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Cognitive impairments and depression in Parkinson's disease

Starkstein *et al*¹ present a follow up study of depression and cognitive impairment in Parkinson's disease (PD). Central to the concept of this paper is the meaning of the term "depression". The literature concerning affective disorder and PD uses the term inconsistently. Various, it has referred to a general clinical opinion of a morbid state; to a state diagnosed by the summation of symptoms and signs greater than a cut-off score on an ordinal rating scale; and to a clearly defined syndrome as described in DSM III "Major Depression". The latter usage is preferable. "Major Depression" has been criticised because "many physically sick individuals could be included simply on account of their physical illness and without the necessity to postulate the presence of mental disorder".² Of the additional features (in addition to lowered mood) which are required to diagnose "Major Depression", most can occur solely as a result of PD. Dakof and Mendelsohn³ stated "For Parkinson patients, many of these symptoms are likely to be part of the primary pathology of parkinsonism and not an indication of depression. At present, there is no way to make a distinction".

Starkstein *et al* do not appear to have appreciated these difficulties. They used a very low cut-off (7 and above) on the HDRS, an ordinal rating scale. About half the items on the HDRS could be confused by the cross over of the features of affective disorder and PD. They validated this against DSM-III "Major Depression" which has problems as described above. Furthermore, the DSM III diagnosis was made by using the PSE which generates diagnoses from ICD-9 rather than DSM-III. This procedure should be validated itself before being used as a "gold standard" for validating the HDRS, as the two classificatory systems differ radically regarding depressive syndromes. Hence "depression" as diagnosed by Starkstein *et al* is unlikely to bear any resemblance to any condition diagnosed by psychiatrists. The finding of significantly more tremor, akinesia and rigidity in the depressed group is consistent with the notion that higher HDRS scores are associated with more severe PD, and do not necessarily reflect the presence of a depressive disorder. The low levels (in numbers and dosage) of treatment in the depressed group suggests that the overall degree of morbidity was low.

Further confusion arises from the method of use of MMSE scores. The authors use the MMSE score itself rather than the cut-off of 23 as stated. The use of mean values makes it difficult to determine the clinical significance of the changes reported because of the ceiling effect of its maximum value of thirty. The important information clinically is how many people become demented during follow up. In table four the large standard deviation for the last mean for MMSE in the depressed group suggests some subjects obtained very low scores accounting for the differences in the means. This use of parametric statistics for data which is non-parametric in nature is not appropriate.

These factors greatly reduce the value of the findings of this study. Unless the confusion surrounding the definition and diagnosis of affective disorder and cognitive impairment are resolved, it is unlikely that issues in this area will be clarified.

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Starkstein *et al* reply:

Madeley *et al* raise several points regarding our study. They make the important observation that major depression should be diagnosed based on diagnostic criteria, such as in DSM-III. We certainly agree with this observation, and we acknowledged in the paper the limitation of using a cut-off score on a depression scale. However, in a recent study in which we used DSM-III criteria, we found similar results, for example, patients with PD and major depression showed a significantly faster cognitive decline than patients with PD and no depression (Starkstein *et al*, unpublished).

The second issue raised by Madeley *et al* is also an important one. Whether depression can be reliably diagnosed in the presence of a neurological disorder has been recently examined by our group.^{1–2} For PD, we found we can diagnose depression using slightly modified DSM-III criteria for major depression with a sensitivity and specificity of 91% and 100% respectively. Thus we are confident that our diagnosis is not clouded by the presence of the extrapyramidal symptoms of PD.

Even after using non-parametric data, depressed patients showed a significantly faster cognitive decline. Eight of the 18 depressed patients (44%) had a follow up MMSE score in the abnormal range, compared with three of the 31 non-depressed patients (10%) ($\chi^2 = 7.90$, $df = 1$, $p < 0.005$).

We believe the low number of depressed patients with PD receiving treatment for depression is not the consequence of a low degree of morbidity, but the fact that depression may not be diagnosed unless a standardized psychiatric evaluation is used.

Finally, the finding of significantly more severe tremor, rigidity and akinesia in the depressed compared to the non-depressed group is the result of a significantly longer duration of illness. In support, when depressed and non-depressed patients were matched for duration of illness, no significant between-group differences in tremor, rigidity, and akinesia were observed (paired $t = 1.62$, 0.34 , 0.72 , respectively $p = NS$).

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Misconceptions and inappropriate use of terms in hyperthermic syndromes

Progress in understanding the pathophysiology of hyperthermic syndromes is hampered, as much of the literature on neuroleptic malignant syndrome (NMS) is polluted with inadequate terms and thermoregulatory misconceptions. A previously published paper on the subject is also open to criticism.¹

In the first place, the term fever is applied to describe the condition, in which a patient's body temperature is elevated. Second, fever or (hyper) pyrexia and hyperthermia are used as synonyms. Fever or pyrexia results from a hypothalamic controlled elevation of the temperature setpoint. Through coordinated physiological and behavioural responses the body temperature rises until the setpoint is reached.² Hyperthermia is defined as the elevation of body temperature above setpoint, occurring when heat-dissipating mechanisms are defective or insufficient in relation to the internal heat production or excessive environmental heat.² Therefore, elevated body temperatures in cases of NMS should be designated as "hyperthermia".

Also, the term "autonomic dysfunction" is used, describing the autonomic responses (tachycardia, diaphoresis, flushing, and tachypnoea), in patients with hyperthermia.¹ In view of thermoregulation these profound autonomic responses can only be considered adequate in response to the elevated body temperature.

Furthermore, disruption of dopaminergic thermoregulatory mechanisms in the hypothalamus is frequently implicated in the development of hyperthermia in NMS.¹ We think this hypothesis is not justified because in NMS hyperthermia is due to increased muscular heat production, secondary to increased rigidity with tonic contractions following dopaminergic-receptor blockade in the basal ganglia. This is supported by the beneficial effects of the directly acting muscle relaxant sodium dantrolene used in some of the cases with NMS.³

The elevated body temperature is associated with pronounced, and thus adequate (hypothalamic controlled) autonomic responses trying to cope with the heat excess. Concerning the clinical spectrum of hyperthermic syndromes, neuroleptic malignant syndrome (NMS) is becoming a most inappropriate name, used in some cases. While the NMS may take a severe, potentially lethal course, the designation "malignant" hardly seems appropriate in the majority of the cases.^{4,5}

The occurrence of hyperthermic syndromes in Parkinsonian patients strongly resembles NMS. This signifies that impaired central dopaminergic activity in the basal ganglia is the hallmark of a continuum of hyperthermic syndromes, which should