

*"You say po-tay-to, I say pa-tah-to, You say tom-ay-to, I say to-mah-to...Let's call the whole thing off!"*

*Cole Porter*

## 5. PD TERMINOLOGY

### THE NEED FOR UNIFORMITY OF TERMS

Back at the research project, we were recognizing yet another kind of problem: everyone in the program had different words for describing their symptoms. Some pioneers were referring to their tremors as dyskinesia, and others were saying that their dyskinesia was tremor. Rose used the word "kicking" to describe her arm thrashing and head jerking and "shaking" to describe her periods of stone-like rigidity.

Many had invented a vocabulary. Sometimes it took me weeks just to figure out what patients had been talking about specifically when their "power went out," or their "sinking times" were worse. "Sinking" meant foot-sticking-to-the-floor, and a "power failure" was an inhibiting emotional insecurity related to anticipation of drug failure rather than a problem with immobility, as it turns out.

As for the meaning of the word "Off," the definition varied from one person to the next. For example, one patient used the word Off to mean those times when he could scarcely move. Except during the times when his four daily pills were at the height of their fever, he was immobile, his voice was a whisper, and he could not control his drooling. He would therefore complain that he was Off most of the day. Another patient who complained that he'd been Off all day due to drug failure pointed to his day's lousy golf score, a whopping 140 for 18 holes, as proof that he'd been Off. The drastic difference between the meanings of Off in these two PDers was typical of the chimerical way in which PD vocabulary was applied. While this spontaneity of meaning and the individualized PD patois lent an air of mystery, even poetry, to our verbal communications, it was not practical.

Every drug user had his own slant for the official, medically-approved PD terms, and some had word inventions not found in any book. The misinterpretations that PDers made when applying their unique translations to the official PD literature sometimes led to serious, potentially deadly, consequences. Here follow some selections from case studies that help to illustrate the terminology problem.

### *Rufino*

Rufino was still taking his 1000 mg/day of L-dopa even after his FSR practitioner thought Qi had resumed normal flow in his legs. Soon after the Qi flow change he started experiencing violent ticcing. The ticcing, which he called tremor, would ebb for up to an hour, about twenty minutes after he took his drugs, but then get even worse in the second hour of the pills' effectiveness, which is when the drug levels in the brain were at their highest. Also, his violent shaking would calm down about six hours after a dose, when the immediate hit from the drugs wore off. In the morning, the shaking was not as violent as it was in the evening. By the end of the day, as the meds built up in his body, his shaking was so terrible and powerful that his torso and legs would become stone rigid,

unable to move, while his arm went slamming up and down making a huge movement, faster than the eye could follow. He would be gasping for air, his eyes bugging out in fear, while a force beyond all knowledge seemed to take over his arm and wrench it up and down. His athletic adult sons were unable to restrain it in the slightest. They insisted that it was tremor, however, because it would stop for a brief while, up to an hour sometimes, about twenty minutes to half an hour after taking his pills. One son, a physical therapist who felt strongly that this movement was PD tremor, argued that the father needed more drugs. The other son, a sports coach, argued that the drugs appeared to be part of the problem.

Here is a description of the symptoms, written up by the second son.

Dad had taken his Sinemet two hours earlier. It's like this almost every night: Dad awake at 7:52 p.m., tremor immediate. I had to help him up off the couch. As he sat in his chair at about 8:15, he called to me to help him. He had a very pained, panicked look on his face. "This tremor is going crazy right now! I won't let go of my arm!" He was holding his left arm with his right hand and begged me to do something. I grabbed his left hand and forearm. The forearm was ROCK HARD from the spasming of the tremor. I pulled his hand backwards toward his wrist and raised his arm to relieve the spasm, but then his leg did it. It just seemed to lock in there for about 30 seconds. Then it relieved itself a bit. Then it started again.

Eventually, after this patient reduced his medication slowly and carefully over more than a year, the violent ticcing stopped and his original tremor – a small, fluttering, weak movement – reappeared, amazing him and his family. For the first time, the family members were willing to admit that possibly the violent ticcing was a drug-related twitching – not tremor, a normal symptom of Parkinson's disease. The violent ticcing showed no signs of slowing until he got down to 300 mg/day (from his high of 1000 mg/day) and stayed at this lowered level for three months.

Another patient had violent twitching throughout her body until she decreased from a high of 400 mg/day down to 50 mg/day.<sup>1</sup> When she had been at 50 mg/day for nearly two months, the violent shaking abruptly ceased, and she asked me why she was doing "this really annoying, weird, little flutter thing." When I told her that that was her Parkinson's disease tremor, which was the reason she had started taking the L-dopa, she was amazed. "But the jerky twitchy stuff was much worse than this! Who would ever take drugs that cause that violent stuff just to temporarily mask this stupid tremoring?" I had to point out to her that in point of fact, she had done just that.

"But I thought the twitching was the tremor!" she bleated. "That's why I kept increasing my drugs!"

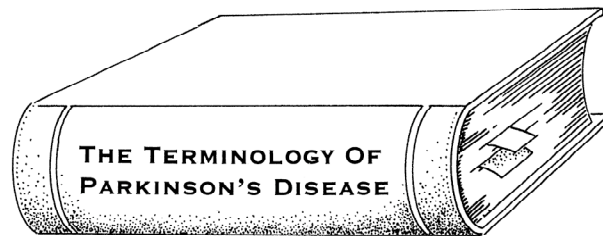
"But I told you that what you had was drug-induced ticcing. I said every week that what you had wasn't tremor, and you didn't believe me. I pointed out that muscle twitching was listed right here in the list of adverse effects of the drugs."

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<sup>1</sup> A person with moderate Parkinson's disease can usually tolerate a dose of 400 mg/day of carbidopa/levodopa without having extreme side effects. However, this patient had apparently recovered from PD. This is why she was grossly overmedicated even at a "mere" 400 mg/day.

“I don’t know what twitching is. I assumed that any extra movement was tremor. What the heck is tremor then, anyway? This stupid, annoying little shaking – is this really the tremor that they talk about?” Why don’t they describe it better in the books?

We needed a standardized vocabulary, and some good, explicit descriptions.



## STANDARDIZED TERMINOLOGY

Based on everything we could read on the subject, we have compiled the following descriptions of the basic Parkinson’s terminologies. If you’re going to go any further with us, you need to be using the same words for the same conditions that we are. So before we get into the really juicy stuff, let’s review some basic terminology that is used and misused by PDers, their friends, and their doctors. These terms are: On, Off, On!, freezing, dystonia, tremor, and dyskinesia.

### ONS AND OFFS

When a person first starts taking antiparkinson’s medications, he can often take two or so pills a day and get even coverage. Even coverage means that there is never an obvious time when the medication starts working or wears off. Instead, a newcomer to the medication may notice that after a few days or months, sleep comes more easily, fluidity of motion returns to some limbs, life just seems a little more worth living, and voice and balance might have some gradual improvement. It may not matter, in these early stages, if the medication is taken in a timely way. Even if an entire dose is forgotten, there may be no apparent lapse in coverage.

Often, a naïve PDer who is at this stage of medication will assure his friends that he’s one of the lucky ones. His attitude might be expressed in this way: “Some of those people have a really bad time with their meds, but they work just fine for me! I’m not going to develop problems with the drugs. I don’t really notice any effect from the drugs; I just feel a little better all day long.” This patient does not yet have an Off/On scenario.

Invariably, after taking PD meds for a few months or maybe a few years, a person will begin to notice that at some times of the day he feels better than at others. His drugs still work most of the time, but at times his movements are nearly as stiff and

uncomfortable as they were before he started taking the drugs. He may notice a real change in functionality if he forgets to take a dose. At this point, the MD will usually prescribe an increase in the medication.<sup>1</sup> Soon, within about two years of starting PD medication, a person will detect noticeable improvement about one to two hours after taking a pill. When the visible (motor) portion of the pill effect wears off, somewhere between two to six hours after the dose, he feels noticeably stiff, depressed, or weak. This is still not On/Off.

Eventually, the drug will start to become “unstable,” to use a biology term. This means that the results of any given dose are not exactly the same as every other dose. This is when a person will start having strange symptoms that are different from the symptoms of unmedicated PD.

Various patterns can indicate that the drugs have become unstable: the onset of medication effectiveness might now start or end with a jolt. The abrupt onset and ending times may be preceded and followed by short periods of feeling even worse than the normal PD feeling. Instead of having a good capacity for movement throughout the day if dosing at regular intervals, this drug user may lurch into movement at certain times. At other times, the pills won't work at all, as evidenced by lack of mobility for several hours after taking a dose. Or a person may have, several doses into the day, a gradual decrease in mobility, until he simply can't move anymore despite taking more pills. Or he may abruptly cease all movement after having been moving just fine. This is the beginning of Offs and Ons. When the Ons and Offs make their first appearances, they may be intermittent, occurring only once a month or so. Over time they may become a regular feature of the drug use. Eventually, these On and Off periods may manifest with no seeming relationship to the timing of the doses. These On and Off episodes are a side effect of long-term use of antiparkinson's medications.

*On: An On is a period during which a person has some modest fluidity of movement. This is in contrast to other periods during which the person has little or no mobility. Not all PD symptoms will disappear during an On. Some PD symptoms may still be apparent even though the person is able to move somewhat. For example, if movement initiation is possible at a somewhat conventional pace, the person is On, even though the person may be drooling or have no facial expression.*

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<sup>1</sup> The implication is that the Parkinson's disease is getting worse, and the dopamine levels in the brain have decreased. Our research suggests that this is possibly not the case. It appears increasingly likely that when the meds become “unpredictable,” it is due to overmedication. It seems, based on our observations, that any amount of buffered L-dopa above 400 mg/day exceeds the amount needed by *any* brain, even in a person with highly advanced Parkinson's disease. (“Buffered levodopa” refers to those medications which combine levodopa with an anti-digestive agent, such as carbidopa.)

Regardless of dosage or stage of Parkinson's (mild to highly advanced), drug withdrawal symptoms – including an apparent increase in PD symptoms – in all our patients who were taking more than 400 mg/day only lasted up to ten weeks, after which the medications became more effective again even at the lower level. The withdrawal symptoms or amplified PD symptoms subsided after the drug reduction phase was over. Our understanding of this is that the medication must have been too high rather than the PD being too advanced. In people taking less than 400 mg/day, this was not always the case – which might mean that these lower doses were actually treating the Parkinson's rather than compensating for addiction. It appears that any amount of buffered levodopa over 400 mg/day is being used to counter the addiction effects rather than the advancing Parkinson's.

*Off: An Off is a period of severely limited mobility or immobility. This Off period is in contrast to those periods when the person can initiate movement and perform the usual activities of daily living at a somewhat conventional pace.*

Again, an On period is not necessarily one in which a person has no symptoms of Parkinson's disease. The On period is a period in which a moderate degree of movement is possible, *as compared to the Off.*

The Off time is not simply any time when a person feels lousy or is showing some symptoms of Parkinson's. Off time refers to those times when he is partially immobilized (motor function comes in fits and starts) or fully immobilized, or partially or fully manifesting his other symptoms related to motor initiation, such as lack of speech or inability to swallow.

### ***Not On, Not Off***

Some people have periods during which they can move but not as well as they would like. This condition is the most difficult to describe with a simple one word term, as it can range all the way from "moving slower than usual" to "not at the top of my form." This in-between phase is best described as "Somewhat On." A patient will do well to make a scale from 1 to 10 to describe what he means by "somewhat On" and monitor himself against this scale. It is important to describe this phase as "somewhat On" rather than "somewhat Off." The Off terminology must be reserved for times of partial (comes and goes) or full immobilization and cessation of motor initiation functions.

It is difficult to be precise here, because many people who cannot initiate a normal stride can still shuffle along. If a person can initiate strides under the influence of the drugs and cannot initiate a stride when the drugs wear off, but can shuffle, he can consider himself to be Off when shuffling. The use of the phrase "initiate movement" refers to the ability to initiate somewhat normal movement. It may still be possible for a person, though Off, to be tremoring, shuffling in slow motion, or falling forward, all forms of abnormal movement. Therefore, there may still be movement of a sort during these times of "immobility."

There are several categories of Off as well, depending on whether or not they are being caused by Parkinson's, inadequate medication, or excess medication. In general, as long as there is the capacity to initiate somewhat normal movements, the person is "somewhat On."

Here is an example to help clarify what is meant by initiation of movement. If a person needs help to get up off the sofa, but once standing can say "Thank you," and then walk across the room, this person is On. If, upon being pulled up from the sofa and supported by the arm, this person shuffles precariously towards the opposite wall, head, eyes and body turning neither to the left nor the right, speaks in a whisper or drools, and the upper body is leaning increasingly forward with relation to the shuffling feet, in danger of falling forward, this person is Off. In the former case, there is some capability for initiation of some normal movement. In the latter case, although there is motion occurring, the PDer cannot initiate any normal movements.

One standard that many patients use is this: if they can move well enough to take their next pill, they are somewhat On. If they are so immobilized that they cannot take a pill and must wait for someone to help them get the next pill in their mouth (fully

immobilized), even if they might be able to yell for help by working themselves, over several minutes, into a mental frenzy, they are Off. This is a good standard. Also, this standard will enable many PDers who complain about their Offs affecting their aim in a game of horseshoes to realize that they were, in fact, never Off during that horseshoe tournament.

These On and Off periods can be gradual or abrupt. They can last several hours or several minutes. They are distinct spans of time, rather than a moment of hesitation in choosing a word or the temporary pause prior to initiating movement. Some patients have long-lasting and predictable Ons or Offs; maybe they are On for most of the day, but then a predictable Off of forty-five minutes duration occurs just after dinner, after which they can move normally again until bedtime.

## ON!

Most patients correctly use the term “On” to refer to any time when they feel even somewhat able to move. However, a few PDers have a slightly different meaning for the phrase “On time.” These patients have learned to use the medications to create a mild sense of euphoria. These patients refer to their bursts of drug-induced confidence and power as their On! (The use of the exclamation point is mine, but it helps make the point and differentiate between the two meanings of On and On! time.) This small group considers ordinary movement, which to the outside observer looks perfectly good, to be merely an in-between stage. Their On! time is those periods when they feel an exalted condition.

A person who is using the drugs to create an On! time of power and confidence will respond with derision to any suggestion that these highs are unnatural and constitute an abuse of the drug. People who have become accustomed to feeling high from their L-dopa cannot understand that there is anything unnatural about their overly bright eyes, their fast speech, and their illogic. They invariably assure one and all that they can be certain that they are not drugged, that they are normal. They point out that, if they were drugged, they would be able to tell. Instead, when they are On!, they feel more normal than they have ever felt.<sup>1</sup>

During their On! time, they often have feelings of supreme confidence. For example, I have had several patients who became compulsive gamblers, helped in part by the invincible, even omniscient feeling created during these On! times.<sup>2</sup> These patients

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<sup>1</sup> This brings the inevitable comparison with Freud’s early research on cocaine, in which he extolled the drug, stating that it was not like a drug, in the way that morphine and opiates were drugs, because the feelings that it produced were so utterly natural. He felt that cocaine was an enhancement of the natural joy that already existed but which was being stifled by complexes. The cocaine merely helped get rid of the complex and bring a person’s natural radiance to the surface. Freud described cocaine as inducing a natural euphoria “which in no way differs from the normal euphoria of the healthy person...”

It was many years later, as he saw the tragic consequences in his friends who were using cocaine, that he began retracting his original statements, and, to the end of his days, he published strongly worded apologies and regrets for his widely published articles on the naturalness and harmlessness of cocaine. In his later writings, Freud called cocaine “the ‘third scourge’ of humanity, after alcohol and heroin.” From R. Julien’s, *A Primer of Drug Action*, Henry Holt and Co, NY, p. 118.

<sup>2</sup> They are, alas, no more successful at gambling than any other John, but they seem incapable of noticing that they are losing more than they are winning.

assure all comers that the drugs do not affect them and that the drugs are merely helping them to express their true selves.

## *Maurice*

Maurice became a complete megalomaniac during his On! times. He could see individual atoms, and they spoke to him. I thought at the time that this was a unique pattern and wasn't even sure if it was drug related. Then during a revisit to Oliver Sacks' book, *Awakenings*, I read that one of his patients also saw atoms under the influence of L-dopa. When I shared this tidbit with my atom-viewing patient, he got very hostile and said that his drugs were not showing him the atoms. Instead, intelligent particles in his mind were imparting the ability to see the atoms. The PD drugs had nothing to do with it. His conversation was filled with unintelligible ramblings about his relationships with the particles – particles that only he could see. Particles were not atoms, by the way. Other people knew about atoms, but only he knew about particles.

This man was driving around town in his speedy car, golfing, gambling, and living an outwardly normal, albeit livelier than normal, life. He told me that he was a very fast driver but that he was safe because the particles told him how to drive. He refused to believe that his drugs were giving him his special particle-viewing powers, but he also felt strongly that PD drugs were designed to be taken to impart an On! feeling, not mere movement. He made no sense most of the time. He often complained to me that he had been Off all day, even though he had been driving around town, golfing, and shopping at the store. I told him that since he was moving, he was not Off, he was On, but he looked at me with gentle pity and told me that I didn't understand.

His wife complained that he twitched and kicked violently all night in his sleep, but he insisted that she was imagining things. She begged him to reduce his medication but he refused. She told me nearly every week that he had become stark raving crazy. He always laughed it off.<sup>1</sup>

So there is a range of understanding of what it means to be On and Off. But in general, and for the purpose of this book, On refers to a condition in which one can initiate some amount of movement, and Off refers to a condition in which one cannot initiate normal movement, can move only slowly in fits and starts with fits outnumbering starts, or cannot move at all. The Off can be either the shuffling slowness and rigidity typical of Parkinson's disease (which may be visible first thing in the morning, before any medication has been taken that day), the exaggerated, crashing Off that occurs shortly after a person's medications wear off, *or the powerful rigidity of overmedication.*

It would be better if there were separate terms for each of these three different states of immobility or slowness, the former caused by PD and the latter two caused by medication, but this is the current convention. But even though we will conform to these standard terms, you will need to learn how to discriminate between PD Offs, Offs that occur after the drug is used up, and Offs from excess medication. More information about

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<sup>1</sup> My student interns nicknamed him Particle Man, a reference to a popular song with that title. He was actually a helpful member of the group: many patients who thought that their drugs should impart an On! decided to reduce their medication after spending a brief time with Particle Man.

the Offs that occur when the dose is expended (Crashes) will be provided in chapter thirteen.

## Freezing

A condition often confused with Off is called Freezing. Freezing is a (usually) short-term, temporary paralysis. The term freezing can cover events such as one's feet suddenly sticking to the floor, or rising up on one's toes when trying to walk forward. Freezing can also include the phenomenon of festinating gait, when the legs sort of freeze up to the extent that they take smaller and smaller steps until they stop altogether, while the torso continues to be propelled forward from its forward momentum. The festinating gait often ends in a crashing halt against a wall or stationary object, or falling to the ground. In addition to the foot freezings just described, freezing can occur in the arms, face, or any other part of the body. It is most noticeable in the feet of course because sudden freezing of the legs or feet sticking to the floor can have the largest motor effect: it stops you dead or dying in your tracks. Freezing does not refer to the "clumsy leg" or perpetually draggy leg that may occur during Parkinson's. This clumsy or draggy leg is usually related to a dystonia, about which you will read later.

The onset of freezing can be quick, like in the western movies where the man with the gun shouts "Everybody freeze!" Freezing does not necessarily extend over a long period of time, although it can. It may go away by itself, or it may require the touch or voice of another to break the spell and get moving again. Freezing is not necessarily a consequence of drug use. It occurs in medicated and unmedicated patients alike, although as a consequence of drug use, incidence of freezing may increase.<sup>1</sup>

For an example of a freezing scenario, one minute the PDer might be walking across a room, and then, abruptly, while crossing through a doorway, become paralyzed, utterly immobile. A moment later, after someone helps him through the doorway, he can move in his normal fashion again. This temporary paralysis in the doorway was not an Off, it was a freezing episode.

Freezing can have very abrupt onset, or it may become apparent after a period of low activity. For example, a person may be reading quietly on the sofa for an hour or two before bedtime. When the time comes to get up, he finds that he cannot move: he is frozen to the sofa. This abrupt moment of freezing may disappear in a few moments if he either puts his mind to it or is given a gentle help up. Sometimes just the mere hand contact from another person is enough to break the spell and allow the freezer to initiate movement. Even auditory cues, such as danceable music, might be able to restore movement initiation in such a case.

This freezing can occur in early stage Parkinson's as well as in advanced PD. Many people, even before they are ever diagnosed, notice that sometimes they have trouble getting moving after having been still for a while. If they are given a supporting hand, or rouse themselves by mental invigoration, they are suddenly able to move perfectly normally.

Research in the 1960's showed that music, human touch, and, in the case of inability to pass through a doorway, visual distraction, were all methods that could break up a freeze. For example, turning to the left or right can be difficult for a PDer. Because

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<sup>1</sup> Nearly all antiparkinson's medications list freezing or bradykinesia (extreme slowness of movement) as one of the adverse effects of the medication.

the anterior-lateral (front-sides) leg muscles are the leg muscles most affected in PD, it is difficult to initiate movement toward the side. The difficulty in turning to the side can cause freezing. By turning in a wide arc rather than trying to make an abrupt turn one can sometimes avoid this type of freezing while turning.

For an example of unmedicated freezing, one of our local PDers, unmedicated-by-choice, manifested this classic form of freezing in his advanced stage of PD: he would shuffle across the room until he came to the wall. Then he would wait until someone came and helped him turn around. Then he would shuffle back to the opposite wall and wait again until help came. Constant moving eased his restlessness, but he was unable to initiate a turn. This form of situational immobility is a type of “freezing.”

A very different type of immobility, also unfortunately named freezing, has developed in the wake of the PD drugs. That type of freezing, which occurs as a response to the medications, will be described in the chapter on dyskinesia.

## **DYSTONIA**

Dystonia (literally wrong muscle tone) is a condition in which a specific body part (usually a limb or the neck) is pulled into an unnatural pose by an overly tight or unresponsive muscle or a pinched nerve. In the mild form, this can look like a clumsy, twisted, or draggy limb, or a tilted head. In its more extreme form, it can look like partial paralysis. It does not look like excess movement, such as tremoring or shaking. Dystonia has a kind of stillness, an inertness or rigidity to it. Because dystonias often appear as paralysis of a body part, they are nearly always wrongly assumed to be neurological in origin and therefore incurable.<sup>1</sup>

### ***Common dystonias***

The distorted body part is being pulled out of its correct place by an overly tight muscle, but the origin of the problem is actually an overly weak muscle, in most cases. Most skeletal muscles (muscles attached to bones, as opposed to internal organ muscles such as heart or stomach muscles) work in paired, opposing sets. When one muscle in the paired set is damaged, its opposing muscle, having no counter balance, will pull uncontested, causing a distortion or twisting in the body part. While a rare form of dystonia can be caused by damage to brain and/or nerve tissues, most dystonias are caused by structural displacements and/or muscle damage. A slightly displaced shoulder from a softball game, a vertebra that has been tweaked to one side during a high school wrestling match, can, over time (sometimes decades), lead to a nearby muscle that is weak, no longer functional, or only partially functional. Its paired, opposing muscle, still able to function, will appear to be, in the absence of its counter muscle, overly tense.<sup>2</sup> The

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<sup>1</sup> Again, up until the last few years of the 20<sup>th</sup> century, anything related to nerves was considered immutable. It was a fact that nerves could not heal or regenerate. After any problem was announced to be a nerve problem, the doctor could wash his hands of it.

<sup>2</sup> Western medicine has until recently stated that all dystonias are due to nerve damage and are therefore incurable. However, osteopaths, chiropractors, and students from many schools of alternative medicine, including Tui Na practitioners, are trained to restore structural components and re-stimulate numbed muscles, thereby curing dystonias. In my own practice, those patients who have been told that they have “permanent brain damage” causing their dystonias have been understandably miffed when their “permanent” condition responds predictably to Tui Na massage.

still-functional half of the muscle pair, no longer being opposed, can progress into a state of excess tonicity, or muscle tone, which is very different from that wooden, even steely, death-like rigidity of Parkinson's disease that can be easily felt in, for example, the anteriolateral muscles of the leg.

### ***Drug-induced dystonias***

Dystonias can also be caused by the PD medications. Because the medications can trigger muscle spasms, they can cause either dystonias or twitches. If the medications set in motion a sustained muscle tension rather than the more common, rapid-fire muscle tensions, dystonias will result instead of ticcing or twitching. In this case, the dystonia is not being caused by one muscle in a pair being weak; the drugs are causing one muscle to tense in an attempt to blow off some of the excess dopamine. These drug-induced dystonias are not related to muscle pair dystonias.

### ***Parkinson's muscle-death dystonias***

Dystonias can also be present in unmedicated PDers. These dystonias, unlike the drug-induced ones, are usually caused by musculoskeletal aberrations. They are usually not neural or chemical imbalances, but instead may be structural or related to the rigor mortis-like muscles that occur in Parkinson's. This extreme rigidity occurs in those muscles that have received incorrect electrical signals for decades. As these muscles become rigid, they no longer pull correctly on their paired muscle. The imbalance between the two paired muscles can cause a distortion, or dystonia.

In Parkinson's disease, these dystonias may exist in addition to the classic PD rigidity of the muscles along the mammary line and lateral leg. Learning to differentiate between medication-induced dystonias, musculoskeletal (injury- or illness-induced) dystonias, and the classic immobility of Parkinson's disease is difficult but important for understanding what the role of the medication is in any given person.

## **TREMOR AND DYSKINESIA**

Dyskinesia and tremor are probably the two most misunderstood words in the lexicon of medicated Parkinson's disease. And yet, determining whether a person is having dyskinesia as opposed to tremor can be critical to understanding whether a person is overmedicated or not, and if so, by how much and when. Unfortunately, nearly all of the antiparkinson's disease medications can cause a form of tremor. This drug-induced tremor is slightly different from the PD tremor, but most doctors do not bother to discriminate between them, which further adds to the confusion.

In anticipation of resistance to the idea that PD meds cause tremor, I will point out right here that "tremor" is clearly listed in the Adverse Effects section of the medication

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If a dystonia does respond to therapy, the neurologist will usually dismiss the recovery, stating that the problem was evidently just a pinched nerve in the neck and not a real dystonia. I had a patient who, when she began to recover from Parkinson's, was told by her neurologist that she had been misdiagnosed and that her Parkinson's symptoms, which had included tremor, facial mask, lack of arm swing, foot dragging, and festinating gait, had evidently all been caused by a pinched nerve in the neck. Depth in this subject is beyond the scope of this book.

inserts for nearly all of the antiparkinson's drugs. Ask your druggist, or read your drug insert. The word tremor will be listed near the top of the list of adverse effects.

Also, for the same reason, namely that most people are highly resistant to the idea that dyskinesia is *not* a part of PD, I will shove in a couple quotes here from a national PD journal to support the statement that dyskinesia comes from the medication: "Within two to five years on levodopa, one-half of people who use L-dopa develop dyskinesias. The movements...(are) not seen in PD patients before levodopa..."

And from the same article, "...Some patients respond minimally to levodopa...(or) ...may doubt their improvement and will stop levodopa. These patients rarely develop dyskinesias."<sup>1</sup>

### ***If alcohol cures depression, then PD drugs cure tremor...***

Because their tremor may subside briefly shortly after taking the medication, many people assume that this is proof that a new tremor or a worsening tremor is not a side effect of the drugs. This is not correct.

In the following example, I will use alcohol, a dopamine enhancer, to represent your PD drug. I am using depression to represent your tremor. Please study the following example carefully, until you understand it deep in your bones: alcohol, in the short term, can ease depression and nervous shaking. Alcohol can, over time, cause increased depression and amplified nervous shakes. Alcohol is an effective treatment for depression in the short term (one to two hours). Alcohol causes worsening depression over the long term. (Six to twelve hours later, there is the likelihood of a hangover, and after extended use, addiction-related changes.) Therefore, the alcoholic will tell you that his shaking is not caused by alcohol. He will prove this to be true by explaining that, shortly after he has a drink, he feels just fine, and his shaking stops for a while. Therefore, contends the alcoholic, alcohol cures shaking and depression. But you and I know that the temporary illusion provided by the alcohol is just that – an illusion. The alcohol does, in fact, cause shaking to worsen over the long term. If the alcoholic had a tendency to shake even before taking up drinking, the alcohol will, over time, exacerbate the shaking.

The alcohol comparison with PD drugs holds up. PD drugs may decrease tremor in the short term, but they can be worsening your tremor in the long term. Therefore, if your tremor improves for a short while shortly after you take medication, this does *not* prove that the tremor is a PD- rather than a medication-induced movement. If your tremor improves for a short while after you take your medication, this does not prove that your medication is not causing or worsening your tremor.

The alcohol comparison is such a great example because almost everyone knows about the two faces of alcohol. In the short term, alcohol can temporarily "cure" depression because alcohol is a stimulant and a disinhibitor of mental or emotional repression. However, in the long term, alcohol is a depressant. Following a drinking

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<sup>1</sup> A.N. Lieberman, MD, Curing Parkinson's disease in our Lifetime: Part 3, Parkinson Report, Fall 2000, Vol. XI, 3, National Parkinson Foundation, Miami, FL, p. 11. Please note the use of the word "rarely." The word "rarely" does not mean that dyskinesia does occur in a small percent of unmedicated PD patients. The use of words such as "rarely" is a convention observed in science writing to denote a remote possibility rather than using the word "never," because "never" is impossible to prove. Also, because other medications, such as the antianxiety drugs and antidepressants, are able to set in motion (tardive) dyskinesias, PDers who do not take antiparkinson's medications but who do take other dopamine-enhancing drugs *may* develop dyskinesias from these drugs, although not as quickly.

binge that was initiated to treat depression, a person will actually be more depressed than he was to begin with. This is well known.

Dopamine-enhancing drugs work in exactly the same way as alcohol; they provide a temporary high, followed by both short-term and long-term increase in those very symptoms that they are intended to treat. Yes, antiparkinson's drugs can cause tremor.

So, with that issue cleared up, let's begin our study of two of the most misunderstood terminologies of PD, dyskinesia and tremor. First, I will explain the literal meaning of the word dyskinesia. Then I will briefly describe both tremor and dyskinesia, emphasizing the differences between them. Then I will go into greater detail on both, giving examples. The explanation will include some physiology of the affected limbs, as well as descriptions of anatomical coping mechanisms that arise in Parkinson's.

### ***Literal definition***

For all you dictionary buffs, the word "dyskinesia" literally means "wrong movement" and refers to any movement which is not correct. This means that, literally speaking, a tremor, being an incorrect type of movement, is a form of dyskinesia.

But in the specialized argot of Parkinson's disease, dyskinesia has a more specific meaning. The "wrong movements" of Parkinson's are divided into two distinct types: tremor, which can be a normal part of Parkinson's, and dyskinesia, which, in the refined language of Parkinson's, refers to those incorrect movements caused by the medication. Again, because this is very important, *tremor can be a normal symptom of Parkinson's disease, and dyskinesia is not.*<sup>1</sup>

The next section of this chapter will be devoted to these two concepts: tremor and dyskinesia. I hope I do not bore you as I pound these two terms into a paste, but I have found that distinguishing between tremor and dyskinesia is one of the most difficult, and most important, discriminations that you can learn. Your doctor may not be able to help you. As mentioned earlier, some doctors in these modern times, accustomed to seeing only medicated patients, actually think that dyskinesias are a symptom of advancing Parkinson's disease; many refer to it, incorrectly, as "worsening tremor."

### **A brief description of tremor**

**Tremor** is vibratory, powerless movement that occurs when the body is relaxed. The classic Parkinson's tremor is called a "resting tremor." A resting tremor is one that occurs when the body is at rest. It may cease while the body is moving. For example, a resting tremor in the hand may stop when the hand is being used in an activity. Even a mere waving of the hand, the briefest of gestures, may cause the tremor to stop temporarily. But after a few moments of stillness of the hand, such as resting the hand in the lap, the resting tremor will recommence. The resting tremor of Parkinson's most often occurs in the fingers, hands, feet, legs, and/or neck and chin.

During times of stress, the tremor can describe a wider arc and become amplified, forming a very large and exaggerated version of the original tremor. This larger movement may include muscles that do not ordinarily participate in the tremor. For example, a patient who normally has tremor in the hand may find that under the influence

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<sup>1</sup> Please review the footnote on page 82.

of social stresses, the muscles of the upper arm also become engaged in a tremor-rate movement. This larger form of tremor is an adrenaline-induced movement that has more power than the usual resting tremor. This is not a form of dyskinesia but it is an adrenaline-enhanced tremor.<sup>1</sup> I will discuss this adrenaline-enhanced tremor in greater detail later in this chapter.

### **A very brief description of dyskinesia**

**Dyskinesia** is muscular, spasming movement. As hinted at heavily in the chapter introduction, dyskinesia is caused not by Parkinson's disease but by the antiparkinson's medications. Dyskinesia is involuntary, medication-induced, excessive movement in the muscles of the neck, face, arms, legs, hands, feet, back, torso, diaphragm, intestines and/or heart.

Dyskinesia movements can be steady and rhythmic or irregular and unpredictable. The movements can be jerky, smooth or writhing. Some of the dyskinesias are simple muscle spasms involving a single muscle, such as the twitch of a facial muscle that lifts the eyebrow just a bit. Other dyskinesias involve groups of muscles, such as the ones that cause grimacing, complete with cheek, mouth, and chin contortions.

Some muscle spasms can be fairly bizarre, such as one eye rolling up, heavenward, and getting stuck in that position for a while. Some are not so much spasms as they are rapid, repeated movements, such as repeated tongue thrusting or toe curling. Still other movements, such as a quick, involuntary swat through the air as if batting at an imaginary fly, can appear almost normal. These can sometimes be explained away by the mildly embarrassed PDer saying, "I thought I saw a gnat..." These movements are involuntary movements, not under conscious control. They can occur in both the so-called "voluntary muscles," such as the arms and legs, and also in the "involuntary muscles," such as the heart, the diaphragm, and the muscles that line the digestive tract. Dyskinesia looks and feels like muscular activity and has some power behind it.

Just to forewarn you, there is a powerful type of dyskinesic movement that is repetitive, like tremor, and which can occur in nearly the same parts of the body as the tremor. It is a hideous parody of the tremor. This dyskinesia appears to amplify an existing tremor pattern, while also involving muscles that are not normally used in tremor. This vigorous shaking is powerful, violent, and often painful.

I've touched only briefly on dyskinesia in this chapter, merely defining it, because after you've read in chapter nine just how and why the brain responds to excess dopamine, there will be an entire chapter dedicated to dyskinesia, the whole dyskinesia, and nothing but the dyskinesia. We realized at some point in our journey that the dyskinesias can, in most patients, be the key to medication evaluation. Dyskinesias may yield the best clues as to whether or not a PDer is undermedicated, dangerously overmedicated or even addicted. By tracking the dyskinesias, we could often calculate

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<sup>1</sup> In the first few editions of my book, *Recovery from Parkinson's Disease*, I referred to the adrenaline-enhanced tremors as "adrenaline dyskinesia." Technically, this was correct, because dyskinesia is any form of wrong movement. But because of the confusion generated by the use of this term, I am now referring to this as adrenaline tremor. This is probably better, as this change of nomenclature enables us to separate the movements into tremors, which are a normal part of PD, and dyskinesia, which is caused by the medication. The most important thing is being able to differentiate between what is being caused by the drugs and what is not.

just where the drug levels were in a person's brain. Tracking them was a lot trickier than we'd hoped: there were layers upon layers of information hidden in the onset delay, duration, and amplitude of dyskinesias when these patterns were tracked over several weeks. So in this chapter, we will simply define most of the terms. In chapter eleven, you will learn more about dyskinesia than you ever wanted to know.

## TREMOR: MORE DETAILS

Now, let's dig deeper into the tremor thing. Because distinguishing between tremor and dyskinesia is so crucial, here is an expansion on the above descriptions: the resting tremor of Parkinson's disease is a rhythmic, vibrating motion in specific body parts. It occurs when the PDer is awake. Tremor usually stops when the person is deeply relaxed, such as when watching TV, having a massage, or dropping off to sleep. It stops during sleep, though sometimes a faint tremoring can occur during dreaming. The resting tremor of PD most often appears in the hand, arm, leg, foot or chin. What does the tremor look like in these areas?

### The Shaking Palsy

James Parkinson, who first described this illness in such supreme detail that no one has since improved upon his description, chose, with great care, the word "tremor" to describe the movements of his patients. According to the dictionary, tremor is "a trembling, shaking, or shivering; a vibratory or quivering motion."<sup>1</sup>

The name that James Parkinson gave to the condition that he studied was "the shaking palsy." (Palsy comes from the same root as paralysis and refers to the immobility part of the disorder.) Parkinson's disease, if unmedicated, is a syndrome of lack of movement, sometimes accompanied by a frail, vibrating quiver. Parkinson's disease does *not* include the violent, repetitive muscle spasms that can afflict those who are taking the medication. It will confound you to refer to the powerful, dyskinetic muscle movements that are caused by the medication as "tremor," even those that mimic the movement in your tremor areas. So from here on out, if you have been referring to powerful, rapid repetitive twitching, ticcing, facial grimacing, exaggerated tremoring, arm twisting, and leg thrashing as "tremor," please stop.

I cannot tell you how many people, while flailing in every direction, look at me in astonishment when I muse that their medications seem to be causing some excess movement. They indignantly retort, "You must be crazy! Don't you see how I'm tremoring? I need more medication, if anything!"

I recall Sonny protesting my suggestion that he was moving excessively by saying, "Look at how bad my tremor is!" as his clenched fist slammed repeatedly into his lower back and his spine arched painfully backward, pulling the back of his head down in the direction of his low back. His wife shook her head and rolled her eyes as if they had this argument twenty times a day. "That's not tremor, that's dyskinesia," she sighed.

So what does a tremor look like? How can I describe it in words? Tremor looks like the fluttering of the leaves on a quaking aspen tree when there is the slightest wisp of wind. Tremor is *not* the groaning and twisting of the limbs of the mighty oak tree when the hurricane hits. Tremor is not muscular. It is not caused by "involuntary muscle

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<sup>1</sup> *Webster's New World Dictionary*. World Publishing Co., NY, 1970.

tension,” despite what it says in the 1960’s medical books. People with PD can tell you that tremor (in the early days of their PD, before the medication) never really felt like muscles clenching and relaxing too quickly. Tremor is not caused by muscle strength but by muscle weakness, vibrating in time with the internal theta wave in the brain.

### ***Hand tremor***

In the hand, tremor takes many forms. When it occurs between the thumb and index finger, it is called “pill rolling.” Less common are the fourth (ring) finger tremor and the five-finger tremor in which all the straightened fingers make fluttery, quick vibrating movements towards and away from the palm. Sometimes there is tremoring in the arm, but because of the weight of the arm, these movements are harder to see. Very often, tremoring in the arm is a feeling more than a visible motion, although, in some cases, tremor can cause the entire arm, neck, and chin to vibrate.

Sometimes the tremor is obvious to all observers, and sometimes it is not. For example, a tremor in an upper arm muscle, such as the bicep, may not cause the arm to bounce as long as the arm is held closely up against the torso. But an activity that relies on the bicep, such as holding an open choir book, will reveal the tremoring as the book bounces up and down.

### ***Foot tremor***

In the foot, tremoring is a steady, quick, small vibration. It is more common for the foot to move as if the toes are bouncing up and down off the floor than for the tremoring to be side-to-side, although the side-to-side does sometimes occur. Because the foot and leg are so much bigger than the hand and arm, these motions will tend to be a little larger in the lower extremities, but they are still fairly small, very rhythmic, and vibratory.

Foot tremor often occurs when one leg is crossed over the other. The leg that is crossing over, suspended over the opposite knee and hanging down unsupported, might make quick, vibrating motions of the lower leg or foot. Tremor of the foot may also occur almost any time that weight is off the foot. Leg tremoring is also a quick, vibrating motion. When standing, a leg tremor can cause the leg to bounce up and down off the floor just a bit, resembling the foot tapping of an impatient person.

### ***Chin tremor***

Tremoring can also occur in the chin and neck. It is a small, vibratory, trembling motion. Chin tremor can resemble the little trembling of the lower lip that occurs when a person is on the verge of crying but trying not to show it. Neck tremor can sometimes cause the entire head to vibrate.

### ***Tremor's cause: weakness and atrophy***

Examine closely the hand of a person with a pill-rolling tremor in the thumb and index finger. Notice that the muscle that is supposed to be alongside the 2<sup>nd</sup> metacarpal (the bone of your hand that connects the wrist to the index finger) is greatly atrophied. (This is the muscle that should pop up, bulging a little, when you press the thumb up tightly to the side of the hand, as you do when making a salute with all five fingers.) The tremor occurs because this muscle is atrophied, not because it is working too hard. If tremor were caused by muscle power, one would expect there to be strong muscle development in the areas of tremor. What we find is just the opposite: tremor occurs in areas of atrophy.

In Parkinson's disease, muscles in specific areas become slowly atrophied, and the communication of these areas with the corresponding motor area of the brain becomes increasingly weak, even dormant. In the case of the pill rolling tremor, when the deterioration in the hand muscle gets to the point that brain control over this part of the hand is gone, *and* if one has the internal tremoring in the brain which has been making him feel edgy inside for years, if not decades, this internal shaking of the brain will be, in the beginning, relieved when the muscles of the hands so deteriorate that the hand begins to exhibit a bounding pattern that moves in the same time and rhythm as the internal shaking.<sup>1</sup>

When the tremor first appears, its movement actually relieves some of the mental tension caused by the internal shaking. But the internal shaking continues to worsen over time, and eventually, the physical vibrating, together with the internal vibrating, can become traumatic. Meanwhile, as the PD progresses, the parts of the body that were damaged in the arm and leg along the lines in figures 5.1 and 5.2 become increasingly atrophied or rigid, respectively.

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<sup>1</sup> Not everyone with PD has tremor. This internal schism in the brain is usually only present in those who have a history of foot injury on only one side of the body. Those PDer's who received similar injuries on both feet tend not to have tremor. They still have the shut down of the dopamine-producing system and atrophy along the arms and legs, but it is symmetrical and does not create the internal schism that so rattles the brain of the tremoring PDer.

*The Large Intestine channel*

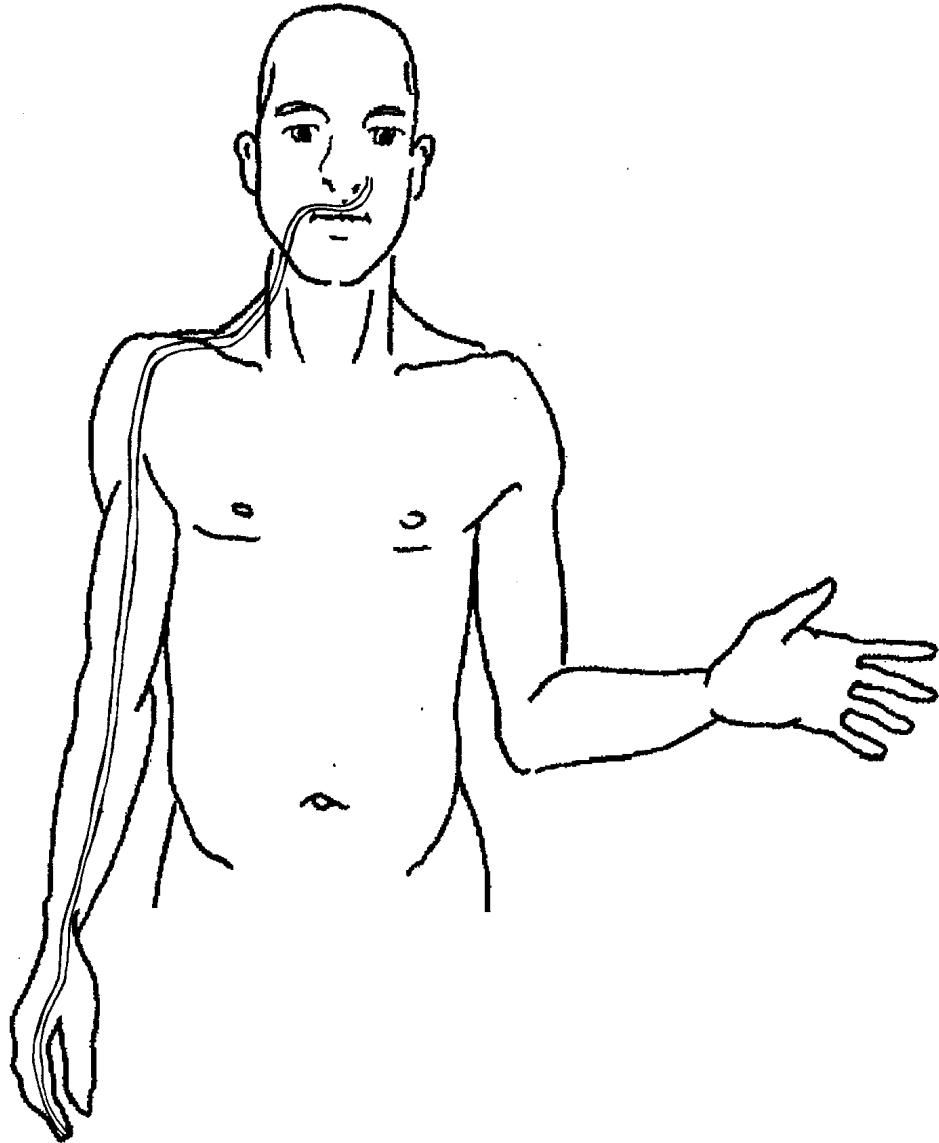


fig 5.1

The obvious atrophy in the muscle on the hand by the 2<sup>nd</sup> metacarpal continues in a line up those muscles that run up the arm to the face. In primary Parkinson's disease, the hand and arm tremors occur primarily along the line seen in fig 5.1.<sup>1</sup>

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<sup>1</sup> In compound PD, other injuries are present in addition to the classic foot injury that is present in all PDers. In this form of PD, the location of the additional injuries and insults will determine which areas become atrophied and which become rigid. The almost infinite combinations possible account for the uniqueness of the symptoms of compound PDers; no two are alike.

As these areas up the arm weaken and brain contact further declines, they too are unable to resist moving in time with the driving electrical drumbeat of internal tremor in the brain. These atrophied areas shake in time with the injury-induced theta wave pattern because they cannot resist it – they are without control, having no strength and no connection to the brain's motor area.

If there is weakness along other leg channels (most often in the Gall Bladder channel), these damaged areas also cannot resist the compelling vibrations of the internal tremor, especially as the Stomach channel is not able to provide stabilization. Obvious quivering in the leg can manifest.

These quivering movements are called tremor. They are neither powerful nor muscular: they are the helpless movements that occur in abandoned body parts. During moments of movement, these useless areas are swept up in the movement of adjacent, still-functional muscles. At these moments, the tremor stops. When the supporting movement of the adjacent muscles ceases, the atrophied areas once more pick up the rhythm of the internal tremor.

There can sometimes be pain or spasm along the edges of these atrophied muscle lines. The pain and tension come from the muscles that border this corridor of degeneration. The body uses those muscles that are alongside the enfeebled ones in an attempt to still the tremor, but these muscles get exhausted from trying to control the errant groups that are trembling away. Hours of trembling in fingers or arms can cause painful tenderness or soreness in those muscles of the arm that can still work. So even though the tremor itself is not muscular, the efforts of the body to control the tremor are muscular, and these can cause tightness, tension and pain in the muscles.

Also, just because the movements of tremor are described here as fluttering or vibratory, this does not mean that they are insignificant or harmless. Incessant tremors can aggravate attempts at eating, speaking, dressing, writing, and nearly all the basic functions of life.

The relentless, ineffectual tremor can cause tremendous aggravation. Tremor can be a terrible symptom. Just because tremor is feeble does not mean that it doesn't drive one to desperation.

### *The Stomach channel*

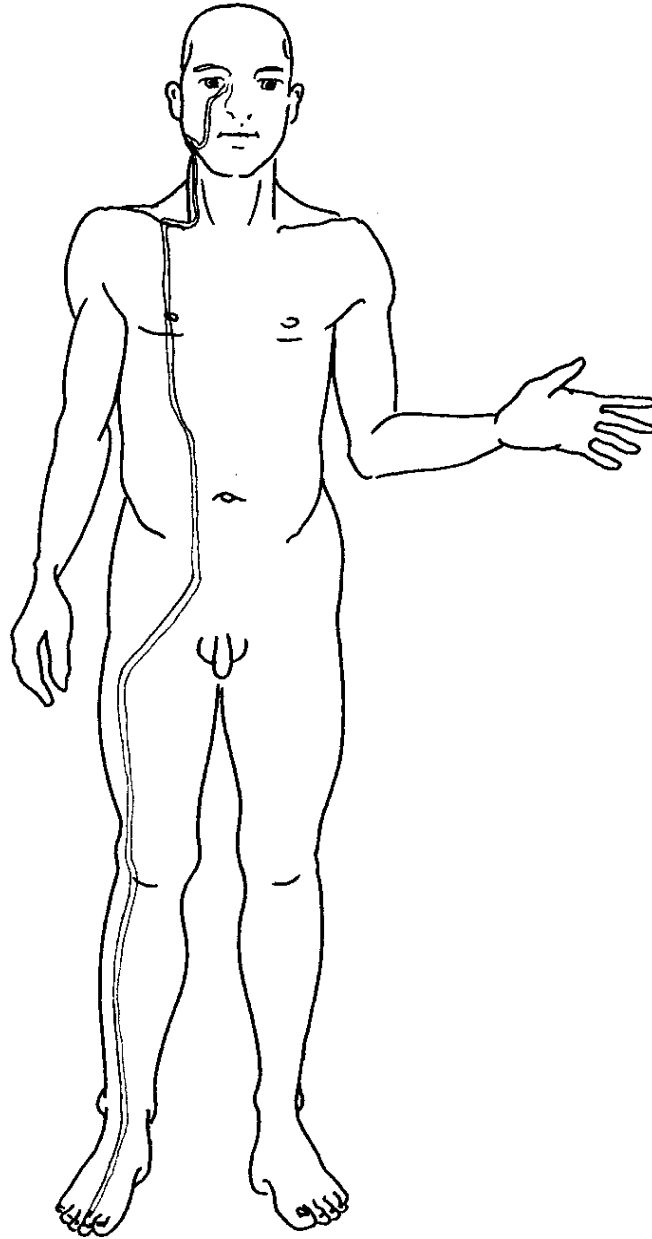


fig 5.2

The neck, chin, and muscles of the torso and leg become steely and unresponsive to brain command (as during rigor mortis<sup>1</sup>) along the lines of the Stomach channel.

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<sup>1</sup> Rigor mortis is the Latin term for the extreme rigidity of skeletal muscle that occurs a few hours after a person dies; the muscles become hard as wood and inflexible.

## Adrenaline Tremor

There is a form of Parkinson's movement that looks like a gross exaggeration of the tremor.<sup>1</sup> This adrenaline-driven, large version of tremor occurs naturally in PD and is not related to the medication. This type of tremor is produced by situations that create a sense of extreme anxiety or fear. It was hypothesized in my first writings that it is powered by adrenaline, and this hypothesis seems to be holding up.

In the book *The Shaking Palsy* by James Parkinson (dated 1817), he notes that even a person with advanced Parkinson's disease can move quickly and smoothly in times of emergency. This is most likely due to the release of adrenaline during an emergency. Adrenaline is sometimes referred to as the "fight or flight" molecule, the chemical of emergency.

Even in the early stages of Parkinson's a patient may experience adrenaline tremor. In a stressful, adrenaline-producing situation, adrenaline goes to all muscles, *even the damaged ones*. Adrenaline goes to the brain and also goes directly to the muscles. In an emergency, under the influence of adrenaline, the normal, dopamine-using system of motor function is overridden by the emergency mode.<sup>2</sup> Although they are very different (opposites, really), if you consider dopamine to be a movement/thought initiator during moments of calm, you might think of adrenaline as a form of super-dopamine: it initiates super-fast movement or thought in times of emergency. A person with Parkinson's might not have much movement-initiating dopamine, but he will still have the ability to manufacture movement-initiating adrenaline during dire emergencies. Under the influence of adrenaline he may demonstrate brain and motor function that resemble normal movement.

Adrenaline can cause amplification of tremor in unmedicated PD. How? How does adrenaline affect the damaged areas, those areas that tremor, since those areas are not in contact with the brain?

Ordinarily, the nerves of the tissues that are on the line of degradation no longer get switched on or off because, in addition to these particular muscles being physically degenerated, their nerves have been turned off in the brain. Even if dopamine is present in the brain it cannot get through to these damaged areas, these areas that flutter in helpless tremor. But when adrenaline takes over, the situation is altered. Adrenaline floods the body, not just the brain, and can go directly to the muscles, *whether they are damaged or not*. Adrenaline allows a person with a broken leg to run away from danger, even though under normal circumstances he would not be able to walk. In a person with Parkinson's disease, a sudden rush of adrenaline gives to those damaged areas along the arm and leg a jolt of "action" chemistry. These areas, ordinarily turned "off" at all times

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<sup>1</sup> This material is from *Recovery from Parkinson's Disease*. It is included again here because so many people taking medication cannot tell the difference between drug-induced dyskinesia, tremor, and adrenaline tremor. If you know this material already, please skip this part. It is detailed, even redundant.

<sup>2</sup> Actually, Parkinson's disease begins to manifest when life-long super-high adrenaline levels start to ebb. However, in an emergency, the adrenal glands can still pump out an emergency dose of adrenaline. When this occurs, a PDer may appear to have normal large motor function again, until the emergency passes. Then, when the adrenaline level recedes back to the wearied, diminished level, the Parkinson's is once again evident.

due to damage, will, under the influence of adrenaline, receive the same “on” signal as all the other muscles in the body.

(Eventually, in cases of very late stage Parkinson's, the body shuts down the movement mechanisms so completely that even adrenaline cannot get through. But this occurs slowly, gradually, over years, and is only apparent in very, very late stage Parkinson's disease.)

### ***Adrenaline in the healthy muscles***

The majority of the muscles in the body of a Parkinson's disease sufferer are in pretty good shape until the Parkinson's disease becomes advanced, and they have the capability for conscious control.<sup>1</sup> These healthy muscles are in contact with the brain and take orders from up top. The brain can control these healthy muscles, even in the presence of adrenaline, and conscious decisions can be made as to whether or not to use these adrenaline-enhanced muscles. That means that if there is an emergency and the body gets flooded with adrenaline, imparting super-human strength to the muscles, these muscles are poised, at full attention, waiting for the brain to give the signal on how to behave in this particular emergency.

### ***Adrenaline in uncontrolled muscles***

However, the weak muscles along the lines of degradation also get charged with adrenaline, but they are no longer under brain control. They no longer connect to the central command. They are no longer coordinated, and the nerves that used to govern them consciously are dormant. So when these damaged areas receive a jolt of adrenaline, they flex and pump, back and forth, back and forth, in a pattern that looks like a magnification of the tremor. They are not healthy tissues, but they can still move a bit when hit with the jackhammer of adrenaline. But there is no motor control coming from Central Command. The brain can't communicate with these areas; they are renegades under the influence of adrenaline. These damaged areas, with their dormant nerve connections to the brain, cannot be controlled consciously like the other healthy muscles.

When these muscles are flooded with adrenaline, they pump back and forth in a very predictable set of motions, namely the motions determined by the function of the specific muscles along the line of degradation. The adrenaline tremor occurs at times when the rest of the body receives a surge of adrenaline, so that powerful, controlled movement is possible in the healthy (*non-tremoring*) areas. Meanwhile, the tremor (damaged) areas explode into uncontrolled movement. These adrenaline-triggered motions in the arm look like variations on a salute and in the leg look like someone tapping his toes violently. They are caused by the frantic flexing and pumping of those muscles that underlie the lines of degradation shown in figure 5.1 or 5.2.

These adrenaline-induced motions are different from the super-fast, inhumanly powerful ticcings of the drug-induced tremor enhancement that can even paralyze the rest of the body, as if Off, while simultaneously hammering away in one or two specific (usually tremor-affected) areas.

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<sup>1</sup> Just a reminder – a decrease in dopamine does not cause Parkinson's disease; Parkinson's disease causes a decrease in dopamine.

## ***Splinting***

In non-emergency situations, when there is not too much adrenaline sloshing around, the healthy muscles will compensate for the ones that have been weakened by incorrect Qi flow. This is called splinting, when healthy muscles do their own job plus the jobs of other nearby muscles that are out of action, for whatever reason. The net effect is that the body can move somewhat smoothly even though not all muscles are working exactly up to snuff.

## ***Emergency movement***

In cases of emergency, even a fairly advanced PDer can move somewhat normally. The tired adrenals produce a blast of adrenaline. Adrenaline is always an alternative neurotransmitter (alternative to dopamine) for initiating motor function. However, adrenaline is most often associated with emergencies in which a person must respond with physical movement. For example, in the case of a pre-civilization dire emergency, a caveman PDer could move perfectly well during the course of the emergency; his surge of adrenaline could initiate all necessary movements of running and fighting. During such active times, his resting tremor would not manifest – because his body was not at rest. The healthy muscles adjacent to his damaged ones were so powered by the surge of adrenaline that they controlled the tremor motions.

## ***Modern man: immobile during “civilized” emergencies***

However, in our modern world, the exact opposite situation can occur in an emergency. In our co-called civilized<sup>1</sup> world, stress is more often found in situations where frantic running or staging a violent attack is unsuitable. Stress can occur while driving the car or while addressing a room full of hostile board members. In tense situations such as these, adrenaline levels surge. The heartbeat increases accordingly, blood flow to the muscles picks up, and the muscles are primed to respond to the slightest command. But the modern person, sitting in a car or speaking from a dais, can't run or throw his weight about when the adrenaline hits. No, Civilized Person orders his or her muscles to be perfectly still, in spite of the stress. The body may be primed for a primitive fight or flight response but the modern social situations that cause stress must be met with the modern social conventions; these most often call for absence of movement, or polite, restrained movement. Instead of the body firing off a muscle response, the modern person must hold that energy inside, even appearing to be at rest. In an adrenaline-producing situation, either on the freeway or at the in-laws, the healthy, adrenaline-saturated muscles of the modern man are poised and alert, not moving, waiting for cues from the brain.

But what happens to those weak and damaged areas, with their damaged nerves and poor brain/motor coordination, which are no longer under conscious control? The adrenaline hits those areas too. Those muscles start firing off. Back and forth, up and down, those muscles contract and relax as fast as they can with all of their limited strength. The other nearby muscles tense up in a hopeless effort to control the adrenaline-charged, spasming muscles. But those "lost" muscles are not under conscious control, and

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<sup>1</sup> Mahatma Gandhi, when asked during a visit to London what he thought of western civilization, replied politely that he thought it would be a good idea.

they jerk and spasm with all the rude, animal strength they can muster. This *animal* strength is much more powerful than the *conscious* strength of a healthy muscle, and thus powerful “adrenaline tremor” occurs. The arm movements typically take one of two forms. The most common motion of adrenaline tremor is “the salute.” The arm goes up and down as if the index finger is going from chest level up to the outer edge of the eyebrow. The next most common form is the “pledge of allegiance,” in which the arm flails back and forth from the side of the body to across the chest. Adrenaline tremor usually lasts for no more than twenty minutes at a stretch, and while it is tiring and can even cause muscle soreness or severe pain, it is not excruciatingly terrifying in the way that super-human drug-induced ticcing can be.

Adrenaline tremor commonly occurs during public speaking or any situation where the body is trying to be relatively calm and controlled but stress is also present. In situations like eating or public speaking, self-consciousness about the tremor itself can amplify into adrenaline tremoring. In such a case, there may be quite a bit of uncontrolled movement going on anywhere along the lines of degradation as well as some rebounding occurring from the rest of the body. Most of this movement will cease, however, when the stress is reduced. Then the body can revert back to the relatively calm, resting tremor.

## **Tremor starters**

### ***Self-Awareness***

Adrenaline tremor occurs during moments of high stress and anxiety and also during moments of return to self-awareness. An example of a “return to self-awareness” is the snap back to reality that occurs after watching an enthralling movie or play. A PDer watching a captivating play might utterly cease to tremor for as long as he is more aware of the characters of the play than he is of himself. But when his consciousness suddenly reverts back to himself, he may have a short burst of adrenaline tremor. The tremor may stop during the play because the concentration is so focused that the internal tremor, which is a signal of self-injury, is bypassed when the attention is focused on others. But once the concern comes back to self, the self remembers that it is injured, restarts its screaming for attention, and the internal tremor is up and running again. For the same reason, the tremor suddenly kicks up during public speaking or any other event that makes a person self-conscious. A sure way to get the tremor started up is to realize that the tremoring has stopped. This bit of self-examination goes straight to the self-awareness zone and triggers the internal source of fear, setting off the internal tremor. The physical tremor follows shortly. The beginning of the tremor at these moments can be almost as large as adrenaline tremor, but after a few minutes, the short fizz of self-consciousness adrenaline wears off and the tremor subsides into the usual fluttering motion.

### ***Wake up tremor***

Many patients experience a burst of adrenaline tremor when they make the transition from sleep to wakefulness. The startling realization, “I’m here! I’m me, and I’m alive,” can often elicit a tremor response even in a person who has almost no tremor during the rest of the day. It can occur in those who take antiparkinson’s drugs and in those who have never taken the medication. It resembles the startle reflex in newborn babies, a reflex that sometimes initiates cute (in babies), infantile tremoring in the chin or along the lines of the Large Intestine and Stomach channels.<sup>1</sup>

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<sup>1</sup> My respected teacher, Jeffery Pang, descended from a long lineage of Asian doctors, taught us that the Large Intestine and Stomach channels are the last to become operational in the human. All the other channels work in utero, but these two do not start until birth. The reason should be obvious: movement of the GI tract in utero would move meconium into the amniotic fluid whence it would flow into the lungs. Also, stomach activity is not needed, since all nutrients are coming from the mother. This is why infant muscle growth and coordination along these channels is retarded relative to other muscle function and coordination.

At the time of birth, the Du channel, which starts at the anus and is most closely related to the spine and midbrain, drops down from its distribution center just between the eyebrows and connects to the upper lip. This triggers the flow of Qi skirting the lips, Qi that is the beginning and end, respectively, of the Stomach and Large Intestine channels. The descent of Qi from the point between the eyebrows to the center of the upper lip also signifies, in some cultures, the descent of the consciousness from the third eye (center of superconsciousness) to the orifice of the mouth (ego awareness). These two channels are the last to develop in the infant and they are the first to weaken with the aging process. This is why Parkinson’s, which accelerates the degeneration of these two channels, so closely resembles those symptoms of aging that, in turn, resemble symptoms of infantilism.

This bout of strong morning tremor may seem as if it lasts an hour but usually it only lasts about five minutes. Many recovering patients have found that this wake up tremor is one of the last symptoms to disappear. It lingers for a long time, as a sort of reminder that the Parkinson's really did exist, that it wasn't just a bad dream or a misdiagnosis.

### ***Tremoring with meals***

A person who has Parkinson's disease and is not yet taking medication will sometimes have adrenaline tremor at meals. The mild, resting tremor can make it difficult to perform the simple actions of eating and drinking. A filled water glass lifted up by a tremoring hand can splash and spill. A tremoring hand trying to hold a fork loaded with rice will likely send rice flying everywhere. This causes tension and stress, the stress causes adrenaline to kick in, and this causes adrenaline tremor, the larger, bouncing movements. It is hard to tell sometimes when the resting tremor leaves off and the adrenaline tremor sets in during a situation like this.

Because the tremoring at meals is such a common symptom, it may be that it is not only anxiety that is triggering the adrenaline but also something deeper. People with Parkinson's all have a history of foot injury in the electrical channel that is named the Stomach Channel. This channel runs from the head to the toes and is involved in the functioning of the gastrointestinal tract. It may be that the stimulation to the Stomach Channel from the anticipation or process of eating plays a part in the mealtime tremor scenario.

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An infant learning to use the index finger or legs will often have large, gleeful tremoring along the same lines as that of most Parkinson's patients. Consider a baby in a high chair who has just been handed a spoon. His first clumsy attempts to hold the spoon might initiate repetitive ticcings in his arm, from the index finger up to the shoulder, with the result that the spoon, held with a death grip, bangs up and down ceaselessly on the tray. If he is enjoying himself, his legs might join in the game. His little legs, like his arm with the spoon, will bounce at the same rate as a Parkinson's tremor. As pureed food flies through the air, his little legs jerk back and forth as his arm jerks up and down, and the spoon bangs noisily and rhythmically on the tray. The mother laughs and the baby laughs. The baby makes no attempt to stop this rhythmic up and down movement, which means that the muscles alongside of the immature, spasming ones are not tense or painful. Eventually, as the muscles along these lines develop a relationship with the motor area in the brain, the baby learns to control a movement that could technically be called, in Parkinson's, "excitement-type," or adrenaline-type, tremor.

## **Summary of incorrect use of the word “tremor”**

Patients often tell me that their medication-induced clenched fists and facial grimaces are “tremors.” There is a trend to refer to any excessive or uncontrolled movement as tremor. When medicated patients start having little twitches in their toes or little facial tics, they rarely appreciate that it is caused by their medication. They assume it is a symptom of Parkinson's disease. As these movements grow into powerful muscle spasms in their gut, face, arms and legs, most people conclude that these new movements must be yet another manifestation of tremor. Sometimes, patients and even some doctors will deem these new movements to indicate a worsening of the PD, indicating the need for an increase in medication. They are wrong.

### ***Poverty of movement***

Why this confusion? Maybe people forget that Parkinson's disease is a syndrome of immobility and rigidity. In addition to forgetting this, maybe they extrapolate from the idea that the only uncontrolled movement in PD is tremor. Then, when their medications cause all sorts of new movement, they just assume that tremor is the word to describe it all. So as a reminder, once again: in unmedicated PD, as the disease advances, movement decreases. The only exception to this immobility and slowness of movement is the small, vibrating tremor and its companion, short-term, adrenaline-triggered tremor.

### ***Still more symptoms***

The terminology in this chapter only applies to symptoms of Parkinson's disease, with or without medication. What about descriptive words for the symptoms of drug withdrawal? The drug reduction symptoms that we were seeing in cases like Becky's didn't necessarily fit the descriptions of any of the basic Parkinson's symptoms, whether drugged or not drugged. The next chapter is selections from Becky's journal, picking up where we left off at the end of the previous chapter. From working with Becky and other patients who were reducing their medication, we had to suspect that there was something going on with drug reduction that wasn't simply the reemergence of PD symptoms. These new symptoms will be named and described in chapter thirteen.

## *Summary*

Due to the use of incorrect nomenclature regarding the symptoms of Parkinson's disease, there is often much confusion generated between patient and doctor, and between patients and otherwise helpful literature. Hopefully, this chapter has provided some clues to why these symptoms occur and the correct nomenclature for each.

When you can differentiate between On, Off, and On!, and if you can distinguish between freezing and dystonia, and between tremor and dyskinesia, then you will be able to construct an accurate mental image of my patients in the upcoming case studies. From this, you may be able to determine parallels or differences with your own case. Also, by mastering the standard vocabulary, you will be able to communicate clearly with your doctor and other PDers with regard to your symptoms. I recall sitting in PD support group meetings where one person after another would discuss recent problems and experiences using inventive references to symptoms, and sympathetic heads would nod in support, but the bafflement in all eyes was clear: no one but the speaker knew which physical symptoms were being discussed.

By being more exact, honing our babble into precise communication – reversing the trend of the ages – we can be more effective in helping ourselves and sharing answers.

