A hypothetical relation between repressive dynamism and Parkinsonian onset—the factor of sympathetic neural balance

I may have a piece of information regarding Parkinson's and a possible factor in ameliorating the odds of developing active symptomatology. It is just a theory, gained through my self-psychoanalysis.

This idea occurred to me this morning over breakfast.

A few basic ideas must first be understood:

- a. Tourette's and OCD, in some instances, are associated with volumetric changes in the basal ganglia. The basal ganglia is an unusual cluster of nuclei, which instates and removes inhibitions (Gazzaniga, 2009). It appears to me, to act as a physiological corollary to the repressive processes. In my self-analysis I have discovered that the illness, OCD, was caused by the repression of libido into the unconscious. This was accomplished in two ways: 1. Regression and subsequent repression of libidinal ideations first changed into aggressive ones, and, 2. Repression of overt sexual ideations.
- b. The ascending activating system provides cortical tone to the mental system as a whole, and is rightly thought to be the source of the *undifferentiated libidinal energy* which powers thought and mental activity in general (Kaplan-Solms & Solms, 2002, p. 267). This ascending activating system, which works on but few neurotransmitters, is to be understood as undifferentiated potential, the particular configuration of elements in which, define all of our emotional states, which may appear as opposites, but are in fact formed by physiological recombinations of the same internally intraconnected substrate: undifferentiated libido. When a *hateful*, or a plainly *libidinal/sexual* ideation, is brought to consciousness *and analyzed*, in both cases the result of the fixation being disbanded is identical: the release of the system to its unbound state of potential—undifferentiated libido is released in all cases (Norman, 2011).
- c. Some of the most basic and primary neural circuitry formed in development, the sympathetic and parasympathetic limbic circuits formed via maternal interaction, which connect the limbic and orbitofrontal areas, are respectively, a sort of pleasure center and cut off for the same, the sympathetic dopaminergically modulated, the parasympathetic noradrenergically modulated, which respond dynamically to internal and social conditions (Schore as cited in Kaplan-Solms & Solms, 2002, pp. 234-237; Norman, 2013). The sympathetic is our pleasure, libido, dopamine, energetic expression and pleasure, the parasympathetic is as an early prototype of repression, as shame.
- d. The brain is a *causally bi-directional* electro-chemical system. Our thoughts are created within a physiological substrate—the nervous system, and in turn, we can see that our thoughts are but patterns of dynamic electro-chemistry, and the dynamic electro-chemistry of the nervous system is in turn, nothing but our thoughts. Therefore our thoughts can affect the electro-chemical configuration of processes which is the physiology of the brain, and vice versa.

OK—now for the idea:

I believe the mental system *in its overall energetic distribution* can be seen as noradrenergic or dopaminergic in its distribution of overall balance. This balance can be encouraged one way or the other. The therapeutic goal is a reversal of repression, both in terms of a redistribution of noradrenergic energetic potential to unrepressed dopaminergic, and also, in terms of the removal of unconscious libidinal ideations from under repression to affect the system as to increase its dopaminergic constituent, and alleviate active Parkinsonian symptomatology. If I am correct, the onset of symptoms may be delayed or curtailed entirely, depending on the level of physical degradation, or lack there of. The system in its state of function *can be assisted* so as to achieve a better outcome, or, prevent the emergence of full-blown symptoms. There are two avenues of support for this notion:

I base this assertion on the following a-priori experiment, carried out many, many times, and just understood as to its implications, this morning:

1. A self-psychoanalysis is not a simple proposition which is finished cleanly. Basic patterns of internal orientation must be altered, a proposition which entails much time and effort—a long back and forth. This circumstance allows one to observe the difference between the two states, one ill and repressed, one well and accepting of the ideations which were under repression, as they exchange places in consciousness. When the illness reinstates itself, my hand develops a tremor, distinctly Parkinsonian in its slow rhythmic oscillations. My mood plummets and I am ill. I use a technique called the "open emotional posture" to access the hidden sexual ideation, and it is always either an angry violent ideation, sadistic, or overtly sexual, always, and then, release the idea to consciousness. At this point the idea must be analyzed. If sexual or sadistic, the resistance must be removed, the distortions clarified, or the symptom, the tremor, worsens. The tremor symbolizes the vacillation between the two wishes, the punitive moral wish for repression, and the id wish, which inhibit each other as a paralysis in a dream, which also works thusly, or an hysterical symptom, which again is formed by suspension between two opposite wishes. Once the inhibition, the punitive, has been removed, the sexual or aggressive wish can be removed from shame...repression lifted. The sexual is then easily admitted into consciousness, the aggressive then is revealed as but a frustrated libidinal wish, and that wish is then admitted into consciousness, and the system responds with a flood of pleasurable sensations, and the tremor stops. Here we see the removal of a repression and the redistribution of the mental system to a state of dopaminergic predominance, of sympathetic predominance.

I suggest that there will be a correlation of negative value between the predominance of sympathetic dopaminergic neural balance, and Parkinson's. Put another way: There will be a negative correlation between Parkinson's and psychoanalysis. Or: Psychoanalysis offers a prophylactic service of some proportion against the onset of Parkinson's. The mechanism of therapeutic efficacy is an increase in dopaminergic predominance. The brain and mind form a causally bi-directional system.

2. Another bit of proof leading toward this idea can be found in an analysis of the conditions of schizophrenia, and hysterical psychosis. In our hypothetical therapeutic model, we are seeking the removal of repressions to increase dopaminergic modulatory balance in the brain. This idea can be supported as we look at those conditions which are characterized by hallucination, which to a great extent indicates the return to consciousness of repressed libidinal content (Freud, 1911 pp. 1-82...citations abound here). In this case, the case of schizophrenia or hysterical psychosis with hallucination, the treatment of choice is Haldol, Thorazine, or another antipsychotic, which as you are probably aware, works by inducing first stage Parkinson's via a blockade of the dopaminergic neuronal system (Goodman & Gilman, 1985). So repression is instituted, and unconscious sexual ideations curtailed from emergence into consciousness by the same means—dopaminergic modulatory alteration. So we can see, that to allow a sexual ideation up from under repression is a function, at least in part, of a relative increase in dopaminergic activity in the system (5-HT is involved with repression as well), as those with hallucinatory illness have just such an excessive dopaminergic balance. Schizophrenics display just this sort of dynamic, with heightened limbic activity, and reduced frontal activity, reduced repressive activity from the dorsolateral prefrontal cortex (Hobson, 2002).

So I believe that symptoms of Parkinson's and the syndrome in general, may be wholly or partially responsive to psychoanalytical intervention. Much of dopaminergic balance is subject to influence by purely mental factors, and may demonstrate no small measure of flexibility and adaptive dynamism to combat the onset of this condition, should the subject be willing to take an unvarnished and severe look into their own mental processes, and in so doing, liberate the unbound libido associated with the now properly allocated ascending activating system, and, limbic-orbitofrontal circuitry, and hence, the dopaminergic constituent, which is under the sway of repressive and analytic influence.

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References:

Freud, S. (1911-1913). The standard edition of the complete psychological works of Sigmund Freud volume twelve: Case history of Schreber, Papers on technique, and other works.

London: Hogarth Press.

Gazzaniga, M., et. al., (2009). *Cognitive neuroscience: The biology of the mind.*London: Norton Press.

Goodman, A., Gilman, L. (1985). The pharmacological basis of therapeutics. New York, NY.: Macmillan Press.

Hobson, J. A., & Pace-Schott, E. F. (2002).

The cognitive neuroscience of sleep: Neuronal systems, consciousness and learning. *Nature Reviews Neuroscience*, (3). doi:10.1038/nrn915

Kaplan-Solms, K., & Solms, M. (2002).

Clinical studies in neuropsychoanalysis: Introduction to a depth neuropsychology.

London: Karnac Press.

Norman, R. (2011). The tangible self. O'Brien, OR.: Standing Dead Publications.

Norman, R. (2013). Who Fired Prometheus? The historical genesis and ontology of super-ego and the castration complex: The destructuralization and repair of modern personality—An essay in five parts. The Black Watch: The Journal of Unconscious Psychology and Self-Psychoanalysis. Retrieved from: www.thejournalofunconsciouspsychology.com

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