2 Secondary prevention of vascular disease by prolonged antiplatelet treatment—Antiplatelet trialists' collaboration. *BMJ* 1988; 296:320–31.

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.

P MADELEY
CA BIGGINS
JL BOYD

RHS MINDHAM
Department of Psychiatry,
University of Leeds, Leeds, UK

- 1 Starkstein SE, Bolduc PL, Mayberg HS, Preziosi TJ, Robinson RG. Cognitive impairments and depression in Parkinson's disease: a follow up study. *J Neurol Neurosurg Psychiatry* 1990;53:597–602.
- 2 Snaith RP. The concepts of mild depression. *Br J Psychiat* 1987;150:387–93.
- 3 Dakof GA, Mendelsohn GA. Parkinson's disease: the psychological aspects of a chronic illness. *Psychol Bull* 1986;99:375–87.

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 $p = NS$).

SERGIO E STARKSTEIN
PAULA L BOLDUC
HELEN S MAYBERG
THOMAS J PREZIOSI
ROBERT G ROBINSON

Department of Psychiatry,
John Hopkins Hospital, Baltimore, MD, USA

- 1 Fedoroff JP, Lipsey JR, Starkstein SE, Forrester A, Price TR, Robinson RG. Phenomenological comparison of major depression following stroke, myocardial infarction and spinal cord lesions. *J Affect Dis* 1990 (in press).
- 2 Starkstein SE, Preziosi TJ, Forrester AW, Robinson RG. Specificity of affective and autonomic symptoms of depression in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1990;53:869–73.

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

therefore be designated as "hyperthermic syndromes with impaired dopaminergic activity".

BOB J VAN HILTEN
RAYMUND AC ROOS
Department of Neurology,
Leiden University Hospital,
2300 RC Leiden, The Netherlands

- 1 Srinivasan AV, Murugapappan M, Krishnamurthy SG, Zaheer Ahmed Sayeed. Neuroleptic malignant syndrome. *J Neurol Neurosurg Psychiatry* 1990;53:514-16.
- 2 Dinarello CA, Cannon JG, Wolff SM. New concepts on the pathogenesis of fever. *Rev Infect Dis* 1988;10:168-90.
- 3 Guzé BH, Baxter LR. Neuroleptic malignant syndrome. *N Engl J Med* 1985;313:163-6.
- 4 Levinson DF, Simpson GM. Neuroleptic-induced extrapyramidal symptoms with fever. *Arch Gen Psychiatry* 1986;43:839-48.
- 5 Clarke CE, Shand D, Yuill GM, Green MHP. Clinical spectrum of neuroleptic malignant syndrome. *Lancet* 1988;ii:969-70.
- 2 Singer C, Sanchez-Ramos J, Weiner WJ, Ackerman M. Sexual dysfunction in parkinsonian men. *Neurology* 1989;39 (Suppl 1):145.
- 3 Singer C, Sanchez-Ramos J, Weiner WJ. Autonomic dysfunction in male PD patients. *Neurology* 1989;39 (Suppl 1):142.
- 4 Singer C, Weiner WJ, Sanchez-Ramos JR, Ackerman M. Sexual dysfunction in men with Parkinson's disease. *J Neurol Rehab* 1990 (in press).
- 5 Singer C, Weiner WJ, Ackerman M, Sanchez-Ramos J. Sexual dysfunction in early Parkinson's disease. *Neurology* 1990;40 (Suppl 1):221.

Extracting the umlaut

Omitting the umlaut from Sträussler¹ further encourages the common but incorrect pronunciation as Strow rather than the correct Stroy.

WB MATTHEWS
Sandford on Thames, Oxford

- 1 Hart J Jr, Gordon B. Early-onset dementia and extrapyramidal disease: clinicopathological variant of Berstmann-Sträussler-Sheinker or Alzheimer's disease? *J Neurol Neurosurg Psychiatry* 1990;53:932-4.

Sexual function in patients with Parkinson's disease

We read with interest the report by Brown *et al* on sexual function in patients with Parkinson's disease (PD) and their partners.¹ We have come to similar conclusions in our own work on the subject.²⁻⁴

Our study involved Parkinsonian men only (mean age 65.8) and compared them to a group of healthy elderly non-Parkinsonian men (mean age 70.4). Our finding of a prevalence of erectile dysfunction of 60.4% in the study group compared with 37.5% in the control group was significant and comparable to the figure of 60% by Brown *et al*. There were, however, a few differences. Our group was more than double the size, randomly selected and with an average age more representative of the Parkinsonian male population. Presence of dysautonomic symptoms, as also noted by Brown *et al*, length of levodopa therapy or age did not appear to be significant factors, since they were equally prevalent in dysfunctional and nondysfunctional patients. In our more recent report on a group of men in the early stages of PD,⁵ where the prevalence of erectile dysfunction was lower (31%), we did not find depression as playing any role. Poor marital adjustment by the patients' wives, on the other hand, was frequently found, in agreement with the report of increased spousal strain.¹

We think that PD represents a risk factor for development of erectile dysfunction. It is not clear to us whether the additional presence of other risk factors is required or whether PD alone can bring about the dysfunction. We think that severity of disease may play a role and we are not convinced depression may be important except in a minority of cases. We agree with Brown *et al* that all therapeutic modalities available to other couples should be offered to PD patients and their spouses. We would also advocate that such an offer be preceded by an equally thorough diagnostic evaluation looking for all known mechanisms of sexual dysfunction.

CARLOS SINGER
WILLIAM J WEINER
J SANCHEZ-RAMOS
MARK ACKERMAN*
Departments of Neurology and Psychology,*
University of Miami, School of Medicine,
Miami, Florida, USA

- 1 Brown RG, Jahanshahi M, Quinn N, Marsden CD. Sexual function in patients with Parkinson's disease and their partners. *J Neurol Neurosurg Psychiatry* 1990;53:480-6.

something in all circumstances: a symptom of the enthusiasm of the less experienced. The A to Z plan is at first sight user-friendly, but I found it irritating. "Acoustic nerve—see calorics, cranial nerves, hearing, vertigo; Meningioma—see computed tomography, tumour; Subarachnoid haemorrhage—see haemorrhage; Sydenham's chorea—see chorioathetosis" (sic).

The authors and editors have plainly laboured hard to distil so much practical information into so small a space. They are to be congratulated on the result which will be a popular and valuable aid to all juniors in the wards.

JMS PEARCE

Tumour Stereotaxis. By PATRICK J KELLY. (Pp 409 Illustrated; Price: £74.00.) 1991. London, W.B. Saunders Co. ISBN 0-7216-5360-X.

This is a major monograph by a single author who presents his personal experience of stereotactic neurosurgery for brain tumours in a lucid and authoritative way. He approaches the subject by way of a detailed historical introduction covering the personalities and stereotactic methods which have lead up to the development of modern stereotactic instruments. The author describes all the most commonly used stereotactic systems but devotes most space to the philosophy behind the design and implementation of his own system, that is the Kelly-Goerss or Compass Instrument.

Other necessary requirements for contemporary stereotaxy are described. Thus, one chapter is devoted to features of operating theatre design to accommodate stereotactic work efficiently and another to the integration of the computer as a neurosurgical instrument. The author, and his colleagues in medical physics and computing, were pioneers in the use of neuro-imaging to control volumetric excision of brain tumours; employing stereotactic systems which allowed interaction between the surgeon and the diagnostic brain images available in the scanner to take place in real time during the course of a craniotomy performed under stereotactic conditions. This book is the operating manual for this system. The theoretical technical limits of accuracy achievable are discussed in depth and the reader is made aware of how practical answers to many problems have been arrived at. The clinical application to tumour biopsy and excision is described with reference to the author's very large clinical series and detailed descriptions are also provided of stereotactic third ventriculostomy and of stereotactic interstitial and external beam radiotherapy together with radiosurgery. The author describes and evaluates classical non-stereotactic neurosurgical operative techniques and demonstrates the particular indications for which stereotactic methods represent improvement in accuracy and lessened morbidity. He also makes the point that stereotactic surgery can save money in health care. In the final section he reviews future trends including robotic methods and holographic imaging displays.

This monograph is a *tour de force* by a leading stereotactic neurosurgeon, and will rank alongside that small number of similarly distinguished publications, including some

All titles reviewed here are available from the BMJ Bookshop, PO Box 295, London WC1H 9TE. Prices include postage in the United Kingdom and for members of the British Forces Overseas, but overseas customers should add £2 per item for postage and packing. Payment can be made by cheque in sterling drawn on a United Kingdom bank, or by credit card (Mastercard, Visa or American Express) stating card number, expiry date, and your full name.

The Little Black Book of Neurology. A Manual for Neurologic House Officers 2nd Edition. Philadelphia, Mosby Year Book. Edited by J S BONNER AND J J BONNER. (Pp 435; Price: £11.50.) London, Wolfe Publishing 1991. ISBN 0 8151 2321 3.

This 2nd edition succeeds Stephen Thurston's successful 1987 edition, written by residents for residents, in a format fit for the pocket of the white coat. It is a mine of concise, useful information, presented in staccato style, but quite intelligibly. The 47 tables and 42 figures are exceptionally useful condensations of material which the resident will need but will be unable to find quickly elsewhere. The contents are more or less comprehensive and cover all the emergencies and most of the "cold cases" a resident is likely to see in the wards or emergency room. The style is necessarily didactic and the advice generally sound though some will take exception to the apparent compulsion to do

BOOK REVIEWS