opposed to the idea that "we know our limbs belong to us because they obey us", we know that our limbs belong to us because they provide us with sensory input that is recognised as self. Many patients with movement disorders or paralysis lose control of their limbs but still have no difficulty in realising them as self. Indeed even in "phantom limb" there is sense of self due to central processes in the absence of a limb. Our patient, as do others with anosognosia and primary abnormalities of central sensory systems, shows perhaps that it is central sensory processes that are the key to identifying "self". We know our limbs not because they obey us but because there is a pattern of sensory activation that accompanies our own limb movements. When this pattern never reaches specific cortical regions, then the limb is not perceived as self; called "amorphosynthesis" by Denny-Brown and Banker.¹ Self stimulation by the centrally deafferented limb in "sensory" or "posterior" alien hand syndrome, or kinaesthetic stimuli due to movement of the limb as in the "anterior" or "motor" alien hand syndrome, is perceived as due to another person or thing without critical questioning. This raises the most interesting question of what brain region is deafferented in the anterior alien hand syndrome where the sensory processing is intact.

It is not our clinical experience nor the conclusions based on published reports that all patients suffering with alien hand syndrome are terrified by the effected limb. In one author's experience (BHP), two patients with alien hand syndrome and related intermanual conflict were irritated by but not terrified by their opposing limbs simultaneously vying for a cigarette or book. Another patient was amused but rather indifferent to his affected left side. The most terrifying situation we have heard is when the patient identified his affected left side as belonging to his mother in law! A patient reported by Heilman's group² with persistent alien hand syndrome referred to it as "my little sister". Similar to our experience, they suggest that a predisposing personality type may be necessary given that most patients with colossal infarcts or tumours do not emphasise this complaint.

Unlike our case of limited duration, the persistence of alien hand syndrome seems dependent on mesial frontal dysfunction. These patients rarely deny that the affected limb belongs to them. Instead, they understand it in terms of their "anarchic hand". Hence, although the initial syndrome may result in disjointed and terrifying perceptions, it seems that the brain quickly re-establishes its control by presently unknown adaptive capacities. Furthermore, why it almost exclusively involves the left body side in right handed people remains unknown. Studying this syndrome in greater detail may yield additional insights into the pathophysiology of denial and misidentification.

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Vasomotor reactivity is exhausted in transient ischaemic attacks with limb shaking

The article of Baumgartner and Baumgartner entitled "Vasomotor reactivity is exhausted in transient ischaemic attacks with limb shaking"¹ provides interesting new information regarding the nature of involuntary limb movements contralateral to haemodynamic failure from severe carotid artery occlusive disease. The authors evoke an "exhausted cerebral vasoreactivity in the hemispheres opposite the involuntary limb movements". In their report, involuntary movements affected only the limbs, and displayed no tonic contraction, tonic-clonic jerking, or Jacksonian march and no epileptic activity during or between the attacks. These findings led the authors to strongly argue against seizures as the cause of limb shaking in these transient ischaemic events.

In contradistinction, a 72 year old right handed man was admitted to our hospital with a 3 month history of episodic weakness and numbness of the right arm. The patient then had six discrete stereotypic episodes of right arm weakness and clumsiness that were also associated with difficulty in speaking. Several episodes of dysarthria, numbness and weakness of the right arm and leg (MRC grade 4/5) were seen, unrelated to posture, some of which occurred when the patient was supine. Most episodes were characterised by slight tremulousness and asterixis-like movements of the outstretched right arm. There was a return to baseline functioning between events.² Video/EEG monitoring, however, showed low voltage spikes in the left central-parietal head regions contralateral to the facial twitching and the right arm and right leg weakness. Although ongoing clinical and EEG seizure activity stopped after 2 mg intravenous lorazepam, they reoccurred after loading with phenytoin. Because angiography disclosed a greater than 95% stenosis of the left internal carotid artery (while the patient was treated with phenytoin at a concentration of 16.5 mg/l), the patient was anticoagulated with heparin, but episodes continued. It was only after a left carotid endarterectomy that all episodes of weakness, tremulousness, and EEG epileptiform activity stopped. They have not recurred over the past 5 years.

The literature includes several cases of focal motor inhibitory seizures causing weakness.^{3,4} Although it is impossible to prove a negative, it could be argued that although no epileptiform or other evidence of seizure activity is present in a particular case, the abolition of ongoing clinical and EEG evidence of inhibitory motor activity by intravenous diazepam argues in favour, at least in part, of an ictal contribution. The fact that in virtually all reported cases, abnormal movements are more definitively resolved by carotid endarterectomy, argues for an underlying ischaemic aetiology that induces focal seizures. There are few reports that clearly delineate the interaction and association of inhibitory focal motor seizures and transient ischaemic attacks, as there are few sequential trials of antiseizure drugs or anticoagulation (under EEG monitoring) and finally carotid endarterectomy. Several authors support the concept of an inhibition of motor function in parietal and secondary somatosensory re-

gions by seizure activity which then interrupts the sensory feedback loop to motor integration with inhibition of subcortical and cortical areas.⁵

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- 1 Baumgartner RW, Baumgartner I. Vasomotor reactivity is exhausted in transient ischaemic attacks with limb shaking. *J Neurol Neurosurg Psychiatry* 1998;65:561-4.
- 2 Kaplan PW. Focal seizures resembling transient ischemic attacks due to subclinical ischemia. *Cerebrovasc Dis* 1993;3:241-3.
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- 4 Tinuper P, Aguglia U, Laudadio S, et al. Prolonged ictal paralysis: electroencephalographic confirmation of its epileptic nature. *Clin Electroenceph* 1987;18:12-14.
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Baumgartner and Baumgartner reply:

We are grateful for the response of Kaplan to our short report. We agree that somatic inhibitory seizures may mimic transient ischaemic attacks (TIAs). Such TIAs are associated with negative symptoms such as sensorimotor deficits and difficulty with speaking, EEG evidence of seizure activity, and ceasing of the TIAs after the administration of an anticonvulsant drug.^{1,2} Limb shaking TIAs, however, differ from TIAs related to inhibitory seizures in several ways. (1) They are associated with positive phenomena (limb shaking), and the involuntary movements do not affect the facial muscles. (2) Patients with attacks of shaking movements of the limbs have no EEG evidence of epileptic activity, and involuntary movements do not stop after administration of anticonvulsive therapy. (3) Although the patient presented by Kaplan had a 95% stenosis of the left internal carotid artery, it is unclear whether haemodynamic failure was present or not, because no studies evaluating the haemodynamic reserve of the homolateral hemisphere were presented. This is in accordance with the finding that the involuntary movements as well as the sensorimotor deficits of Kaplans' patient were not related to posture. (4) The pathogenesis is thought to be due to disinhibition of subcortical control mechanisms as a result of ischaemia.

In our opinion, it is not clear whether the asterixis-like movements of the outstretched right arm of Kaplan's patient are due to epileptic seizures, because unilateral asterixis of the outstretched arm has been reported with contralateral vascular lesions affecting almost all cerebral structures involved in motion control including ischaemia in the territory of the middle cerebral artery.³

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1 Kaplan PW. Focal seizures resembling transient ischemic attacks due to subclinical ischemia. *Cerebrovasc Dis* 1993;3:241-3.

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BOOK REVIEWS

Neurology for the MRCP Part II. By DAVID SMITH. (Pp164, £15.99). Published by Arnold, London 1998. ISBN 034069175.

To the MRCP candidate neurology is one of the more daunting specialties. The unfamiliar nerve conduction study and the frankly mysterious EEG can distress an otherwise well rounded senior house officer. Despite the fact that much of neurology is commonly seen on a general medical ward—strokes, dementias and so forth—the general perception is of an unimaginable list of eponymous syndromes and obscure signs. Rather than dwell on the last, in this book Dr Smith tries to address the commoner complaints as examination style questions each with a “simple clinical lesson”. The “grey case” section, for instance, includes questions on multiple sclerosis, cluster headache, and HSV encephalitis, while broadening the topics to include postinfective demyelination, chronic hemiparesis, and acute haemorrhagic encephalomyelitis. There is, however, a tendency for the discussion after each question to be rather brief. A fuller explanation, with more allowance for the reader's ignorance, would have been appreciated. The data interpretation section is somewhat better, covering CSF, EEG, and other data extremely well. Perhaps a little too well; would an MRCP candidate really be expected to recognise the characteristic EEG of Creutzfeldt-Jakob disease? I surely hope not. Finally, the slide tests are disappointing. If anything, neurology lends itself best to this section of the written examination but it is let down by the poor quality of some of the images in this book. This is especially unfortunate, as other images in the same section are remarkably impressive. The Sturge-Weber skull radiograph and central pontine myelinolysis MRI are beautiful. In summary, this is a creditable first edition. I look forward to the second.

STEFAN MARCINIAK

An Atlas of Multiple Sclerosis. Edited by Charles M Poser. (Pp 134, £48.00/\$85.00). Published by Parthenon Publishing, Carnforth 1998. ISBN 1-85070-946-7.

This book, after a short introduction to some of the fundamental features of the disease goes on to provide some 117 illustrations of aspects of the disease from Cruveilhier's plates to histopathological specimens and also a heavy leaning to imaging particularly magnetic resonance scanning, as might be expected. There is no doubting the aesthetic impact of this short book. In addition, the fact that these illustrations emanate from a well established figure in the multiple sclerosis world and are likely to be a representative set

of personal teaching slides from a successful academic career all vouch for the provenance and informative nature of the atlas. However the place of such a book within a neurologist's library has to be questioned. There are a plethora of high quality textbooks devoted to all aspects of multiple sclerosis, all well illustrated and most in colour. They provide in depth analysis of all aspects of the disease and although their illustrations tend to be smaller this is where I would choose to put my money. It may be that the circulation of this book will be higher than expected as it is likely to be a popular choice for some pharmaceutical companies.

NEIL ROBERTSON

Calvarial and Dural Reconstruction. Edited by SETTI R RENGACHARY and EDWARD C BENZEL. (Pp 200). Published by AANS, Illinois 1998. ISBN 1-879284-63-4.

This monograph is the latest to be produced by the American Association of Neurological Surgeons as part of its *Neurosurgical Topics* series. It begins by tracing the history of calvarial reconstruction from ancient times. There follows a discussion of the different autologous donor sites and synthetic materials currently available for repair of calvarial and facial defects. The merits, disadvantages, and contraindications of each are considered. Dural substitutes are then dealt with in similar fashion. Specific problems, such as scalp reconstruction, the management of comminuted frontal sinus fractures, and reconstruction of the anterior skull base are the subject of separate chapters. The final part of the book is devoted to craniostomies. A review of current knowledge on pathogenesis is followed by a good account of some of the more common techniques used to treat single suture synostosis. Understandably, in a book of this type there is space only for an overview of the treatment and complications of multiple suture involvement, but the chapter provides well chosen references for further reading.

The reconstruction of traumatic and post-surgical calvarial defects occupies the bulk of this volume, and is dealt with very effectively. Operative techniques and the relative merits of various materials are covered in a clear and concise manner. By contrast, the section on aural substitutes is a little disappointing because it does not provide the reader with reasoned argument on how to select the most appropriate graft from the sometimes bewildering variety of autologous, synthetic, and xenograft materials which are available when vascularised pericranial tissue is not an option.

Craniosynostosis is a topic which is covered very well in standard paediatric neurosurgical texts and it is not worth buying this book for that section alone. However, the account of techniques for repair of calvarial defects is excellent and merits the inclusion of this text in a departmental library.

ROBERT MACFARLANE

Echoenhancers and Transcranial Color Duplex Sonography. Edited by ULRICH BOGDANN, GEORG BECKER, and FELIX SCHLACHETZKI. (Pp 420). Published by Blackwell Science, Oxford 1998. ISBN 0-632-04856-5.

Transcranial colour duplex sonography is an ultrasound technique which is becoming

increasingly available for the non-invasive imaging of intracranial structures, particularly the basal cerebral arteries. There are now four principal components to the technique: B mode ultrasound which can be used to image the brain parenchyma; colour coded Doppler which provides a colour image of the basal vessels; spectral analysis of pulsed wave Doppler which is used to derive blood flow velocities; and latterly “power” Doppler which is also used for vascular imaging following analysis of the amplitude rather than the frequency of the reflected ultrasound beam. In addition, echocontrast agents are now available which can increase the signal to noise ratio and thus help overcome some of the detrimental acoustic effects of the skull.

This volume of 400 pages and liberal colour diagrams and prints is edited by three exponents of the technique. Thirty one chapters written by a further 20 contributors cover topics from the history of transcranial ultrasound, through the physics of Doppler ultrasound to potential clinical applications. The book is helpfully split into two sections with the theoretical aspects described in the first half and clinical aspects in the second.

This is certainly a specialised book and will really only appeal to those interested in, or wishing to develop, expertise in transcranial colour coded ultrasound. For such readers the technical chapters on instrumentation, signal processing, echocontrast agents, harmonic imaging etc will certainly provide a complete understanding of the principles behind the technique. I think that some of the errors made in the interpretation of vascular ultrasound examinations are due to an incomplete understanding of the physics of Doppler ultrasound, hence the attention paid to this area is commendable.

The clinical section covers the examination technique, normal reference values, the main categories of cerebrovascular disease, and also contains chapters on areas which may be less immediately suitable for ultrasound study. For example, the findings in head trauma, tumours, psychiatric disorders, and movement disorders are the subject of separate chapters. Although I have no problem with enthusiasm for this technique a little pragmatism would not go amiss. A more balanced discussion of the limitations as well as potentials of the technique could have been applied.

As with any book with multiple authors there is some variation in style and overlap, particularly in the introductions and conclusions of the chapters. Nevertheless, it is a comprehensive current review of transcranial colour coded sonography. Although the reader must decide exactly how this technique fits into clinical practice the book will certainly stimulate some ideas.

PETER MARTIN

Spinal Cord Diseases-Diagnosis and Treatment. Edited by GORDON L ENGLER, JONATHAN COLE, and W LOUIS MERTON. (Pp 675, US\$195). Published by Marcel Dekker, New York, 1998. ISBN 0-8247-9489-3.

This is volume 47 of a series entitled *Neurological Disease and Therapy*, series editor W C Koller. This volume is edited by an American surgeon and two British neurophysiologists. Most of the 45 contributors are American or British, almost half of whom, including Dr Cole, are from Southampton. The book begins with a pathophysiological