankles and stops at the top of the foot. This line corresponds to the jaw-to-midfoot portion of the Stomach channel.

This rigidity makes it difficult to turn the neck from side to side or look behind when driving. The tightening along the front-side of the neck and over the clavicle pulls the shoulders forward into the classic hunched posture of Parkinson's. This type of tightening pulls the head forward and downward, as if the neck is shortening. The distance between the earlobes and the shoulders decreases.

It can look as if the shoulders are pushing upwards to reach the earlobes. This example may give you a good visual sense of this: many women have noticed that for several years prior to their diagnosis with Parkinson's they could no longer wear dangling earrings; the same earrings that used to dangle in space reaching halfway to their epaulets would now rest, slumping, on their raised shoulders.

Rigidity along the torso makes turning in bed more difficult. Ordinarily, a person trying to turn over in bed moves his shoulders, and then the torso, and finally the hips and legs. When the neck, torso, and hips start to move as a rigid unit, one must wrench the whole mass, from neck down to hips, as one piece. This leads, in the beginning, to the belief that one's mattress is not firm enough. After replacing the mattress and realizing that the bed was not the problem, people use a variety of methods to turn over: using the headboard for leverage, bringing the knees up and shoulders forward, making the body as compact as possible before pushing off

¹ LM Jr. McPhee ST, Papadakis MA: *Current Medical Diagnosis and Treatment* (ed 35), Appleton and Lange, Stamford, CT, 1996, pp 885-557.

against the bed with the shoulders or hips and heaving the whole unit over in one move, or asking the spouse to give them a shove.

This rigidity in the muscles that run over the collarbone may also make it stressful, painful, or impossible to raise the arms over the head for an extended period.

Another line of rigidity extends from the point on the wrist crease on the dorsum (the back, not the inside) of the wrist nearest to the junction of the index finger and thumb, and travels upwards past the outer end of the elbow crease, over the bicep, across the front-top of the shoulder, and over the neck to the side of the mouth.

This path corresponds to the above-wrist portion of the Large Intestine channel.

Rigidity at the wrists and ankles prevents a smooth flow of movement when making circles with the wrist or ankle; rotation of the limbs at these joints results in a jerky, “cogwheel” motion. Instead of rotating in a smooth circle, the ankle or wrist rotation motion features a pause and a skip in the vicinity of the thumb section of the wrist, or the front and anterior/lateral portion of the ankle. This pause and skip is due to rigidity in these two, very specific areas.

As Parkinson’s worsens, the increasing slowness and increasing rigidity combine to create a shuffling, labored gait and extreme constraint of movement, and difficulty turning to the side (moving the legs in the anteriolateral direction) when walking.

3. Tremor

The tremor of Parkinson’s often is, for the first few years at least, a “resting tremor.” Resting tremor occurs when the tremory limb is inactive, at rest. In other words, although an index finger may tremor against the thumb when a person is sitting still, activities using the hands will make the tremor stop. Once the limb is at rest, the tremor starts up again. Over time (months or decades), the tremor may worsen so that it occurs even during activity. It may get worse during times of stress or when trying to eat.

The most common form of tremor is the classic “pill rolling” tremor of the hand, in which the index finger rests briefly on the thumb and then bounces off the thumb at 4 to 8 cycles per second.

Sometimes the tremor extends up from the hand and involves the arm. A less common form of hand tremor occurs in the third and fourth finger. This tremor may cause the stiffened digits to vibrate in a fluttering motion or else make a back and forth motion at the wrist.

Tremor may occur in the lower limbs. Tremor can manifest in the neck or jaw. “Although it may ultimately be present in all limbs, the tremor is commonly confined to one limb or to the limbs on one side for months or years before it becomes more generalized.”¹

In times of calm, the tremor is a small quavering movement. A larger, back and forth, semi-rhythmic, involuntary movement with some power behind it can occur during times of stress or anxiety. This larger movement looks like an extreme exaggeration of the quavery, vibrating resting tremor.

4. Postural Instability

In addition to the festinating gait discussed earlier, many people with Parkinson’s have a tendency to fall. Some fall mostly forwards, some fall mostly backwards, some teeter from side

¹ LM Jr. McPhee ST, Papadakis MA: *Current Medical Diagnosis and Treatment* (ed 35), Appleton and Lange, Stamford, CT, 1996, pp. 885-557.

to side, and some find that, when walking, they tend to crash unpredictably into walls and furniture.

Most of these falls stem from the inability to send quick enough mental instructions to those muscles that are supposed to make tiny, balancing compensations: the brain can no longer initiate these automatic body balancing movements subconsciously. This inability to make movement corrections automatically, subconsciously, combined with extreme slowness of muscle response, may lead to frequent falls. A healthy person can, without even thinking, throw out an arm, leg, hip or neck to correct for some unbalanced movement. People with Parkinson's cannot make these quick, automatic movements. Their movements become increasing conscious efforts, and increasingly slow. As a result, the slightest off-center teetering is likely to lead to a fall.¹

This inability to compensate subconsciously is best demonstrated by the balance test that some doctors use for confirming a diagnosis of Parkinson's: the candidate stands with his back to the wall, about three inches away from the wall. The tester gives a quick nudge, or tap, on the shoulder, as if gently pushing the candidate towards the wall.

A healthy person will easily and automatically compensate for the nudge by moving the shoulders, arms, waist, hip, knees, and/or feet, in what are practically invisible movements, in such a way as to prevent falling backwards. A person with Parkinson's may go straight back, thudding into the wall, unable to stop himself. The test is most effective when the shoulder tap is done on the Side of the body where Symptoms First Appeared (SSFA).

Again, as a reminder, all of these symptoms are based on people with Parkinson's who are *not* taking medications. The frequent falls that occur when, due to medication, a person feels impervious to harm are somewhat different from the falls that occur in unmedicated Parkinson's.

FORMING A DIAGNOSIS

The official western medicine position on diagnosing Parkinson's is that a diagnosis of Parkinson's disease cannot be actually confirmed. There is an understanding honored by most MDs that, in order for a person to be diagnosed with Parkinson's, a person must present with symptoms from three of the four main symptoms categories (the Big Four). Again, the four categories are: poverty of movement (also called bradykinesia), rigidity, resting tremor, and poor balance. If a person has symptoms in only two categories, the understanding is that there should be several types of problems in both of those two categories before a diagnosis of Parkinson's can be made. Also, if symptoms from only two categories are seen, neither of these categories should be the postural instability. The most important category is the poverty of movement, slowness.

¹ Many well-meaning physical therapists teach classes in how to keep people with Parkinson's from falling. These classes are pretty much worthless if they try to teach PDers the importance of bending at the knees, leaning in the opposite direction, or any movement related technique. A person with Parkinson's, on the way down to the floor, usually cannot execute a conscious movement such as "I shall drop to my knees." For him to so drop will take a massive amount of conscious effort and the eventual movement so generated may not occur until long after he has already hit the ground. The most helpful advice from these classes is advice oriented towards emphasis on using a good walker or sharing the name of someone who you can hire to install safety bars in the bathroom. Well-meaning advice on the best ways *to move* in order to keep from falling, such as, "try to roll with it, break the impact of the fall," simply miss the point – these people can't move quickly enough to perform counter-actions, no matter how well planned they are.

A common misconception among the general public is that “anything that tremors is Parkinson’s disease.” This is not true. Many illnesses, ranging from blood sugar disorders to heart disease to post-polio syndrome, may cause tremor. Also, other tremor-specific disorders such as familial tremor or essential tremor are not related to Parkinson’s disease.

The list of known side effects of many drugs, especially the antidepressant and anti-anxiety drugs, include tremor or tardive tremor. “Tardive” means “shows up later.” The tremor from legal or illegal drug use or abuse may not even appear until decades after the user has stopped taking the drugs.¹ Therefore, just tremor, without symptoms from other categories, does *not* support a diagnosis of Parkinson’s disease. However, many people – and even some poorly informed MDs – do not realize this. They imagine that anything that tremors must be Parkinson’s. These people are not correct. Again, many syndromes include tremor.

Because of the uncertainty in a PD diagnosis, neurologists will usually request a brain scan of a person in whom PD is suspected. The brain scan cannot confirm a diagnosis of Parkinson’s; the scan is to rule out the possibility of a stroke or a tumor, events that can sometimes create symptoms similar to those of Parkinson’s. Both stroke (bleeding or blood clot in the brain) and tumor show up nicely in a brain scan – Parkinson’s does not. Therefore, if a person has several Parkinson’s symptoms in three of the four categories *and* no obvious sign of tumor or cerebral trauma, the doctor may give a diagnosis of Parkinson’s by default: no other diagnosis presents itself.

Atypical and non-classic Parkinson’s

If one’s doctor should say that one doesn’t have classic Parkinson’s, or has “atypical Parkinson’s,” bear this in mind: classical Parkinson’s takes time to develop. A person may have early Parkinson’s that does not *yet* look classic. However, with a degenerative disorder, it may be just a matter of time. Because Parkinson’s is degenerative, trying to diagnose it early, especially when the symptoms are still intermittent, is trying to hit a moving target. On the other hand, “atypical” may mean that one has all the symptoms of classic Parkinson’s *plus* some other symptoms that might indicate another problem is present at the same time. Most doctors do not bother to go much more deeply into the matter than whether or not they can fit a PD-ish label on it. They never, in our experience, bother to differentiate between drug- or toxin-induced parkinsonism and idiopathic Parkinson’s disease.

MISDIAGNOSIS OF PARKINSON’S DISEASE

Misdiagnosis in Parkinson’s disease is notoriously rampant. Even Parkinson’s specialists sometimes argue amongst themselves as to whether or not a particular person actually has idiopathic Parkinson’s. Aside from the basic definition of PD put forward by James Parkinson in 1817, there is currently much disagreement about what, technically, constitutes Parkinson’s disease.

¹ I spoke with a doctor who works primarily with VA in-house patients. He told me that thirty years ago, about 4% of the vets had tremor. Since the late 1990s, a majority of the vets have some form of tremor. He attributes this dramatic increase in tremor to the aging of vets who were given methamphetamines during WWII and the Korean war. The methamphetamines, which are dopamine-enhancing drugs, were usually used to help soldiers and especially pilots stay alert when sleep was not an option. Methamphetamine use is known to cause a tardive tremor that may not manifest for decades, or even until old age – long after the drug usage stopped.

Misdiagnosis discovered in autopsy studies

Depending on which study you read, somewhere between 25% and 30% of the people diagnosed with Parkinson's disease do not actually have PD. These numbers have been generated by various autopsy studies, in which it was found, during autopsy, that a supposed PDer had no Parkinson's-like brain cell modifications. In our own clinic, approximately 30% of the supposed PDers that came looking for treatment did not even begin to fit the standard, western medicine description of Parkinson's disease.

Differing opinions

We've had patients who went from one doctor to another, trying to get a firm diagnosis. One of our patients was told that he certainly had PD by one neurologist, and two subsequent neurologists said that they could not possibly support a diagnosis of Parkinson's disease. In this patient's case, because his family wanted a diagnosis of Parkinson's and the other two neuros weren't sure what he had but suspected Alzheimer's, the family decided to go with the first doctor, who insisted that he take antiparkinson's medications immediately. The medications did not help. In fact, his main problem, which was confusion leading to slow responses, rapidly worsened – probably due to the mind-altering properties of his medication.

Also, we have met a few patients whose misdiagnosis of PD by their neurologists might constitute acts of gross negligence or even malpractice.¹ As an aside, when we examined these glaringly misdiagnosed people, they clearly did *not* have a Qi irregularity in the leg.

PET scan controversy

Even the new PET scans, which can reveal areas of diminished dopamine receptor activity in the brain, but which do not measure dopamine levels or show changes in the substantia nigra cells, are *not* accepted as definitive by all neurologists. In fact, the scans have added a new level of complexity to the discussion: in one study, 14% of the people who had been

¹ In one class that I was teaching, one patient stood out. Her only symptom was a weak arm that didn't swing at all. Three years earlier, she had hurt her arm at work. She had woken up the morning after hurting her arm with a right arm that didn't swing and severe weakness in the fingers of her right hand. She was thirty-six years old. She had no other symptoms of Parkinson's. She saw a neurologist that week. The doctor told her that she must start dopamine agonist medication immediately or she would get worse. She had been taking the medication (at a very low level, because it had not worked at the higher level but she thought she should take something) for three years when she came to my class. It hadn't helped her arm to swing. She realized, looking around the classroom, that what she had didn't match what all the other patients had, in symptoms or in personality. The second day of class, after all the others had spoken in turn about themselves and their symptoms, she stated her case: "I'm not like the rest of you here. You have a certain way about you; I don't mean your symptoms. I'm just a girl with a bad marriage who works in a pub. I don't fit in here."

All the students in the class were able to confirm that the Qi was *not* running backwards in her legs.

After a one-hour treatment session with her the next day, during which I repositioned her arm in the shoulder socket – after which it swung normally – we determined that her problem had been a displaced arm caused by having improperly lifted a heavy basket of chips out of the fryer at work.

Another patient who was grossly misdiagnosed to the point of malpractice had a similar situation, except that his forearm immobility started when his "tennis elbow" surgery failed to heal correctly. The forearm hung limp, useless, trembling after the surgery. The surgeon told him that the operation had been a success, but that he could no longer move his forearm due to the overnight appearance of Parkinson's. Fifteen years later, his only so-called Parkinson's symptom was the unchanged immobility and trembling of the one forearm; he had lived fifteen years in dread of the sudden appearance of the other PD symptoms, none of which had ever appeared.

confirmed by a *panel* of Parkinson's *MD specialists* as having PD had PET scans that were perfectly normal. Does this mean that the doctors were wrong, or the scans?

I also had a patient who had tremor and no other symptoms of Parkinson's disease. Her western trained doctor, like me, was certain that she did not have Parkinson's disease. However, she was adamant that she needed a PET scan. The scan showed reduced dopamine receptor function. That was over a year ago. She still shows no signs of Parkinson's disease other than a tremor.

PET scans do not provide a definitive diagnosis of Parkinson's disease. In fact, their use is increasingly controversial.

As Dr. William Weiner, MD, Chair of the department of Neurology at the University of Maryland Medical Center, said in an interview with *Neurology Today*, "If I saw a patient who I thought had parkinsonism, and I sent him out for a scan and it [the scan] came back perfectly normal, I wouldn't change my diagnosis."¹

The L-dopa test

Recently, despite the fact that nearly anyone who is moving slowly for nearly any reason, including pain or depression, will move better under the influence of L-dopa (a powerful, mind-altering, mood-altering drug), some uninformed doctors have been using L-dopa as a test for Parkinson's disease. If a person with some Parkinson-like symptoms responds to L-dopa, these misguided doctors feel that their diagnosis of PD has been confirmed.

This specious reasoning ignores the fact that many disorders, not just PD, respond well to L-dopa. It also ignores the fact that drug-induced parkinsonism, a PD look-alike that can be triggered by many antianxiety and antidepressant drugs as well as many of the illegal mind-altering drugs sold on the street, will respond very nicely to L-dopa. However, in this latter case, if there is no underlying idiopathic Parkinson's disease, the L-dopa will not only mask the presenting symptoms, it can accelerate the permanent brain damage caused by previous drug use.

The final irony of testing for Parkinson's by using L-dopa is this: people with idiopathic Parkinson's usually do not respond to the medications for several weeks. If the drugs are dosed correctly, as the L-dopa manufacturers point out in their drug inserts, the full benefit of L-dopa may not be evident for even up to ten weeks. However, in a person who has some illness other than Parkinson's, such as drug-induced parkinsonism, depression, exhaustion, chronic fatigue syndrome, or any other illness in which dopamine may be temporarily reduced but the overall dopamine system (the dopamine receptors, dopamine transport molecules, dopamine reuptake enzymes) is still functional, the L-dopa might work very quickly. In idiopathic Parkinson's, the entire dopamine system is somewhat dormant and slow to respond to medication.

In other words, a rapid response to L-dopa might indicate that the person does *not* have idiopathic Parkinson's. A person with idiopathic Parkinson's will respond to L-dopa, but only after the medication has begun to accumulate in the brain. If correct dosage levels – levels that accumulate slowly over ten weeks – are used, a person with idiopathic PD may not have a response to L-dopa for several weeks. Yet, increasingly, doctors with little understanding of the illness or of how the drugs work are using a rapid positive response to high levels of L-dopa as proof of Parkinson's. Some of them give the patient a very, very high dose of L-dopa to "test" for Parkinson's. In these cases, the PDer may notice some mild benefit within a few days. Then

¹ "Study Examines Role of Imaging in Diagnosing Parkinson's Disease," *Neurology Today*, Aug. 2005, p. 47-48.

again, so will nearly anyone else. Also, PDers are particularly susceptible to the placebo response. Hopefully, this fad of making a diagnosis of idiopathic PD on the basis of a rapid (within a day or two) positive response to pharmaceutical dopamine – a fad unsupported by any good research – will soon fade.

Gender bias

We also learned that the motor problems of our male PDers had been immediately acknowledged by their MDs. Women, on the other hand, were often told that their slowdown of motor function, tremor, or rigidity was due to depression or dissatisfaction with life. Many of our female PDers said that it took several visits to the neurologist, spread over several years, before the good doctor admitted that a neuro-motor problem existed.¹

Incompetence

In addition to all this, it must sadly be admitted that many neurologists are incompetent to make an informed diagnosis of Parkinson's. Many cases of PD misdiagnosis are clear to the naked eye. The misdiagnoses run both ways: we saw one "PDer" who clearly had nothing worse than a bad outcome from a knee surgery. We saw another woman who had long had all the symptoms of Parkinson's but whose neurologist had, for ten years, refused to give her a diagnosis of Parkinson's, because, despite her rigidity, poverty of movement, balance problems, and tremor, she was still able to force a smile; this neurologist (wrongly) considered a frozen face and only a frozen face to be the gold standard for diagnosing Parkinson's disease.²

Drug- or toxin-induced Parkinsonism

Some patients have come to us with a long history of using antidepressant or anti-anxiety drugs and sometimes a large history of recreational drug use as well. Very often, their facial twitching and various spasms do not resemble in the slightest the classic tremors of Parkinson's, and, aside from these twitchings and ticcings, they have no classic symptoms of PD. And yet,

¹ One internationally renowned business-woman, only forty years old, with symptoms of fairly advanced Parkinson's (she was using a walker when I met her), was told by her neurologist that, if she would just get married, all her problems would go away. She did actually marry less than a year later. Her symptoms continued to worsen.

² I recall the case of the neurologist who performed the Babinski reflex test (testing the foot's response to a finger stroke on the sole of the foot) on a patient, in order to confirm a diagnosis of PD, and wrote up in his report that he got a negative result – and the patient had his hard-soled shoes on the whole time! The doctor had "tested" the soles of his *shoes* instead of the soles of his feet while looking for a toe-curl response!

Often, in these blatant cases of misdiagnosis, the "cure" to the actual problem is a simple one. Other times, the problem is completely baffling but, nevertheless, is not consistent with a diagnosis of idiopathic Parkinson's disease. Many times, doctors skirt the whole issue by declaring the person to have some kind of "parkinsonism," and then offering drugs for the illness as if it were Parkinson's disease. This is unconscionable: the drugs, when taken by a person who does not have idiopathic Parkinson's disease, can rapidly cause a decline in brain function, and do permanent brain damage. Usually, those people who rapidly develop dyskinesias from the drugs or who need rapid increase in dosage due to addiction or to compensate for rapid development of side-effects are people who do *not* have idiopathic Parkinson's disease. Their problems with the drugs are due to the fact that they did *not* have idiopathic Parkinson's disease. Only people with idiopathic PD or subclinical (not yet obvious) idiopathic PD can actually tolerate the drugs without having over-rapid appearance of side effects. Even PDers only do well with the drugs *if they are dosed correctly* – a remote contingency indeed, based on the hundreds of prescriptions we have seen.

they were given a diagnosis of Parkinson's disease by their neurologists. Interestingly, these patients never had backwards-running Qi in their legs.

Self-diagnosis

Self-diagnosis is very often misdiagnosis. Many illnesses can create some symptoms that seem similar to the written descriptions of Parkinson's and yet, to the trained eye, they are clearly not Parkinson's disease. Many of the self-diagnosed patients we have met did not come within a kilometer of actually having idiopathic Parkinson's disease.

I recall one obese patient with poor diet who had not taken any regular exercise since she was in her 30's, who decided that she had Parkinson's disease because, at age 86, her swollen legs moved slowly (although she talked a mile a minute and gestured very, very rapidly as she spoke). Also, she was increasingly stiff in the morning and had trouble turning over in bed. Furthermore, her arthritic hands caused her to write very slowly. She was adamant that I diagnose her with Parkinson's disease so that I could "fix" these problems. She had stumbled across my work and was so pleased to learn that there was a cure for her "condition." Her doctor agreed with me; she did not have even a hint of Parkinson's disease. She didn't believe him, either.

TREATMENT

Drugs

When I started my research in the 1990s, pharmaceutical (drug) treatment for Parkinson's disease consisted of a dopamine precursor (L-dopa), dopamine agonists (dopamine act-alike molecules), anticholinergics (drugs that stop acetylcholine, the neurotransmitter that transmits brain signals to muscles) and drugs that inhibited the breakdown of dopamine (MAO inhibitors). Shortly after I started this study, drugs that prevent the breakdown of L-dopa in the digestive tract and/or bloodstream were added to the list.

Although most MDs do not realize that the various drugs are best used for specific symptoms, the drugs do have different results. For example, if motor function is still good and anxiety-related tremor is the primary problem, anticholinergic drugs might be used to reduce the tremors by the mechanism of sedating mental and motor function.

If poverty of movement rather than tremor is the most problematic symptom, dopaminergic medications might be a better choice.

Dopaminergic drugs can have many adverse effects. Some adverse effects of the dopaminergic drugs (L-dopa, dopamine agonists, MAO inhibitors) are dyskinesia (erratic, uncontrolled and excessive movement), dystonia (excess muscle tension), insomnia, irregular heartbeat, and mind and mood alterations.

More problematically, these drugs are highly addictive and can cause death of dopamine-producing cells (parkinsonism) and a decline in the number of active dopamine receptors.¹

¹ The direct relationship between the amount of L-dopa dose and a decline in dopamine receptors was proven in the Elldopa study of 2003. For a discussion of the way that this finding was downplayed in the study, a study conducted in part by an employee of a company that is paid to test drugs for FDA approval, please see "Levodopa and the Progression of Parkinson's Disease," *New England Journal of Medicine*, March 31, 2005, p. 1386, Walton-Hadlock, J.L.

Increasing amounts of medication must then be taken to compensate for the addiction and drug-induced brain changes. As the dosage increases, the side effects of the drugs can become hellish – very often, the side effects become more problematic than the actual symptoms of Parkinson’s disease.

Brain destruction

Because patients eventually become unresponsive to drugs or develop intolerable side effects within five to ten years, a new field of experimental, highly intrusive procedures was being practiced even as late as the 1990s. Thalamotomy and pallidotomy, which involve killing brain tissue via electrocoagulation for the purpose of diminishing drug side effects, began in the 1980s and was discontinued by the end of the 20th century.

Stem cell implantation

Experiments with surgical implantation of adrenal medullary or fetal substantia nigra tissue has had, for the most part, disastrous results. One of the more curious results came from the placebo patients in one experiment. These placebo patients had surgeries, but unbeknownst to them nothing was actually implanted in their brains: they had “sham” surgeries. In the younger group of placebo patients, they obtained very good results: their Parkinson’s symptoms were greatly reduced for a long period of time (more than a year)!¹

Many of the people who received the actual tissue implants, however, had ghastly side effects. Some of these side effects included perpetually violent movement that resembled the dyskinesia of over-medication from dopamine-enhancing drugs. Some others had no dopamine-related changes, but the implanted cells developed into teeth and optic tissue. The best results were those obtained by the younger group that received the placebo treatment: surgery, but no actual implantation of fetal cells.

These early experiments have been, for the most part, ignored by those clamoring for stem-cell research money to “find a cure for Parkinson’s,” even though there is a strong feeling among most Parkinson’s researchers that stem-cells will not yield good results in Parkinson’s disease. After all, even if someone found a way to guarantee that the implanted cells would produce a controlled level of dopamine, it seems obvious that a body determined to induce dormancy in its own dopamine cells would be able to eventually extinguish the dopamine production in other, introduced cells, unless those new cells were growing out of control, like rogues, causing violent symptoms of dopamine excess.

Until the actual cause of the dopamine cell dormancy is turned off, there is little to be hoped for in introducing more dopamine cells. Then again, if the cause of the dopamine dormancy is known, it makes more sense to treat that *source* problem, rather than the side effect of decline in dopamine-producing cells.

¹ This otherwise inexplicable long-lasting improvement in movement may be explained by the fact that a brain-opening surgery constitutes a trauma. As such, the surgery and its sequelae may be able to create a trauma-induced increase in adrenaline that will not climb back down until the effects of the surgery have completely healed. This violent boost to the otherwise flagging adrenaline system may be enough to propel a person into somewhat normal movement, just as we see when, in an extreme emergency, an immobile PDer can move with grace and speed using his temporarily loaded adrenaline system. Oppositely, the surge of dopamine that occurs when a PDer feels safe, which occurs in response to placebos, “safe activities,” or maybe even “successful brain surgeries,” can also allow a PDer to initiate movement almost normally for as long as his heart feels safe.

The dying dopamine-cell theory

Younger researchers acknowledge that many PD symptoms do not appear to be dopamine related. Many agree that, since dopamine-cell death is not actually the problem, growing new dopamine-producing cells via stem cell or any other cell source is not the answer. In my very limited experience, it is the older neurologists who still are convinced by the dying dopamine-cell theory.

Meanwhile, since 2001, the National Institute on Drug Abuse has named dopamine the neurotransmitter of pleasure and addiction. As we learn more about dopamine, the failures of the PD-dopamine theory loom larger. In the 1950s, dopamine was thought to be the neurotransmitter of relaxation, the opposite of acetylcholine, the neurotransmitter that conveys muscle tension signals from the brain. This came about when doctors saw that people with Parkinson's responded to dopamine: they concluded, bizarrely enough, that PD must be a disease of excess strength: too much acetylcholine relative to the amount of dopamine. That didn't actually account for most of the symptoms of PD. Anyone who spends time with an *unmedicated* PDer can tell you this disorder is not caused by excess strength and vigor.

This theory had been completely abandoned by researchers by the late 1990s. However, many of the older clinical neurologists (clinical means working with patients, not doing research) have remained utterly unaware of the changes, in the last few decades, of our scientific understanding of dopamine.

The old PD-dopamine theories simply do not fit the facts of the illness. But in the absence of any new theories, some doctors continue to promulgate the old ones.

Brain stimulating implants

Ever since the beginning of the twenty first century, Deep Brain Stimulating (DBS) implants have been receiving excellent reviews from the company that makes the implants and from some of the doctors doing the very expensive implanting surgeries. The stories of those people who have done well with the implants are easy to find on the Internet. All of the write-ups of "satisfied customers" that I have seen were sponsored by the company that makes the implants.¹

Independent researchers are finding that the results of the implants are mixed, at best. The DBS can temporarily (up to one year) reduce the drug dosage need of the PDer. However, once the brain has grown accustomed to the DBS, the need for ever-increasing amounts of the drugs seems to continue to progress, just as it did before the implants.²

¹ I have had to read many newspaper articles *very* carefully before finding that the nationally distributed press releases were, in fact, releases from the manufacturer of the DBS system – advertising disguised as news. In one case, our local paper ran an article on a local man, showing how well he was doing with the brain implant. I had to dig a bit before I found out that the information for the article had been provided, not by an intrepid reporter, but by the doctor who had done the work and the DBS-making company. I am certain that many doctors push for these surgeries with the best possible motive. However, they do seem blissfully unaware of the risks. In my line, I am more likely to hear from people whose DBS surgeries were a disaster.

I recall reading a report from the Canadian public health system that explained why they did not support DBS surgeries: the results were very uneven and the very real risks were not worth the short-term benefit that a minority of people received.

² A strong proof that the doctors fully expect their DBS patients to need drug increases within a year was brought home to me via an early patient of mine who had recovered from PD after ten years on medication, who did

A new development in the DBS field is the finding that altering the brain disruption signal of the implant on a regular basis helps to maintain the effectiveness of the implants over a longer time period. Still, the long-term effectiveness and side effects have not yet been determined.

Our program and the DBS implants are not compatible.

Brain implants are effective, but do not increase dopamine

A very important and highly disregarded finding is that the DBS implants do not increase dopamine levels, yet they allow a person to move with better control. This fits in with the research that shows that dopamine insufficiency is not the sole factor in many Parkinson's symptoms. Again, and follow me closely here, the DBS implants often provide some short term improvement in movement control *without altering the dopamine situation.*

survive the hell of getting completely off his medications. Dominic's previously rigid body parts were limp, and his previously weak ones were getting stronger. He was moving much better than he had been when I first met him, but he was not at full strength yet and probably never would be due to brain damage from the medications. He was plagued by fears and doubts. When his fears got the better of him, a violent tremor would appear. (This is a common problem with PDers who were in our recovery program who used medications prior to working with us.) He lived alone: his children convinced him he should have a brain implant. (There was more to it; he had been on a waitlist for the implants since before he had recovered. His doctor pressured him by saying that if he didn't have the implants when his name came up, he could never have them. The doctor, humoring him, assured him that if he was truly recovering, he could simply turn off the implants if he ever fully recovered. And so Dominic decided to have the surgery.)

At a DBS conference two years after Dominic's surgery, the doctor told the audience that he had a patient who was completely off all medications. Not only that, continued the doctor, he had been off medications for two years. The audience response was one of universal disbelief. Dominic's doctor was accused of lying. No doctor in the audience had ever seen, *despite their assurances to patients to the contrary*, someone with an implant who actually no longer needed medication. This anecdote, told to me by Dominic's sons, should give the lie to the stories doctors tell about "permanently" getting off of antiparkinson's medication following DBS surgery.

As to why Dominic did so well, his Parkinson's was gone but he was living with an inability to initiate adrenaline *or* dopamine release when he was worried. (We had not yet figured out the dissociation part of the puzzle at that time.) The implants do not increase dopamine, they cause the release of adrenaline. Since what Dominic now had was adrenaline- and dopamine-release insufficiency *when worried*, and brain damage from drugs but not actual Parkinson's disease, the implant worked well for him: it encouraged adrenaline release.

However, in my own experience, all my DBS patients except for Dominic have had, ultimately, horrible experiences. Their problems ranged from relentless insomnia (no sleep in six months!) to a continual feeling of fire ants crawling through the skin. I have also heard of ambulatory PDers who became wheel-chair bound as an immediate result of DBS surgery. I will no longer work with a DBS patient. Our treatment protocol is not recommended for people with DBS systems. Recovery from Parkinson's, according to our hypotheses and experiences, requires that a person make the switch from the adrenaline system to the dopamine system. The DBS system works by relentlessly goosing the adrenaline system. Although doctors point quickly to the fact that the implant's electrical signals can be turned off, the implants themselves are never removed. Removal would be a very high-risk event, risking tearing and bleeding in the brain. Doctors do *not* perform removal of the DBS, once it is installed.

As long as the implants are in the brain, they are a mild source of trauma. In fact, they provide enough adrenaline-releasing trauma that most people notice an improvement in their symptoms immediately after surgery, before the stimulation is turned on. This may be related to the responses observed in the placebo patients who had sham brain surgeries: the stress of the cerebral intrusion may create enough fear deep in the organism that adrenaline levels rise for a considerable time. In the case of the implants, the intrusion is small. The brain soon accommodates to it, thus necessitating the need for the electrical shocks to provide further release of adrenaline.

Where is the research headed?

Increasingly, the old, 1950s decision that insisted on a *causative* relationship between idiopathic dopamine-cell decline and Parkinson's seems to not actually hold up. But even today, in 2007, these extremely important points suggesting that the dopamine change seen in Parkinson's is only a small part of the story have not made a dent in the ongoing clinical treatment of Parkinson's. The dominant clinical paradigm of the day still holds that all the symptoms of Parkinson's disease are due purely to a shortage of dopamine. This shortage is, in the current thinking, caused by an inexplicable decrease in the brain's production of dopamine.

The idea that the brain might be *intentionally* decreasing dopamine-making cells because the brain is getting few calls for dopamine release; because an environmental cause (coming from outside the body; injuries are considered "environmental" triggers) is inhibiting dopamine release; or because a dissociation response is causing a tilt towards adrenaline and away from dopamine, is never even considered.

These ideas would suggest that the body is actually behaving in the way that the body is supposed to behave during time of injury or dissociation-inducing trauma, except that the injury never healed and the mental signal to end the dissociation was never initiated. In other words, there is no actual pathology, no real physical illness present: the body is doing exactly what the mind is telling it to do. The result of these electrical and mental instructions is partial dormancy in dopamine-producing cells that weren't being used anyway. This hypothesis is consistent with the fact the researchers cannot actually find a pathology at work in Parkinson's disease.

Researchers *can* find the physiological *results* of reversed Qi in the Stomach channel: cell reundifferentiation in the substantia nigra, debris floating around in the dormant cells, and a decrease in the number of the heart's sympathetic nerve connections. What researchers aren't figuring out is the reason that the body is making these changes.

And so, despite evidence to the contrary, and a drastically new understanding of the role of dopamine, Parkinson's disease is still considered by most clinical doctors as an unfathomable case of, for no apparent reason, low dopamine and nothing but low dopamine.

SUMMARY: HOPELESSNESS

When I started inquiring into the syndrome known as Parkinson's disease in 1998, the view at that time was that dopamine-producing cells in the midbrain were dying, reason unknown. The ensuing dopamine shortage caused all the symptoms of Parkinson's disease: poverty of movement, rigidity, tremor, and balance problems. Brain cells were thought to be incapable of healing or regrowth, and therefore Parkinson's was incurable. Dopamine was considered to be a movement neurotransmitter, not because of research proving it to be so, but because L-dopa allowed movement in PDers.¹ L-dopa or other dopamine-enhancing drugs were

¹ In 2007, almost no one except for old timers in the field of Parkinson's disease considers that dopamine is a movement neurotransmitter. Everyone else has more or less accepted the research of the National Institute on Drug Abuse and the research in psychiatry, research that identifies dopamine as a major neurotransmitter in regulating mood and seeking behaviors.

The reason that pharmaceutical dopamine imparts movement in PDers is that it alters their mood, shifting their behavior into a dopamine system-dominant pattern. PDers' normal system for movement, the adrenaline system, is also still turned on, though it is increasingly operating on a "minimal release" basis. Sometimes, the sensations of joy from the pharmaceutical dopamine can create such a feeling of well-being that the adrenaline system is temporarily turned off. As soon as the drug wears off, however, the habitual thoughts that employ the adrenaline-nerve resume. Others find that their negative or cynical thought patterns are so engrained that even when

the treatment, but their effectiveness waned quickly. Surgical treatments sometimes provided short-term benefits, but as the brain continued to “deteriorate,” the effectiveness of these treatments also waned.

By the year 2000, it had been determined that, in fact, the substantia nigra cells in idiopathic Parkinson’s patients were not dead, but they simply weren’t releasing/producing dopamine. They had altered, reverting back to a different, rather neutral type of cell. Then again, in people with drug- or toxin-induced parkinsonism, these same cells *are dead*.

Regardless of this fact, the paradigm presented to the general public continues to declare that idiopathic Parkinson’s is caused by the “loss” of dopamine-producing cells. New research continues to be performed on a model (usually represented by lab rats) in which the brain cells are killed, even though the brain cells in people with idiopathic PD are not dead.

Throughout this book, I will be redundant with this “cells are not dead” motif because some PDer have a hard time registering this information after they hear misinformed MDs, or after they read inaccurate health articles caroling the old canard about dead dopamine-producing cells.

Also, by the end of the 20th century, it was recognized that dopamine was a major neurotransmitter. Not only was it a precursor molecule that readied the brain for activities such as movement, acting as the go between for consciousness and action, but it also had a role in regulating body temperature and mood, controlling appetite, integrating left and right brain activities, monitoring the immune system, and being the neurotransmitter of joy.

Curiously enough, addictive drugs and chemicals, including alcohol, cocaine, methamphetamine, the opiates, and nicotine, are all addictive because they all elevate dopamine levels. Dopamine is the neurotransmitter of addiction. Part of the reason that elevated dopamine causes addiction is that dopamine is one of the most carefully self-regulated of all neurotransmitters in the body.

Finally, a quick websearch can find solid, recent research showing that various physiological responses such as certain vision reflexes and speech reflexes are different in PDer than they are in control subjects. And these reflexes remain different whether or not the PDer is given an effective level of dopamine-enhancing drugs. In other words, dopamine is not the whole story – not by a long shot.¹

Old paradigms die hard

And yet, despite all the new, conflicting research, including much new research coming in that proves *many* Parkinson’s symptoms are *not* dopamine related, the old Parkinson’s model

flush with dopamine, their adrenaline system behaviors and thoughts are still apparent. In this case, anxiety from the still-turned-on, but not releasing, adrenaline system combines with the flush of joy and joy-induced movement given by the drugs.

Dopamine does not create movement, per se. Acetylcholine makes movement. Dopamine bridges the connection between the joyful idea of movement and the imaging of movement. After the idea has been created and transmitted by dopamine, the actual movement signals that travel along the nerves and out to the muscles are sent via acetylcholine.

¹ Examples of such articles are: Grande, Laura J., Crosson Bruce, Heilman Kenneth, et al, “Visual selective attention in Parkinson’s disease: Dissociation of exogenous and endogenous inhibition,” *Neuropsychology*, 2006, vol. 20, n3, pp. 370-382 and Jurkowski, A.J., Stepp, E., et al, “Variable foreperiod deficits in Parkinson’s disease: dissociation across reflexive and voluntary behaviors,” *Brain and Cognition*, v. 58, n1, p. 49-61.

remains the dominant clinical paradigm. In this model, dopamine exists to serve only as a movement neurotransmitter; the dopamine-producing brain cells are dying of “no known cause” even though research continues to prove that (prepare for a redundancy) they are not dead.

One of the most disheartening things I learned while researching this subject is that my patients’ MDs are, for the most part, uninformed about any research that has happened since they were in med school. To an alarming extent, their post-school information has been almost entirely provided by people with something to sell: drug companies and the deep brain stimulating system manufacturer. Most clinical MDs have no idea whatsoever of the Parkinson’s research findings that have occurred since they got out of school.

Even among researchers, the inertia in the field is widespread. For the most part, the discovery that substantia nigra cells are dormant rather than dead has been of little interest or even of negative interest to PD researchers; most of their work relies on a “Parkinson’s model.” A Parkinson’s model is a mouse with toxin-induced parkinsonism: a mouse whose midbrain cells have been killed. Again, this is a very different situation from that of idiopathic Parkinson’s disease.¹

Among clinical doctors who are out in the field diagnosing patients and prescribing medicine, the new research is more or less ignored. Most clinical doctors cannot keep up with the tremendous amount of research that is going on, even within their own field. There is simply too much information coming in all the time.

It can take up to twenty years for new research to infiltrate the medical community. The exceptions, of course, are medications or treatments that are heavily advertised by their manufacturers. Oppositely, research findings that might decrease the sales of drugs, such as the news that dopamine-enhancing drugs cause permanent brain damage, rarely receive large publicity even within the medical field. Also, unpopular research conclusions have a difficult time getting repeat funding. And so the inertia in the advancement of medical knowledge lumbers along.²

¹ In 2005, I read yet another article on how exercise appears to prevent the worsening of Parkinson’s. This perky article was based on a medical study that showed mice whose brain cells had been killed via toxins were able to improve their physical condition via exercise. This experiment demonstrates *nothing* about idiopathic parkinson’s disease. It does suggest that people with brain damage from toxins can benefit from exercise. However, people with idiopathic Parkinson’s do not have brain cell damage or brain cell death. People with Parkinson’s have an electrical signal in the head that prevents the release of dopamine, regardless of how much a person exercises. People with Parkinson’s, when they do exercise, tend to do it with adrenaline and intensity, rather than dopamine and joy. I have seen that, while exercise improves symptoms of Parkinson’s disease for several minutes or for several hours after the exercise, those people who exercise the most vigorously tend to have the most rapidly developing cases of Parkinson’s. In my limited experience, it appears that the more vigorously the PDer uses his incorrect adrenaline-dopamine relationship, the faster the illness seems to progress.

² For more information see: Walton-Hadlock, JL, *Medications of Parkinson’s Disease or Once Upon A Pill: patient experiences with dopamine-enhancing drugs and supplements*. Parkinson’s Recovery Project, 2003, Appendix 5, “Why Your Doctor Thinks The Way He Does: Fifty years of changing dopamine theories,” pp 555-575 and Appendix 6, “Dopamine Fallacies,” pp 577- 592.

