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Book reviews

Stereotaxis in Parkinson Syndrome By R Hassler, F Mundinger, and T Riechert (pp 315; DM 280, $156.80) Heidelberg: Springer Verlag, 1979.

This is a beautifully produced volume by three acknowledged experts in the field of stereotaxic procedures for Parkinsonism. A series of clear, coloured diagrams plainly depicts their concept of the neuronal pathways involved for the development of the clinical syndrome as well as for the placement of lesions for the relief of rigidity and tremor. The authors report on nearly 3500 operations with the low mortality rate of 0·8%. From post-mortem material their stereotaxic method was seen to have achieved a high degree of accuracy. Electrical stimulation was used for a physiological check for the position of the lesion to be made, using a bi-polar electrode with a gap of 5 mm which, of course, would cover a relatively large area of the thalamus. Little mention has been made of the electrical recording in the thalamus as pioneered by French and Canadian workers in this field; this method provides a unique opportunity for further research into the normal thalamus and regrettably has been lost.

There is an atlas made from sections at 2 mm intervals in the frontal plane through a normal thalamus of a sufferer from Parkinsonism, with photographs of fibre and Nissl preparations superimposed on a grid. It is gratifying to see that the FM–PC plane is used for the base line and reference points. Perhaps because of the small magnification of ×5, the rationale for some of the boundary lines of the named compartments is not always clear. There is thought to be no need for a variability study since the authors relate the target zones to the mid-point between the foramen of Munro and posterior commissure. This view does not accord with one detailed variability study of the thalamus where the anterior border of CeM, an easily recognised structure, has a scatter of 4 mm in the sagittal plane, and the spread is greater if the mid-thalamic plane is used (which is perhaps comparable to the middle of their "base line" of the thalamus) rather than the posterior-inferior margin of the foramen of Munro. In this atlas the CeM does not appear until 18 mm posterior to FM, which the reviewer considers to be 4 mm more posterior than usual.

Some surgeons will disagree that a pallido-thalamic interruption is the more desirable lesion for rigidity, and a more posterior dentatothalamic lesion for tremor. It is the experience of the reviewer that a 6 mm long coagulation, whose posterior limit extends into that zone where electrical responses are obtained from muscle stretching in the opposite upper limb, gives good relief from both tremor and rigidity, and it probably lies in what the authors call Vim and Vop.

These are perhaps minor criticisms of a most careful and comprehensive work which should be in the possession of all those engaged in this field.

JOHN ANDREW

Multiple Sclerosis in Childhood, Diagnosis and Prophylaxis By EJ Field (pp 111; $12.75) Springfield: Charles C Thomas, 1980.

Over the past decade Professor Field's work on immunological abnormalities in multiple sclerosis has aroused considerable interest. It is useful to have this work summarised in a single volume in which a variety of laboratory tests are described in considerable detail and claims made that they are of value in diagnosis of patients with the condition, as well as in detecting potential sufferers from multiple sclerosis. In essence, Professor Field thinks that patients who have clinical evidence of multiple sclerosis have an inherited defect of membrane metabolism—the membrane of oligodendrocytes possibly sharing this defect—which renders them susceptible to the disease. Furthermore, he believes that laboratory tests of changes in the electrophoretic mobility of lymphocytes, red blood cells and macrophages induced by two particular unsaturated fatty acids (linoleic and arachidonic acids) can differentiate not only patients with clinically definite multiple sclerosis, but will also detect relatives of such patients who have inherited the membrane defect necessary to acquire the disease. He further claims that these deviations from normal may be corrected by giving unsaturated fatty acid supplementation, and extrapolates from this into the area of prophylactic therapy, believing that correction of the defect in childhood will prevent the development of multiple sclerosis later.

Needless to say, there has been considerable debate about Professor Field's work both at the laboratory and clinical levels. The techniques involved in assessing electrophoretic mobility are fastidious in the extreme, a point emphasised by the author. The accuracy of timing of migration of the various cells has been questioned, and certainly the magnitude of alteration of mobility is near to the limits of visual timing. Perhaps more controversial, is the concept that there is an inherent defect in the cell membrane of patients and their relatives, and that this is the fundamental defect in multiple sclerosis, correction of which will prevent the disease. Professor Field has claimed that it is possible to use his laboratory methods for the early diagnosis of multiple sclerosis in children who have relatives with multiple sclerosis, and in patients with the first suspicious symptom or sign. The whole problem of whether the abnormality detected by Professor Field lies in defects of the membrane of the marker cell or the plasma, has been questioned, a point discussed but rejected by the author. Even if this is accepted, many neurologists would be doubtful of Field's claim that prolonged administration of gamma linoleate to children at risk will later prevent them developing clinical signs of the disorder. The statement by the author that gamma linoleate is beneficial to patients with established multiple sclerosis is crucial to his hypothesis, but without convincing foundation. He wrongly quotes the only double blind trial of the use of this substance in multiple sclerosis (Bates *et al* 1978[1]) which showed that, if anything, gamma linoleate was harmful. This is a poor rationale for giving such a substance to children who by a series of laboratory tests are thought to be at risk of developing multiple sclerosis. Furthermore, the author glosses over the problem of giving prophylactic therapy to children, particularly the profound psychological difficulties that can arise.