

## “An essay on the shaking palsy” 200 years old

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If James Parkinson could attend one of the Parkinson congresses this year, he would certainly be surprised to see how far-reaching his small publication had become. Quite possibly, he would also be disappointed in discovering how very much we still adhere to his case descriptions and how very little we have learned over these past 200 years. He described patients with movement disorders and never asserted the claim that he had discovered a new nosological entity. Charcot was the first to coin the term “Maladie de Parkinson” and later generations worked out the official definition as a disease.

And thus, we find ourselves inadvertently caught up in an induction-deduction loop: Parkinson described a small number of patients with similar symptoms, and then after that, a disease was defined deductively. And now, finally, we draw conclusions from theory and classify individual cases inductively, which, in turn, results in a tendency towards self-confirmation of that very same theory, that is, the disease so defined, and in the long run, any awareness for a crisis in basic fundamentals has been completely lacking.

We could pose the hypothetical question as to what would have happened if instead of the “Essay on the shaking palsy” Shy-Drager Syndrome or Richardson’s Syndrome had first been published. Most probably, these diseases would not have been classified as atypical

Parkinson syndromes, but rather as independent diseases. The question is completely justified as to why CBD, PSP, and MSA are viewed as atypical Parkinson syndromes, although tauopathies only have but a limited common intersection with the synucleinopathies. This may well be due to the fact that these disease occur so infrequently.

In addition, this thought brings us to the problem of definitive classification. Formulating a hypothesis of course presupposes that the conditions be defined, but the hypothesis itself is an assumption and thus requires inductive verification. The mere fact that a hypothesis has not been falsified in no way supports its validity. And so, our insistence that bradykinesia and rigor and/or tremor define a Parkinson syndrome fits the process of formulating an hypothesis, but does not confirm the accuracy of that hypothesis nor any conclusions based on it. Even when we would like to see its accuracy be confirmed, the results of pathology and imagery techniques do not prove the diagnoses, since these are based on the hypotheses. They merely reflect the current status of our knowledge.

One additional problem arises, because we tend to think consecutively. When, for example, a hypothesis has been formulated, findings are usually arranged to fit accordingly, as long as they are not contradictory, they do not change the original assumption. James Parkinson described not only motor symptoms in his patients, but non-motor ones as well. However, for a long time, the motor signs alone were considered as key symptoms, and only in recent years, do we routinely give the non-motor dimensions their due recognition. We now have sufficient data for developing hypotheses which would require a new definition of the diagnostic criteria. However, because the motor symptoms were at the focus of both diagnostic and therapeutic attention for such a long time, the non-motor ones

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are viewed as secondary, having less relevance at all or at best only for the further course of the disease.

A good number of our well-accepted and established “facts” are not in fact congruent and some of our central findings contradict others. It might be of considerable use to us to simply disassemble our standard picture of the disease into its different puzzle pieces and see which pieces do really fit the other ones. Up to now, we have arranged these pieces, so that they fit our hypothetical total picture. Doing this, we might just be doing more justice to the observations made by James Parkinson, thereby getting closer to a solution in the long run. We continue to term the Parkinson’s disease “idiopathic”,

meaning that research, to date, has yet to discover the basic cause of the disease.

Since 2000, we have been organizing an annual Expert Meeting Parkinson where we discuss the current status of our work and any open questions. The results of our workshop are always published timely and so you will find the texts of our last meeting in this issue of the journal. At the recent meeting, we worked intensively on epidemiology, the mechanism of pathogenesis, but also on differential diagnosis and new definitions of the disease. Hereby, it is becoming increasingly clear that our previous approach did not adequately address the challenges, because Parkinson’s is rather a syndrome than a disease.