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GUEST EDITORIAL

Disrupted identities: movement, mind, and memory in Parkinson's disease

For the many clinicians *au fait* with the history of the clinical description of Parkinson's disease, they will be aware that the very earliest description of James Parkinson's "Shaking Palsy" in 1817 explicitly excluded the involvement of cognitive and emotional processes as manifestations of the disease. Within a short time following his treatise, it became all too clear to those in the field that Parkinson's disease is more than just a motor disorder, and as was aptly conceptualized by Paul McHugh, Professor of Psychiatry at Johns Hopkins Hospital from 1975 until 2001, Parkinson's disease is closer to being a "triadic disorder," encompassing motor, cognitive, and psychiatric elements (McHugh, 1989). Even this notion is now outdated, with the triad being accompanied by autonomic, pain, and other non-motor syndromes.

In the late 1980s, the initial focus of these non-motor cognitive psychiatric syndromes was predominantly on depression and psychosis. Parkinson's disease was considered an elegant *in vivo* model of affective disorders based on the localization theory of disruption of the orbitofrontal-basal ganglia–thalamic circuit (Mayberg and Solomon, 1995). This was followed in the late 1990s by a seminal randomized clinical trial (RCT), which led to clozapine being the first antipsychotic licensed to treat psychosis in Parkinson's disease (The Parkinson's Study Group, 1999). Unfortunately, due to the complexities of prescribing clozapine outside of psychiatric settings, the use of this highly effective drug has not been fully exploited in the management of Parkinson-related psychosis. More recently, another atypical antipsychotic, pimavanserin, a selective serotonin 5-HT2A inverse agonist, has also been shown to be effective in reducing symptoms of Parkinson's disease (PD)-related hallucinations and delusions (Cummings *et al.*, 2014).

In 2000, the neuropsychiatric emphasis in Parkinson's disease started to shift more to cognitive impairment and dementia associated with the condition, with the increasing recognition that Parkinson's disease is accompanied by a nearly six times greater incidence of dementia, particularly in those with a later age of onset, the akinetic rigid form of the disease, and older age (Goetz *et al.*, 2008). The field took a significant step forward with two further landmark papers, namely the publication of the clinical diagnostic criteria for dementia associated with Parkinson's disease ("PDD"; Emre *et al.*, 2007) and the successful EXPRESS (Exelon in Parkinson's Disease Dementia Study) trial of rivastigmine in PDD (Emre *et al.*, 2004). The latter paved the way for the first (and to date, only) clinically licensed medication for treatment of the clinical symptoms of PDD. This was followed by a further key development in the form of what some in the field considered the belated consensus diagnostic criteria for mild cognitive impairment (MCI) in PD (PD-MCI or MCI-PD) (Litvan *et al.*, 2012).

These consensus-derived clinical diagnostic criteria for PDD and PD-MCI have added substantially to the creation of a common language to discuss "mind and memory" in relation to Parkinson's disease and have no doubt contributed to the burgeoning literature on Parkinson-related cognitive impairment. But for a long time, the Parkinson's community resisted this label of "PD-MCI", mostly because the evidence needed to give meaningfulness to the construct was lacking. Limited longitudinal data was available to enable us to understand the prognostic implications of such as a diagnosis or clinical categorization; and labels are not benign, labels have meaning. A label can define one as good or bad. As per Richard Feynman's oft-cited observation, knowing the name of a bird in all languages of the world will ultimately tell you nothing about the bird itself. To wit, there is a clear distinction between "knowing the name of something and knowing something." This is where the precariousness of diagnostic labels in medicine comes to the fore. The sociological construct of "labeling" has been used to enlighten medical practice since the 1960s and draws attention to the view that the experience of "being sick" has both physical as well as social consequences (Scheff, 1974).

The peril inherent in the liberal use of diagnostic labels was made evident to me shortly after

I was asked to write this editorial. I had a meeting with two "patient and public voice" (PPV) representatives who have been advisors on our study of a new psychosocial therapy for PDD (The INVEST study; McCormick *et al.*, 2016). I asked for their thoughts about how I should construct this paper. With little hesitation, both individuals clearly expressed their views. The first person, living with Parkinson's, requested that my message should portray the importance of the person as separate from the diagnosis, particularly in the case of impending dementia. The second person, his spouse, wanted me to emphasize that a partner's role in the journey with Parkinson's should not be regarded automatically by professionals as that of a "carer" but, rather, as a spouse, a partner, or perhaps, a support person. These comments underscore the crucial issue of *identity* and how health and disease can impact identity. This is even more so with a complex, insidious process such as Parkinson's, which impacts on mind and memory, as well as the outwardly obvious physical aspects of a movement disorder.

Appositely, this year's BBC Reith lectures, stylishly delivered by the philosopher Kwame Anthony Appia on the topic of "identity," expounded on the theme of people having multiple identities in a poly-identifying world. Key amongst these identities, according to Professor Appia, are those of religion, race and nation, all of which can merge and interact to declare both the public and private portrayals of who we are. As clinicians, we should add another identity to this list – identity manifested through health or loss of health. At various times, one domain of identity can subsume or even dominate other aspects of identity. This is particularly the case with a chronic and multi-faceted condition such as Parkinson's, which affects both body and mind. For those living with Parkinson's, there is the ever-present risk of the illness becoming *the* identity. In the opening passages of Rohinton Mistry's latest novel, "Family Matters," the protagonist, Nariman, an elderly man with Parkinson's, is being subjected to the unwelcome ministrations of his step daughter's "caring": "*Instead, here she was, plaguing him with rules to govern every aspect of his shrunken life. Besides the prohibition against locked doors, he was required to announce his use of the WC.*" For clinicians, we too are prone to the trap of assuming the illness is the person and the label defines the identity. The professional urgency of categorization and intervention pushes one towards this dangerous detachment.

It is clear, therefore, that the time is now ripe to move beyond the labeling of the various clinical manifestations of this perplexing and all-encompassing disease of Parkinson's. While the syndrome labels have set the stage, the performance as it is now emerging needs to take us beyond the "naming of birds" and into a deeper understanding of the disease-based mechanism of Parkinson's and its impact on mind and memory. This effort is being led by such groups as the Oxford Parkinson's Disease Discovery Cohort, one of the largest and best-characterized cohorts of people with Parkinson's in the world. Currently, with over 1,500 recruits to the cohort, the team is investigating potential objective, validated biomarkers for Parkinson's disease that will form the basis of disease stratification leading to a highly-personalized approach to management, based on the specifics of the individual. This approach holds the promise of *preserving* identity for those affected in the way they choose. And, importantly, like the promise of the 1990s of Parkinson-related depression being the Rosetta stone for all affective disorders, a new disease-mechanism-based understanding of Parkinson's, PDD, and PD-MCI may open the door to an understanding of all neurodegenerative diseases.

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