

MATTERS ARISING

Prospective evaluation of a prognostic index for intrinsic supratentorial tumours

In their paper published in this *Journal* Hutton *et al* conclude that histological grade provided no additional information on survival of patients with intrinsic supratentorial tumours.¹ This is surprising and contrary to the findings of other workers.²⁻⁴ One of the problems is that they relied on the Kernohan grading system, which is notoriously subjective. Other groups have pointed out that histological grade using the Dumas-Duport scheme does contribute prognostic information. It would be interesting to know whether if the prognostic index used by the current authors was tested against the Dumas-Duport scheme, histology would still fail to contribute to the prognostic index.

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- Hutton JL, Smith DF, Chadwick DW. Prospective evaluation of a prognostic index for intrinsic supratentorial tumours. *J Neurol Neurosurg Psychiatry* 1995;59:92-4.
- Burger PC, Scheithauer BW. *Tumours of the central nervous system. Atlas of tumor pathology.* Washington, DC: Armed Forces Institute of Pathology, 1994.
- Dumas-Duport C, Scheithauer BW, O'Fallon I, Kelly P. Grading of astrocytomas, a simple and reproducible method. *Cancer* 1988;62:2152-65.
- Kim TS, Halliday AL, Hedley-Whyte ET, Convery K. Correlates of survival and the Dumas-Duport grading system for astrocytomas. *J Neurosurg* 1991;74:27-37.

Hutton *et al* reply:

That histological grade provided no additional information on survival once a binary variable derived from our index was taken into account is a statement of fact. As Dr Hedley-Whyte is surprised by this fact, we suggest that she uses the index, given in the paper, on data which she has collected. It will be interesting to know whether the Dumas-Duport scheme contributes prognostic information which adds to data that can be obtained by non-invasive methods.

Hemiballism in Parkinson's Disease

We read with interest the recent interchange between Obeso *et al* and Inzelberg and Korczyn in the *Journal* (1995;58:645-6). Their discussion, and particularly the accompanying diagrams, outline current wisdom with respect to basal ganglia interactions and how changes in the activity of specific structures account for certain clinical manifestations. One critical clinical observation which is not readily explained by the

current physiological models of the basal ganglia is the striking amelioration of levodopa-induced dyskinesias with ventral posterior medial pallidotomy. The benefit obtained from this procedure with respect to primary parkinsonian signs is generally explained on the basis of a reduction in the overactivity of the GABAergic inhibitory input from the pars interna (GPi) to the thalamus (fig 2B, p 646). However, given the extent of the expected pallidal lesioning with this procedure, a pronounced reduction would have been expected in all pallidal outflow resulting in a similar picture to that depicted in fig 2F (similar to an extensive lesion in the subthalamic nucleus (STN) causing dyskinesia). Instead, in our experience, mild, shortlived (< one hour) contralateral (and occasional ipsilateral) dyskinesias occur at the time of lesioning in most patients followed by almost complete elimination of all forms of levodopa-induced dyskinesias (peak dose dyskinesias, diphasic dyskinesias, and off period dystonia) in the contralateral limbs with additional improvements often seen in ipsilateral limbs as well. If the lesion is misplaced or partial (possibly comparable with the effects proposed in fig 2E with a partial STN lesion), the ameliorative effects of the pallidotomy on levodopa induced dyskinesias may be only shortlived.

There are other important neuroanatomical problems with the simplified schematic diagrams currently used to explain these clinical states. Figure 2 outlines the "indirect pathway" exclusively. However, as Parent and Hazrati^{1,2} have recently pointed out, there are major anatomical flaws in this simplification. These authors argue that the projection from the external segment of the globus pallidus (GPe) to the STN does not directly synapse with neurons projecting on to the GPi and substantia nigra (SN) pars reticulata. Instead, they connect with neurons which project back to the GPe forming a closed loop. The GPe then has a direct connection to the GPi which is all but ignored in current anatomical formulations. Another important criticism of current models emphasised by Parent and Hazrati^{1,2} is the fact that the STN input to the GPi projects predominantly to neurons involved in associative rather than sensorimotor striatal inputs.

It is clear that there is a great deal more to be learned about the interactions of corticobasal ganglia-thalamocortical interactions in health and disease states. Although current models have successfully predicted some clinical findings, they fall well short of satisfactorily explaining many others. Careful electrophysiological, biochemical, and imaging assessments of non-human primates and patients undergoing surgical treatments for Parkinson's disease will hopefully assist in clarifying some of these confounding issues.

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- Parent A, Hazrati L-N. Functional anatomy of the basal ganglia. I. The cortico-basal ganglia-thalamo-cortical loop. *Brain Research Reviews* 1995;20:91-127.
- Parent A, Hazrati L-N. Functional anatomy of the basal ganglia. II. The place of subthalamic nucleus and external pallidum in basal ganglia circuitry. *Brain Research Reviews* 1995;20:128-54.

Inzelberg and Korczyn reply:

We are grateful to Lang and Lozano for their comments. They are correct in pointing to some deficiencies in the basal ganglia circuitry which we have suggested in this *Journal* (1995;58:645-6). Neither have we intended the diagrams to be all inclusive. Intentionally we have omitted the pedunclopontine nucleus, cerebellar contributions, etc, have not differentiated between partial and complete lesions, and have not distinguished between the immediate effects of the lesions and subsequent compensation, to which we refer elsewhere.¹ We hope, nevertheless, that the simplified diagram suggested by us will be of value to clinicians and scientists interested in movement disorders as it "explains" several neurological conditions.

1 Flash T, Inzelberg R, Korczyn AD. *Behav Brain Sci* 1995 (submitted).

NOTICE

Announcement from the British Neuropsychiatry Association: 1996 Summer meeting

The 1996 Summer meeting will be held on 14-16 July at Robinson College, Cambridge. It will include topics on neurodevelopment, language, and the presentation of short scientific papers and single case videos by members. The Association's AGM will be held on 16 July.

For further details of these meetings please contact: Sue Garratt, Administrative Assistant, BNPA, 17 Clocktower Mews, London N1 7BB. Telephone/Fax: 0171 226 5949.

For details of membership of the BNPA, which is open to medical practitioners in psychiatry, neurology, and related clinical neurosciences, please contact: Dr Jonathan Bird, Secretary BNPA, Burden Neurological Hospital, Stoke Lane, Stapleton, Bristol, BS16 1QT. Telephone: 01179 701212 ext 2925/2929 or Sue Garratt at the address given above.

CORRECTION

Cockerell OC, Eckle I, Goodridge DMG, Sander JWA, Shorron SD. Epilepsy in a population of 6000 re-examined: secular trends in first attendance rates, prevalence, and prognosis. *J Neurol Neurosurg Psychiatry* 1995;58:570-60. During production an error occurred in table 2. The third subheading in this table should read *Age and sex specific prevalence rates for 1000 persons for 1983 and 1993*. Not per 100 as printed.