increasing distance from the mid line.² The electrode tip in this case is at coordinates (F 7.7, H 0.0, L 6.8 mm). The bare component of the unipolar electrode extends 3 mm rostral to the tip at a 45 degree angle. In the sagittal plane the variability is approximately 2 mm. In this case therefore the possible structures which may be involved are the centromedian nucleus and parafascicular nucleus of the thalamus, the prerubral field of the mid brain, the medial part of the VPM nucleus of the thalamus, the habenulo-interpeduncular tract, the field H of Forel and the rostral components of the brainstem reticular formation.

Electropathological discharges and localisation: The EEG discharges observed in this case and used for final electrode placement were characteristic of discharges observed in other patients being similarly treated for chronic pain syndromes. The discharges have included an area in the rostral mesencephalon in contiguity with the posteromedial thalamus in which the centromedian nucleus was most frequently implicated. Since the discharges are generated by cellular elements and not by fibre tracts, and since attenuation of the discharges is accompanied by improvement without sensory and motor impairments, it was theorised that the observed effects were due to implication of neither the fibre tracts nor thalamic sensory-motor nuclei.

Clinical reports without histological control: It is unfortunate that precise anatomical localisation of an electrode cannot be given in the present case, nor in any other clinical reports which by necessity are dependant upon a stereotactic atlas of the human brain.¹ The most that one can expect in a brief case report, is to diagrammatically illustrate the anatomic site of localisation and to identify the site by the structure or anatomic system which most likely accounts for the results. In our report, it is expected that the readers will refer to the diagrammatic insert and realise, as did Mr Goadsby, that in all probability the centromedian nucleus is not involved to the exclusion of other structures.

The same reporting methodology was apparently used, in the rat studies quoted by Mr Goadsby, on cerebrovascular changes elicited by electrical stimulation of the centromedian-parafascicular complex in the rat. A non critical reader may think that the (C-M) complex is exclusively responsible for the findings. Although the electrodes are in the (C-M) Complex, the tractus retroflexus of Meynert may also be implicated for the following reason: histological diagrams of the electrode localisations reveal several points next to the tractus retroflexus of Meynert, which runs through the (C-P) complex. That tract, in part, conducts impulses from the anterior diencephalon and septal area, structures which are associated with BP elevations in response to high frequency discharges. Furthermore, after establishing threshold stimulation sites for BP elevations of ≤ 10 mm Hg and then stimulating at the same sites with parameters $3 \times$ threshold, makes one wonder whether those fibre tracts were also implicated at those relatively high levels of stimulation. In addition, 1.5 h after anaesthesia may be insufficient clearance time to obviate chloralose hyperexcitability effects.

Blood flow: The increased cerebral blood flow from stimulation of the centromedian-parafascicular complex observed, by Mraovitch and Seylaz (1987) and Mraovitch *et al* (1986) in the rat, is of interest in view of

our demonstrating increased thalamic blood flow without obvious cortical involvement. One is tempted to speculate that the underlying mechanism is the same in both instances despite the deficiencies in specific anatomical localisation. The evaluation of local blood volume or local glucose utilisation depends on the radiolabelled agent used in SPECT. Iodoamphetamine (IMP) used in this case primarily evaluated rCBF, but probably also indirectly reflected local cell functions or metabolic state.³

Blood pressure: Acute changes in BP, as reported in the rat, were not noted in this patient nor any other during stimulation in the general area of the centromedian nucleus, even at threshold stimulation for sensory-motor responses. Spontaneously and artificially induced after-discharges also were not accompanied by BP changes.

- 1 Schaltenbrand G, Bailey P. *Introduction to stereotactic atlas of the human brain*. Thieme: Stuttgart, 1959.
- 2 Andy OJ, Jurko MF, Sias FR. Subthalamotomy in treatment of Parkinsonian tremor. *J Neurosurg* 1963;20:860-70.
- 3 Lee RL, Hill TC, Hollman BL, Clouse ME. An isopropyl (1-123)p-Iodoamphetamine (IMP) Brain Scans with SPECT: Disordance with Transmission Computed Tomography. *Radiology* 1982;145:795-9.

BOOK REVIEWS

The Right Cerebral Hemisphere and Psychiatric Disorders. By JOHN CUTTING. (Pp 479; £45.00.) Oxford University Press. 1990. ISBN 0-19-261764-8

The fascination with the functional differences of the two cerebral hemispheres lives on. We have come a long way from the quasi-phenomenological theories that attempted to localise discrete functions to specific areas in the right or left hemisphere. More appealing views of the left hemisphere as an analyser and the right as a Gestalt processor have also been superseded by models based on the study of split brain patients and the use of dichotic listening and tachistoscopic techniques to direct information to either hemisphere. One such model put forward by Kosslyn considers that the left hemisphere analyses information along categorical lines, like a library of words or objects, whilst the right hemisphere is more like a guide-book that allows us to get information from the library.

Cutting has found inspiration in Kosslyn's views and his perambulations across the two hemispheres are done with his guide-book firmly in hand. The result of his effort is a unique book that provides a detailed and scholarly review of hemisphere function that will be impossible to find anywhere else. The book is divided into three sections. The first deals with the evidence of the differential functions of the two hemispheres and includes an excellent historical review. The

second deals with focal neuropsychiatric symptoms in the light of differential hemisphere function and includes a useful chapter on tests of hemisphere function, and the third explores the role of hemisphere differences in the causation of psychiatric disorders.

In my view the main strength of the book lies in its second section which skilfully explores the common ground between many psychiatric and neurological phenomena which, at times, have been artificially separated. Various disorders of awareness, language and thought and other symptoms such as delusions are dealt with here. New insights into phenomenology are abundant and I can easily envisage coming back to it in search for an explanation, when puzzled by clinical cases. The last section is perhaps best seen as food for thought and it is less likely to stand the test of time.

Cutting firmly believes that a hemisphere imbalance, with impaired functioning of the right hemisphere, is at the root of schizophrenia. The evidence for this, as Cutting himself points out, is far from conclusive and the recent imaging and neuropathological studies have failed to provide the desired proof. In fact, finding a coherent explanation to encompass the evidence implicating abnormalities in various cerebral sites in schizophrenia is one of the greatest challenges facing psychiatry; and hemisphere imbalance is unlikely to be a satisfactory explanation. The evidence is even less convincing for affective illness and autism. These problems do not detract from the interest of the book, but add to the hope that Cutting will again be tempted to write on the subject when, in a few years time, the biology of psychiatric illness will be better understood. All those interested in the complex relations between brain and mind should read this book.

MARIA A RON

Clinical Neurophysiology of the Vestibular System. 2nd Edition. Contemporary Neurology Series. By R W BALCH AND V HONRUBIA. (Pp 301; Price £38.79). Philadelphia, F. A. Davis Co., 1990. ISBN 0-8036-0584-6.

The first major investigations of clinical disorders of the vestibular system were carried out by Robert Barany in 1907. Since this time, and especially in the last 15 years a veritable deluge of tests has been applied to function and reflexes arising in the vestibular apparatus. Neuro-otology has evolved as a new specialty, but sadly the refinements of clinical diagnosis and treatment of the dizzy patient have lagged behind. Indeed only a handful of diagnoses are used by most audiologists and neurologists. The commonest are: acute vestibular neuronitis, benign positional vertigo, the clinically ill-defined and much overdiagnosed Menière's syndrome, and a variety of clinical assumptions in later life yielding labels such as cervical spondylosis and vertebrobasilar insufficiency often in tendentious fashion.

There is little doubt that the dizzy patient will at least receive a more accurate diagnosis if suitably investigated in a clinically directed neuro-otology laboratory, even if he or she emerges with specific drug or surgical therapies which are often disappointing.

The second edition of this book from UCLA appears some eleven years after the first. It is conventionally divided into three