

## Appendix 6

### DOPAMINE FALLACIES:

#### THE STODGINESS OF SCIENCE HITTING HOME

The human tendency to evaluate current observations through the looking glass of previous theory often leads to perpetuation of disproven ideas even in the face of conflicting evidence. This appendix is a collection of ongoing dopamine myths and falsehoods, some of which affect ongoing research, some of which directly affect patients' daily lives.

For example, as noted previously, the scientific gospel of my youth asserted that dopamine was at its highest levels at night. This mis-fact continued to influence research even into the late 1990's despite voluminous evidence to the contrary. The resistance to change in the field of science was driven home to me personally when I published my first article on Parkinson's disease in 1998.

In it I had hypothesized that the pathological electrical patterns present in PD triggered a constant, if only partial, go-to-sleep signal in the brain. This partial (only present on one side of the brain) go-to-sleep signal had been present in the brain for decades, long before the PD became apparent. This errant "bedtime" signal flashed all day long. I proposed that the decrease in dopamine, as seen in PD, was consistent with this go-to-sleep signal. It was likely, therefore, that the sleep-time electrical pattern is a pattern that causes a shut down in dopamine. To put it more simply, dopamine levels decrease during sleep.

I further proposed that it was the decades-long presence in PDers of a pathological, asymmetrical (so that it was only half a signal rather than a full one), 24 hours a day sleep-inducing electrical pattern (a non-dopamine pattern) that explained why the dopamine-producing cells eventually became dormant: the cells never received a full (symmetrical), daytime, dopamine-producing signal.<sup>1</sup> A waking signal, electrically different from the sleeping signal, is necessary to trigger the production of new dopamine-producing structures in the cells. Because the dopamine-producing cells never received a full electrical signal to produce dopamine, the DNA of the cells, over time, ceased or slowed the building rate of the dark-colored cellular structures in these cells.

All cellular components are always being broken down and replaced with new ones. In Parkinson's disease, the cells of the substantia nigra continually dismantle yesterday's dark-colored, and presumably dopamine-making structures, as per the normal, healthy process. But because, over previous untold decades, there was never an adequate waking signal, no building of replacement structures was ever initiated since the time the aberrant signals began. I proposed that after decades of slow dismantling and no replacement, the dopamine producing cells would slowly revert into cells that were no longer dark. They would be sort of neutral cells, closer in structure to embryonic cells, cells that had not yet been assigned tasks. Biologists say an embryonic cell that doesn't

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<sup>1</sup> The waking hours of PDers are dominated by adrenaline instead of dopamine. Adrenaline is a neurotransmitter that provides ample alertness even when dopamine, a stimulant, is absent or diminished.

yet have a specific task is “undifferentiated.” In Parkinson's disease, the cells of the substantia nigra were no longer dark but had become, instead, a pale pinkish. They had very nearly reverted to an immature, or non-specific condition<sup>1</sup> in which they were re-undifferentiated.<sup>2</sup>

The basic electrical theory portion of this hypothesis was not an original idea. It was ancient Asian medicine. The substantia nigra cell change portion was a logical guess as to what the Asian science might imply in the case of Parkinson's disease. The shocking part of this proposal, the part that was the hardest for me to swallow, was the radically new idea that dopamine was a day-time neurotransmitter, that dopamine was more predominant in an awake person than in a resting person. This paradigm shift was necessary because the electrical alteration that I had seen in Parkinson's disease involved an increase in electrical current in the Gall Bladder channel, a channel that runs at highest levels from 11 p.m. to 1 a.m. – the time when a person is traditionally supposed to be falling asleep, or at least experiencing specific, often sleep-related changes in physiology. This electrical alteration in PDers, combined with the recognized dopamine deficiency in PD, suggested that dopamine in healthy people must be at higher levels in the daytime!

### **Dopamine by day – the challenge**

Though it was in conflict with all my previous training, I had come to this difficult conclusion via two bits of evidence. First, the electrical evidence – my patients all exhibited, *even during the day*, a detectable (by hand) electrical pattern that was identical with a nighttime sleeping pattern.

– A bit of Asian medical background is necessary here: a significant portion of this particular current, known as the Gall Bladder channel, runs alongside the head, directly across from the midbrain. From 11:00 p.m. to 1:00 a.m there should be an amperage increase in the energy flowing through the Gall Bladder channel.<sup>3</sup> A change in any body current will necessarily change the electrical influence on its nearby cells. These faint changes in amperage, their effects on adjacent currents and cell-current iterations, as well as their effects on cells within their sphere of influence are considered, in Asian medicine, to be drivers of the cellular changes that occur during the circadian cycle, as well as the drivers of selective DNA expression. The Gall Bladder channel runs posterior to the temple, passes above the ears, and runs over to the nape of the neck before flowing down to the feet. The head portion of this channel very nearly follows the curve of the brain stem and the midbrain, and changes in this channel exert a large influence over the inner brain. As seen in chapter 24, the Du channel is the main driver of the midbrain. The Gall Bladder channel, running somewhat parallel to but in the *opposite direction* of the Du channel, suppresses the power of the Du channel considerably. When

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<sup>1</sup> It has been known for years that the substantia nigra cells in people with *idiopathic* Parkinson's are not dead. The substantia nigra cells in *toxin- and drug-induced* parkinsonism are dead or severely damaged. The cells in idiopathic PDers are merely altered.

<sup>2</sup> This has recently been proven. With regard to the article that proved reversion to brain cells back to an undifferentiated condition (I believe the word they used was “embryonic”), I have a memo to myself on a scrap of paper, “see Jan 1, *Neuroscience*, University of Houston.” (No year, but possibly 2002.) I am still trying to relocate this reference.

<sup>3</sup> Though this subject is far beyond the scope of this book, it should be noted that every channel in the body has a characteristic period during the day when it experiences a surge in power. The circadian cycles are driven by these surges.

the Du channel is suppressed, brain function, especially frontal lobe function and consciousness, is also suppressed. When the midbrain is suppressed, dopamine production is put on hold. –

I suspected, based on my patient sample, that this electrical aberration of excessive energy in the Gall Bladder channel during the daytime was present in all PDers.

I could also assume, based on their diagnoses of PD and the dopamine connection with PD, that these patients all had decreased dopamine and changes in their dopamine-producing cells. This evidence suggested a relationship between the decreased dopamine of PD and the Gall Bladder channel's go-to-sleep electrical signal. The conclusion drawn from this evidence, that dopamine was an awake-time neurotransmitter, flew in the face of the long established fact that dopamine was a relaxant, present primarily at night.

Secondly, dopamine being a daytime neurotransmitter fit the behavioral evidence: if dopamine were a stimulant, rather than a relaxant, it would explain the strange behaviors of my medicated patients. These patients behaved as if their dopamine-enhancing drugs gave them mental alertness and physical spontaneity, not relaxation or drowsiness. I had been observing the behaviors of my patients who were trying to reduce their L-dopa levels: after they made drug reductions, they behaved just as if they were having withdrawal effects from some of the better-known stimulants (cocaine and methamphetamine). This behavior made no sense according to the dopamine-as-relaxant theory but it suggested, instead, that dopamine was a stimulant.

The logical conclusion, and one that I wrote up in my article, was that dopamine was a waking-time neurotransmitter. I stated that in a healthy person, dopamine levels dropped at night, and guessed that the daily electrical signal change was a contributing trigger. It appeared obvious, based on the evidence. I felt very uneasy about this evidence, and I kept looking for the flaw in my thinking; I had been training for over thirty years in Biology – I knew, like I knew the earth orbited the sun, that dopamine was a relaxant. It was painful for me to propose this radically opposite idea. It is never easy to be a traitor to one's training.

The editor of the journal that was considering publishing my research said that the rest of my hypothesis was well formed and supported, but she had a problem with the dopamine idea: it was an accepted fact that dopamine is produced at higher levels at night. Dopamine is a relaxant. She was willing to give me a chance, however. She said that if I could find even one recent research article stating that dopamine levels were higher in the daytime, refuting all the current books on the subject, she would run the article. She was most dubious.<sup>1</sup>

### ***Dopamine by day – the proof***

Here is where it got exciting. I did a search at the local university's Medline computer station. I printed off the first one hundred abstracts that came up under the search topic Dopamine/Sleep. Those hundred abstracts gloriously demonstrated how we scientists transform a guess into a theory and then try to force subsequent information into the theory, whether it fits or not.<sup>2</sup>

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<sup>1</sup> B.G. Grace, chief editor of the long-running and highly respected *American Journal of Acupuncture*, was the most thorough and exacting editor I have ever worked with. Thank you, B.G.

<sup>2</sup> Some famous examples of forcing the foot of evidence into the wrong size shoe of established facts are the increasingly convoluted charts of the heavenly bodies that were drawn up by pre-Galileo

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

In every one of these studies, the main subject of the research was not dopamine or Parkinson's disease, but was some other illness being studied. The night and day blood dopamine levels had merely been checked as a part of the general blood work. In every case the researcher had noticed that, contrary to the “normal” pattern, all of his subjects had higher blood levels of dopamine in the daytime, lower levels at night. In each article, the researcher concluded that the unanticipated and obviously pathological dopamine levels might be contributing to the illness that was being researched. The various illnesses being researched included PMS, narcolepsy, epilepsy, mood swings, muscle cramping, mental retardation – the list went on and on. In every case, the researcher for each article had suggested that maybe the cause of the illness at hand was the pathologically elevated daytime levels of dopamine and decreased nighttime levels.

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

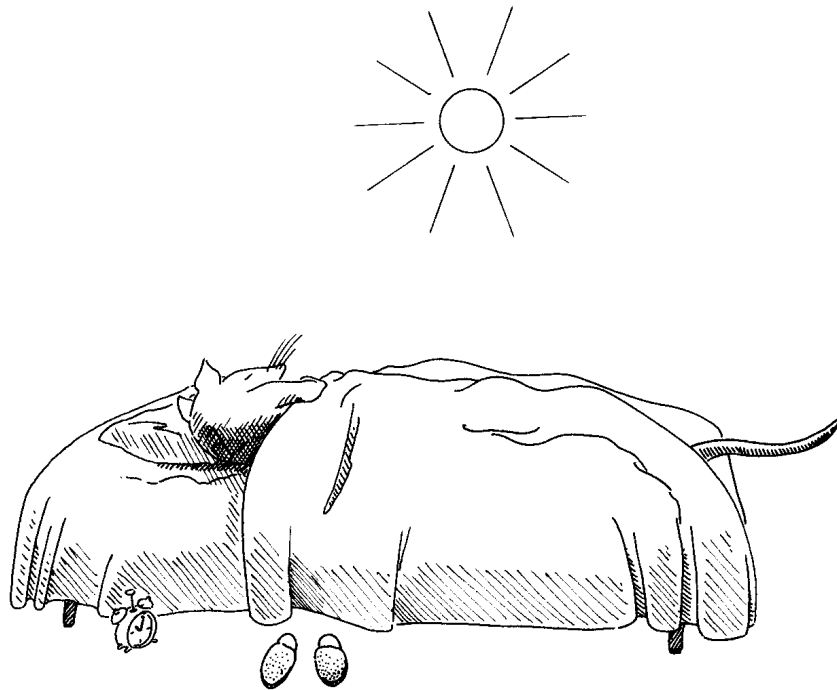
In other words, if a patient was in the headache study, it was probable that this abnormal reversal of the normal dopamine pattern was causing his headaches. Ninety-eight of the one hundred studies followed this pattern. It didn't matter what they were researching; the conclusion in each case was that the illness at hand might be related to the abnormal, reversed situation of blood dopamine levels being higher during the daytime and lower at night.

But, to be fair, there were two studies that had the opposite result. In these two studies they measured not blood dopamine levels, but the actual brain levels of dopamine. This was done by chopping off the heads of the subjects, tossing the heads in a blender, and quickly assaying the results. This gave the most accurate possible reading of brain dopamine levels. In these studies, the dopamine levels were higher at night, thus confirming the facts of dopamine as a nighttime relaxant. There was only one detail that had missed everyone's attention: the subjects in these experiments were rats. Rats are nocturnal – they are active at night, they sleep in the day.

Armed with these research abstracts, abstracts that suggested that 100% of the time human dopamine levels were higher during awake, active time and lower during sleep, and the clinching evidence that nocturnal animals had higher dopamine levels at night during their waking hours, my editor agreed to run my article. She also said, only half in jest, “This contradicts the current facts; they're going to kill you.” This was in 1998, just five years ago. I haven't been killed yet, but hopefully, this vignette illustrates just how hard it is to defy the established “facts.”

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scientists. The invention of the telescope allowed increased accuracy in tracing the paths of the planets. In order that the charts of these bodies might be proven to have an earth-centric orbit, thus supporting the status quo, the descriptions of their travels developed into fantastic shapes and patterns, complete with full stops and mid-air reversals. On the other hand, if, as Galileo proposed, the sun was placed at the center of the solar system, their paths became ovals of simple elegance. The long-standing paradigms of the day made it impossible for such simplicity to be acceptable.



Rats are nocturnal.

***The role of dopamine in hallucinations and psychoses: a subordinate fallacy***

Research published in 1999 confirmed that the drug-induced hallucinations or psychoses experienced by many PDers occur most often in those patients who take dopamine-enhancing medications at night.<sup>1</sup> This corresponds with the well-researched finding that the hallucinations of schizophrenia appear to be caused by a combination of excess dopamine in the lower brain centers coupled with decreased activity in the frontal lobe.

And yet, even today, when my PD patients<sup>2</sup> report terrible problems with insomnia, restlessness, excess movement, and even hallucinations to their doctors, they are nearly always assured by their MDs that what they need is more L-dopa, not less. This thinking is a continuation of the dopamine-equals-relaxation fallacy, combined with the inability of most MDs to keep up with the latest research. Fortunately, most MDs do recognize that hallucinations are a sign of excess dopamine. However, even in my limited experience I have run into a few doctors who needed to have the drug warning's words mailed to them, with the bit about hallucinations highlighted with yellow marking pen, before they accepted that these drugs might be responsible for their patients' hallucinations.

**More dopamine fallacies**

In their attempts to explain away the twitching and spasming being caused by excessive L-dopa, a supposed muscle relaxant, I heard the following explanation from several of my patients: dyskinesia is caused by excess dopamine in the blood and insufficient dopamine in the brain. It was when L-dopa converted into dopamine in the blood before it had a chance to pass through the blood-brain barrier that it triggered all the adverse effects of L-dopa, especially the dyskinesias. Whether they learned this from their doctors, speakers at their support groups, the literature, or they simply misunderstood, I cannot guess. But I heard enough of this theory from my patients, and even from a few doctors, that I include it here.

I have never, to this day, found any actual research that supports the theory that blood dopamine is a stimulant and brain dopamine is a relaxor. In fact, it does appear, based on all the current research in drug addiction, that nearly all the adverse effects of L-dopa, including the dyskinesias, are coming from the brain dopamine excess. (There are a few adrenergic dopamine receptors located outside the brain, especially effecting the bladder and stomach, but these do not relate to the great majority of levodopa-related adverse effects.)

So why is this excess-dopamine-in-the-blood-and-deficient-dopamine-in-the-brain-causes-dyskinesia theory so widespread? Maybe this theory is acceptable because it permits dopamine to remain a relaxant, as previously "proven." In science, any new theory that allows a previous theory to remain standing is going to be a winner.

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<sup>1</sup> "Significant risk factor for developing psychosis in PD is nighttime use of long-acting dopaminergic medications." R. Hauser, T. Zesiewicz, *Parkinson's Disease, Questions and Answers*, Merit International Publishing, 2000, p. 29. (in text reference to Juncos JL, Management of psychotic aspects of Parkinson's disease. *J Clin Psychiatry* 1999; 60 (suppl. 8): 42-53.

<sup>2</sup> These include email correspondents from across the USA and around the world.

***L-dopa additives: how they work***

The idea that blood dopamine causes dyskinesia and brain dopamine does not have played a part in the ongoing reliance on the levodopa additives, also called buffers, and the new levodopa-helper (anti-digestive) drugs, such as Comptan and Tasmar. In the United States, the most popular L-dopa additive is carbidopa (Sinemet). In Great Britain, the most popular is benserazide (Madopar). There are two theories as to why adding carbidopa (or similar molecules patented under different names) to levodopa may be beneficial.

One guess presented by the manufacturer<sup>1</sup> of Sinemet is that carbidopa slows the conversion of levodopa into dopamine in the blood (the blood being an extra-cerebral tissue; in other words, anything outside the brain). The other reason, well-proven, is that carbidopa isn't digested very quickly: by attaching a molecule of carbidopa to the molecule of levodopa, the pair of them can pass through the gut without being mistaken for a bit of dinner.<sup>2</sup>

Without the attached carbidopa, the protein of levodopa can be quickly broken down into small, protein building blocks that the body then uses in the same way as protein from bacon or eggs. In other words, in its pass through the stomach and liver, most levodopa is rendered useless as a drug, and becomes, instead, a snack.<sup>3</sup>

The quick digestion of levodopa was probably the reason that the earliest experiments with levodopa showed no results in Parkinson's patients – the minuscule doses were being digested like hors d'oeuvres. Only when the doses were increased a thousand fold did enough dopamine slip past the digestion process that the tiny amount needed for brain upheaval could get into the cranium, where it then manifested its stupendous effects.<sup>4</sup>

I don't know which of these two carbidopa processes is actually the more significant, the extra-cerebral conversion prevention or the digestion prevention. The latter benefit has been proved. However, many physicians use the former theory to explain dyskinesia despite there being no test/proof basis for the idea that extracerebral dopamine causes dyskinesia.

Therefore, if your doctor is trying to get more dopamine into your brain in an attempt to stop your dyskinesia, he is probably laboring under a false premise. Your problem is that you have too much dopamine in your brain.

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<sup>1</sup> *Physicians' Drug Handbook*, Springhouse, 2001, p. 596.

<sup>2</sup> *Ibid.*

<sup>3</sup> There is another advantage to carbidopa/levodopa that is rarely mentioned: it can be patented. L-dopa exists in nature. In its natural form, it cannot be patented. If levodopa is combined with other compounds in a form that does not exist in nature, this combination can be patented. Such a patent can be worth billions for the drug company that is the first to file for the patent rights.

Conveniently, congress members were persuaded that levodopa, a naturally occurring compound found in fava beans and certain herbs, should be considered a dangerous drug, like opium, and no longer be available for sale over the counter to the general public. At the same time that L-dopa was being legislated out of the health food stores, the patented and expensive version of carbidopa/levodopa was being heralded with great fanfare as the new, improved cure for Parkinson's disease.

<sup>4</sup> Drug addicts have known for decades about the importance of bypassing the voracious digestive system. Bypassing the digestive system via smoking, snorting up the nostril, or injecting drugs directly into the bloodstream enables one to get a drug effect with much, much less drug, up to a thousand times less drug, than is needed to get past the stomach.

***More levodopa adjuncts***

Many of the new drugs, such as Comptan and Tasmar, are attempts to further prevent metabolism of L-dopa in the gut by shutting down critical digestive processes. This tummy turn-off can, in theory, get more of the L-dopa to the brain. These drugs are supposed to be taken when the levodopa drugs show decreasing effectiveness over time, or adverse effects appear. The implication is that it is L-dopa's failure to make it into the brain that is causing these difficulties. The helper drugs increase the payload to the brain.

The possibility that it is L-dopa's presence in the brain that is causing these problems of dyskinesia or decreased effectiveness is never, ever, considered.<sup>1</sup>

This idea that brain dopamine is always the Good dopamine and all other forms of dopamine, including digested dopamine or blood dopamine, are Bad dopamine, goes hand in hand with the idea that brain dopamine is a relaxant and blood dopamine is a stimulant and a causer of problems.

Why am I even bringing this up? When a patient's ever-increasing L-dopa dosage becomes so high that it causes muscle spasms and twitching, eye rolling and tongue thrusting, jerking and shaking, most MDs assure their patients that what they need is still more L-dopa. Since brain dopamine is supposed to be a relaxant, this excess movement, in the eyes of some doctors, indicates a worsening of Parkinson's. (Whatever happened to poverty of movement? Don't forget: many doctors hold that the rigidity is due to excess vigor, and excess movement could conceivably be considered an extension of this excess of strength. The 1960's transformation from viewing Parkinson's disease as a condition of "poverty of movement" to one of "excess strength" is responsible for this honest mistake.) Hence we have the ongoing effort in drug design to get ever-increasing amounts of dopamine into the brains of PDers who are moving *too much*.

Most MDs have embraced the "ever-increasing dopamine" program wholeheartedly – to the extent that many of them have completely abandoned common sense. Now, when a PDer who previously suffered from immobility and poverty of movement suddenly reverses his symptoms and is moving far too much, ticcing and grimacing, his MD may prescribe still more medication.

Under the weight of its own self-belief and political/financial clout, medical science is ponderous and slow to change. Even in the face of obvious problems with the late twentieth century theories of PD, the western medicine search for a cure is still focused, for the most part, on how to get more L-dopa into what is often a dopamine-saturated, dopamine-resisting brain.

Now, in 2003, this may finally be changing. It is not changing because of an admission that the drugs are causing many of the movement problems in PD; it is changing because many American neurologists are beginning to recommend brain surgery for patients whose drugs are propelling them into violent dyskinesias, which are falsely considered to indicate a worsening of Parkinson's disease.

But that is getting ahead of our story. Our story is about how difficult it is to get rid of an incorrect theorem or fact.

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<sup>1</sup> These new drugs appear to cause a rapid worsening of symptoms within a few months. This is not surprising: they serve to increase dopamine levels in the brain – and it is brain dopamine that is causing the adverse effects, including the Ons and Offs that these drugs are trying to prevent. Meanwhile, some doctors continue to prescribe the new drugs, convinced by the ever-worsening adverse effects that what is needed is yet more dopamine!

***Food fallacy: the protein myth***

While we're here separating the wheat from the chaff and the myths from the science, let's look at the popular old wives' tales about protein. The grapevine has it that people with Parkinson's disease should not eat protein. That is NOT the advice given by the pharmaceutical industry.

This myth is an outgrowth of the fact that levodopa is a protein. The advice given by the industry is this: a person should not take levodopa-containing pills immediately before or after a protein-heavy meal. This is a very, very different statement than "a person with PD can't eat protein anymore."

When a person begins to chew a meal that has considerable protein in it, the mouth and tongue detect the protein and signal the stomach to pump some protein digesters into the stomach. Different foods require different digesters that are specific for that type of food. If the stomach is told to produce protein digesters and a person throws back a pill of levodopa, the stomach will digest the pill along with the beef and cheese. Therefore, the wise levodopa user will take his pill an hour before or three hours after he eats a protein-heavy meal. With no obvious, large protein molecules present in the mouth to stimulate a release of protein-digesting enzymes, and with carbidopa somewhat disguising the proteinaceous nature of the levodopa, the small levodopa/carbidopa pill can slip through the stomach pathway without being digested. It is that simple.

This myth, that people with Parkinson's cannot eat protein, has grown to terrible proportions. Easily a fourth of my patients are courting protein deficiency because they have not eaten any significant protein in the years since they were diagnosed with Parkinson's. This is terrible, this is ridiculous, and this is dangerous. I had one patient in my office in tears of self-recrimination because, in a moment of weakness, she'd had eggs for breakfast. She was certain that her Parkinson's would be worsening quickly due to this unforgivable lapse.

And making it all the more strange, if a person refuses protein for a long time, this very absence of protein makes that person's body become so desperate for protein that the little pills of levodopa, which ordinarily would be sneered at by a body that has adequate beans or brisket, now begin to look to a protein-starved body as something worth bothering about. If this pill might be the only protein it is going to get all day, the stomach will produce protein-digesting enzymes just for the pill! Consequently, the starved body focuses its protein-grabbing function on the pill. Ironic, eh?

When my patients started eating protein again, and carefully having their pills an hour before or three hours after a protein-heavy meal, they felt much healthier – *and their pills worked much better*. A person who is getting enough protein and not taking his pills at the same time as protein-heavy meals will find that his pills are not nearly as affected by meal times as they are if he plays games with his protein intake.

By the way, if one is eating a meal that consists primarily of fruit or vegetables, the pill can be taken much closer to meal times. As long as the body is getting adequate protein, a little pill taken about the same time as a fruit salad will slip right past the tongue sensors and the protein-digesting enzymes won't even be alerted.

I was wondering where to fit this in to this book. I suppose this appendix on dopamine myths and false facts is the perfect place. Life with Parkinson's is challenging enough; there's no need to make it a time of protein deprivation as well.

### ***The fallacy of treating immobility with sedatives***

In accordance with the old theory of acetylcholine/dopamine imbalance, people were given anticholinergic drugs to reduce acetylcholine levels. Some patients noticed a subsequent decrease in their tremor and the sedation of their nervous, shuffling pacing. It was assumed, on the basis of these symptom changes, that the acetylcholine/dopamine balance had been improved.

Tremor and restless pacing are the two PD symptoms that are stress-sensitive; an increase in stress can cause increased tremoring. A decrease in stress sometimes allows tremor to decrease. Falling asleep usually stops the tremor completely.

The reason that the anticholinergics appeared to work is that they are essentially knockout drops: they made a person weak, groggy, and sleepy. Anticholinergics decreased tremor because they made a patient tired. This tiredness, meanwhile, from inhibition of acetylcholine, caused most of the other PD symptoms to worsen. Symptoms such as poverty of movement, cogwheeling, micrographia, slow voice, slow thinking, and all the other symptoms that require muscle power or alertness were made worse by the administration of anticholinergics.

To the eye of the doctor, the decrease in tremor seemed to indicate that the patient was doing better. The fact that these sedative drugs sometimes robbed the patient of the strength to get out of a chair or even to think straight had to be balanced against the fact the tremor was repressed. What these doctors didn't realize is that any relaxant, from classical music to antihistamines, would have decreased the tremor. But not knowing this, it did seem as if the calmed tremor was proof of the theory that Parkinson's disease was caused by excess acetylcholine, or, more recently, an imbalance between acetylcholine and dopamine. There are doctors today that still subscribe to this theory and point to the anticholinergic drugs as proof. Certainly, the language of the drug manufacturers supports this thinking: in 2002, in keeping with the decades old acetylcholine/dopamine imbalance theory, the published purpose of these drugs was to "balance cholinergic activity in the basal ganglia."<sup>1</sup>

Today, in a complete reversal, some of the new drugs for Parkinson's disease *elevate* ACh levels. The new thinking here is that, since ACh is necessary for motor function, and PDer's move slowly, maybe they need more ACh, not less. As noted earlier, some doctors, unaware of the processes involved and only aware that both drugs are listed as "antiparkinson's," may well prescribe both these medications to a trusting patient.

### ***"Facing" the facts***

For another glaring example of the way in which medical evidence doesn't necessarily fit the facts, look carefully at the expressionless face of a PDer in advanced, unmedicated (if you can find one) PD. The cheeks sag down heavily. Sometimes, the weight of the lifeless cheeks pressing on the facial bones makes the sinuses feel as if they are collapsing down into the mouth, causing the extreme snoring and sleep apnea sometimes seen in PD.

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<sup>1</sup> *Physician's Drug Handbook*, p. 1064.  
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And yet, as late as 1989, the physiology text at the school where I teach stated that in Parkinson's, "the *rigidity* of the facial muscles gives the face a mask-like appearance"<sup>1</sup> (emphasis added). This is just plain wrong. Rigid muscles are hard, not sagging. Overly tight muscles can cause burning pain, like the pain of a muscle cramp. Rigid muscles in the face would produce the risus sardonicus, the hideous grin that is seen in the case of too much medication. The mask-like, expressionless, *limp* muscles of the Parkinson's face are cold, weak, and numb.<sup>2</sup> The facial muscles in Parkinson's are not rigid with excess tension. They are limp, or even deathly limp, with an utter lack of tension.

When patients instead of doctors describe PD, they say that what they feel is heavy, slow, and dead inside. Though they often have tremendous inner drive, intensity of purpose, and strength of personality, the rigidity that they feel in their bodies is the opposite feeling from strength – it is a feeling of absence, a feeling of leaden lifelessness. However, the claim by the researchers that the loss of muscle tone in the face and the rigor mortis-like woodenness in the muscles along the anteriolateral side of the leg (especially on the side of the body where the PD started) come from an *excess of strength* still remains today in some literature on PD.

### Slanted conclusions

Going far beyond the problem of mere stodgy adherence to faulty conclusions and historical precedent, another problem arises when trying to sort the facts from the fallacies: drug companies have a vested interest in the conclusions of their studies.

For example, the makers of Mirapex, an antiparkinson's dopamine agonist, sponsored a four year study with over 80 participants. The results, written up in January 2002<sup>3</sup> in the *Journal of the American Medical Association* showed, using SPECT scans, that over the 48 months of the study the Parkinson's patients taking L-dopa had a decrease in dopamine transport of 25.5%, while Parkinson's patients using an agonist had a decrease of only 16% in dopamine transport.

Completely ignoring studies that prove that L-dopa causes a decrease in dopamine transport, the Mirapex-sponsored study ingenuously stated that L-dopa did no harm. After all, L-dopa is only dopamine, and people with Parkinson's are dopamine deficient, so giving dopamine cannot hurt them. They then leapt to a wild conclusion that the damage seen in L-dopa users during the study represented the normal damage that occurs from untreated Parkinson's. Because people with Mirapex had measurably less damage than did the people using L-dopa, the manufacturers of Mirapex brazenly were able to make the false conclusion that Mirapex *slowed the progression* of Parkinson's disease!

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<sup>1</sup>G, Tortora, N. Anagnostakos, *Principles of Anatomy and Physiology*, Harper & Row, New York, 1987, p. 332.

<sup>2</sup>The painful cramping and dystonia that sometimes manifest in PD occur when the leaden and unresponsive muscles of damaged tissues cannot maintain a balance with their oppositional, and still-healthy muscles. The healthy muscles pull, but their counter pullers are too ineffectual, and so torsions of the limbs ensue. These torsions are relieved by medication in part because the medications are powerful pain-relievers and mood relaxors – aspects of dopamine that were not recognized until recently. In the absence of pain, the muscles can do splinting, in which working muscles compensate for missing muscles, taking on jobs for which they were not designed. This splinting leads to further pain, but it provides function. As long as the pain relieving function of the drugs is effective, this allows for a joyful, if temporary, resumption of somewhat normal movement in tissues that were dystonic.

<sup>3</sup>*JAMA* 2002; 287: 1653-1661.

## Appendix 6

A report footnoted earlier in this book (see page 42, the Elldopa study) presented measured evidence that people taking L-dopa have a decrease in dopamine transport more than seven times greater than those taking a placebo. Given this information, it does seem that the Mirapex logic, and especially their contention that L-dopa does no harm, is suspiciously self-serving. A less prejudiced conclusion, derived by combining the dopamine transport loss rate seen in the Mirapex study with the result of studies done on placebo (dummy pill) patients, might have been that Mirapex, though less damaging than L-dopa, still nearly doubles the rate at which untreated Parkinson's progresses.

The Mirapex study was conveniently done without any placebo (control) patients, but the conclusion that Mirapex might slow the progression of Parkinson's was widely advertised.

While we are on the subject, and so you will know that I am not personally taking sides in the Mirapex versus Requip contest, it is most likely that all dopamine agonists, not just Mirapex, cause acceleration of Parkinson's disease.

### A nice change of pace

As I dig into the slimy pit of drug company research manipulations, I realize that this chapter is rapidly developing a negative slant. The original aim of this chapter was not to show that profit-seeking pharmaceutical companies do distort the results of their research and publish their false claims in doctors' journals where they are often accepted at face value. My goals in this appendix are to show that old research is hard to uproot; the most well-meaning of doctors have a difficult time in overcoming the paradigms that they learned at their professors' knee. The false root assumptions can not only branch upward and outward into long-lasting fallacies but can also send spreader roots so that, even when the first false root is killed, the errors and fallacies live on.

Unhappily, pointing out the all too human tendency towards adherence to our first teachings can come across, in this medical tome, as anti-doctor. To correct this tone and to bring us back onto a more nourishing perspective, I would like to share the famous prayer written by Maimonides.

#### The Doctor's Prayer

Thy eternal providence has appointed me to watch over the life and health of Thy creatures. May the love for my art actuate me at all times; may neither avarice nor miserliness, nor thirst for glory, or for a great reputation engage my mind; for the enemies of truth and philanthropy could easily deceive me and make me forgetful of my lofty aim of doing good to Thy children.

May I never see in the patient anything but a fellow creature in pain.

Grant me strength, time, and opportunity always to correct what I have acquired, always to extend its domain, for knowledge is immense and the spirit of man can extend indefinitely to enrich itself daily with new requirements.

Today he can discover his errors of yesterday and tomorrow he can obtain a new light on what he thinks himself sure of today. Oh, God, Thou hast appointed me to watch over the life and death of thy creatures; here am I ready for my vocation and now I turn unto my calling.

This humble prayer by Rabbi Moses Maimonides (1135 –1204 AD), a Spanish philosopher and physician who spent most of his adult life in Egypt, is read at some medical school graduations. This prayer makes reference to the ever-changing nature of medicine and, rather than asking for perfect wisdom, asks for the wisdom to be always learning. Most people take it for granted that these noble sentiments motivate their doctors. I know that many doctors work hard to uphold these ideals.

### Continuing education: a doctor's prerogative

Contrary to common belief, there is no requirement that a clinical neurologist must keep up with all the rapidly changing “facts” of brain science. In fact, it would be impossible for him to do so. The new information is coming in so fast that only the researchers can keep up with the changes, and even then they can only stay current within the very narrow field of their specialty. While MDs are required to take a certain number of course hours per year to maintain their license, they are not even required to take these continuing education courses in their own field of specialty, if they have one, nor are they required to take regular exams to test their command of the current findings.

One of the most common email queries I receive is: “How is it possible that my MD has incorrect/outmoded information?” The answer is this: your doctor is human.



Keeping up with the latest: fact or fallacy?

## Summary

The science most often being used today to explain Parkinson's disease is based on logic dating back to more than fifty years ago: logic proven to have been false. The medications being prescribed today for Parkinson's often work in the opposite way from the way they are intended. Because of the multiplied errors in Parkinson's disease theory and misunderstandings about the PD drugs, many treatments are currently being prescribed which actually accelerate the illness or cause side effects that are then treated as if they were an advanced form of the illness.

This premise was stated in chapter one. This appendix was written to show you how and why some of the "scientific" fallacies about Parkinson's have been perpetuated. I have included some of the revelations that I had while doing this research, the first being dopamine's role as a daytime stimulant – despite all the rhubarb about its being a relaxor. A disappointing corollary to this new tidbit was the realization that massive amounts of data already existed that indicated that dopamine was a stimulant but that, with their noses buried in the notebooks of assumptions, no researcher had bothered to stand back and take a gander at the amassed data from a larger perspective.

A bemused moment was enjoyed when I read, in a single paragraph in a respected Parkinson's journal, both that dopamine cells disappeared and that dopamine cells were not lost (see footnote, page 569).

As I pored over thousands of the latest research reports in this field, and was swamped with nearly as many reports from patients about their doctors' advice, theories, and prescribing patterns, I awoke to the fact that modern medicine simply does not reflect modern research; many MDs are practicing medicine based on illogically "proven" or even long-since disproven fallacies.<sup>1</sup> This was perhaps the most disheartening insight of all.

Ultimately, these painful revelations of doctor fallacies were all to the good. When, eventually, we had to advise our medicated patients, "The advice that your highly qualified doctor gives you may be wrong, and even harmful," I was ready, thanks to my experiences with outdated or outright wrong medical pronouncements, to stand by those strong and painful words.

Your inclination may be to disbelieve that your doctor is giving you dangerous, even deadly, drugs. Your spouse, friends, and loved ones may vehemently disagree with you if you say that the drugs are dangerous. They will probably side with your MD. Most of all, they will want to believe in Science. Unfortunately, there are wise MDs and there are ignorant MDs. There are wise practitioners of Asian medicine and there are ignorant practitioners of Asian medicine. A well-meaning heart and a diploma, even prizes and acclaim, do not necessarily translate into accurate or up-to-date knowledge.

Because of the tremendous amount of rapidly changing information in the realm of medicine, and because, ultimately, who we are and why we have the particular illness we do may be more related to deep mysteries of spirit than to the results of lab tests, the greatest qualities a doctor can have, whether he is eastern or western, are intuition,

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<sup>1</sup> See Appendix 8, p. 560, which quotes the article "Clinical Practice Lags Behind Medical Research" from the *Journal of the American Medical Association (JAMA 2002; 287:1653-1661)*.

## Appendix 6

willingness to learn, and enough humility to say “I don’t know,” or even, “Forgive me, I was wrong.” These qualities are not yet being taught even in the best of medical schools.

Hopefully, this appendix demonstrated that science is not actually about facts. Science is about politics, money, and faith. New evidence is usually convoluted to make it conform to the status quo. Proofs, like statistics, can always be arranged to make the desired case. As long as you regard science as a sacred cow and your MD as its priest, you may not be able to make intelligent decisions about your antiparkinson’s medications.