

NOTICE

British Neurosurgery Research Group Meeting

The fourth British Neurosurgery Research Group Meeting will be held in Newcastle on 20-21 March 1997.

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MATTERS ARISING

Impaired procedural learning after damage to the left supplementary motor area

Ackermann *et al*¹ have recently reported on procedural learning in a patient with a lesion of the left mesiofrontal cortex. The patient had no deficit when tracking a visual target by his right hand. However, the patient's performance was severely impaired in a mirrored tracking task. It was concluded that the finding confirms "previous PET results (the reader is referred to studies by Seitz *et al*² and Grafton *et al*³) indicating the supplementary motor area to be a part of circuits mediating visuomotor skill acquisition."

In the study of Seitz *et al*² subjects were taught a sequence of finger to digit oppositions before PET. PET was performed during initial learning of the sequence, advanced learning, and during skilled performance. They found activation of the supplementary motor area during initial learning as well as during skilled performance and no evidence for a specific involvement of the supplementary motor area in motor learning. Grafton *et al*³ employed a pursuit rotor task. Activation in the posterior part of the supplementary motor area was found to increase with the level of skilled performance. There was no indication of a role of the supplementary motor area in motor learning. Furthermore, the task of Grafton *et al*³ corresponded to the control task of Ackermann *et al*¹ in which the patient's performance was not impaired. The notion that these two PET studies indicate "the supplementary motor area to be part of circuits mediating visuomotor skill acquisition" (Ackermann *et al*¹) seems not to be clear.

There are studies on visuomotor learning to which Ackermann *et al*¹ do not refer. Lang *et al*⁴ have studied brain potentials when subjects learned to track a visual target in a mirror reversed way. Brain potentials were more negative in frontolateral and frontomesial recordings with the learning task than with a control task (non-inverse visual tracking). The functional relevance of this finding was shown by demonstrating intersubject correlations between additional frontal negativity and the success of motor learning. This result was confirmed in subsequent studies.^{5,6} The later study⁶ employed two independent tech-

niques of functional brain imaging, brain potentials and regional cerebral blood flow (rCBF) measurements by SPECT. With motor learning (compared with the control task) there was a significant increase of rCBF in the frontomesial cortex, the middle frontal gyrus (of either hemisphere), basal ganglia, and cerebellum. More recently, Kornhuber *et al*^{7,8} have studied visuomotor learning in 53 patients with chronic frontal lobe lesions. These patients performed a mirror reversed tracking task with non-inverted tracking as a control. Out of 53 patients 16 showed impaired motor learning. The critical region was in the transition zone between the supplementary motor area and the anterior portion of the cingulate gyrus of both hemispheres.

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- 1 Ackermann H, Daum I, Schugens MM, Grodd W. Impaired procedural learning after damage to the left supplementary motor area (SMA). *J Neurol Neurosurg Psychiatry* 1996; 60:94-7.
- 2 Seitz RJ, Roland PE, Bohm C, *et al*. Motor learning in man: a positron emission tomographic study. *Neuroreport* 1990;1:57-66.
- 3 Grafton ST, Mazziotta JC, Presty S, *et al*. Functional anatomy of human procedural learning determined with regional cerebral blood flow and PET. *J Neurosci* 1992;12: 2542-8.
- 4 Lang W, Lang M, Kornhuber A, *et al*. Human cerebral potentials and visuomotor learning. *Pflügers Arch* 1983;399:342-4.
- 5 Lang W, Lang M, Kornhuber A, Kornhuber HH. Electrophysiological evidence for right frontal lobe dominance in spatial visuomotor learning. *Arch Ital Biol* 1986;124:1-13.
- 6 Lang W, Lang M, Podreka I, *et al*. DC-potential shifts and regional cerebral blood flow reveal frontal cortex involvement in human visuomotor learning. *Exp Brain Res* 1988;71: 353-64.
- 7 Kornhuber AW, Lang W, Becker M, *et al*. Topography of critical regions for unilateral and bimanual motor learning within the frontal lobe of man. *J Neurol* 1994;241:258.
- 8 Kornhuber AW, Lang W, Becker M, *et al*. Unimanual motor learning impaired by frontomedial and insular lesions in man. *J Neurol* 1995;242:586-78.

Ackermann *et al* reply:

With regard to the comments by Lang and Kornhuber on our paper¹:

(1) Both PET studies referred to in our paper report activation of the supplementary motor area during acquisition of a new motor task (early motor learning). The fact that there was also supplementary motor area activation during "skilled performance" does not argue against a role for the supplementary motor area in motor learning. Even if the new movement sequence could be executed reasonably well at the end of the testing session, there may still be room for further improvement and therefore for further learning while subjects execute the task. For example, in motor conditioning subjects may reach an asymptote during a one hour learning session, but still show further learning in subsequent training sessions after intervals of hours or days.

(2) We are well aware that motor learning has been studied extensively using electrophysiological as well as SPECT technology, and we agree that the papers by Lang and Kornhuber are important in this regard. However, an exhaustive review of this literature is clearly beyond the scope of a short report, and therefore we concentrated on representative studies based on the latest technology—that is, PET.

- 1 Ackermann H, Daum I, Schugens MM, Grodd W. Impaired procedural learning after damage to the left supplementary motor area (SMA). *J Neurol Neurosurg Psychiatry* 1996; 61:94-7.

The pathway possibly responsible for the occurrence of isolated lateropulsion of the trunk

Bertholon *et al* recently described a patient with a lesion in the medial part of the cerebellar peduncle presenting with isolated body lateropulsion to the side of the lesion.¹ I had previously reported a case with a similar clinical picture and reviewed the medical literature.² The lesion in my patient involved the white matter in the inferior portion of the ipsilateral cerebellar hemisphere, possibly in the territory of the lateral branch of the posterior inferior cerebellar artery (IPICA). Spinocerebellar, cuneocerebellar, reticulocerebellar, and olivocerebellar fibres run in the inferior cerebellar peduncle. The topographical localisation of each fibre system within the inferior cerebellar peduncle has not been fully clarified.³ I assume that the fibres in the lesions of both the patient of Bertholon *et al* and my patient may be the fibres that mediate the same function in balancing, stance, and gait, and the destination of these fibres may be the neurons located in the paravermian region of the inferior cerebellar hemisphere.

Lesions involving other parts of the cerebellum or brainstem may produce signs of lateropulsion; however, they are usually associated with additional neurological deficits. Amarenco *et al* reported several patients with lesions in the medial branch of the PICA (mPICA); most of the patients with lateropulsion were associated with vertigo.⁴ However, in their patients, lateropulsion of the trunk persisted longer than other signs and eventually became "isolated". Together with the findings in my patient, this suggests that fibres mediating balance may pass through the territory of the mPICA and reach the territory of the IPICA. Separation between the fibres mediating balance and the more medially placed fibres mediating vestibular function should occur so that an "isolated" neurological deficit becomes possible. Indeed, in the cat, primary vestibular fibres end mainly in the flocculus and nodulus, whereas fibres in the dorsal spinocerebellar tract end more laterally, in the intermediate zone.^{5,6} The assumption that selective lesion of the dorsal spinocerebellar tract produces signs of "isolated lateropulsion of the trunk" in humans awaits proof.

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- 1 Bertholon P, Michel D, Convers P, Antoine JC. Isolated body lateropulsion caused by a lesion of the cerebellar peduncles. *J Neurol Neurosurg Psychiatry* 1996;60:356-7.
- 2 Shan DE, Wang V, Chen JT. Isolated lateropulsion of the trunk in cerebellar infarct. *Clin Neurol Neurosurg* 1995;97:195-8.
- 3 Iwata M, Hirano A. Localization of olivo-

- cerebellar fibers in inferior cerebellar peduncle in man. *J Neurol Sci* 1978;38:327-35.
- 4 Amarenco P, Rouillet E, Hommel M, Chaine P, Marteau R. Infarction in the territory of the medial branch of the posterior inferior cerebellar artery. *J Neurol Neurosurg Psychiatry* 1990;53:731-5.
 - 5 Brodal A, Hoivik B. Site and mode of termination of primary vestibulocerebellar fibers in the cat. An experimental study with silver impregnation methods. *Arch Ital Biol* 1964; 102:1-21.
 - 6 Grant G. Spinal course and somatotopically localized termination of the spinocerebellar tracts. An experimental study in the cat. *Acta Physiol Scand* 1962;56(suppl 193):1-45.

BOOK REVIEWS

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.

Neurosonology. Edited by CHARLES H TEGELER, VIKEN L BABIDIAN and CAMILO R GOMEZ. (Pp 513.) Published by Mosby-Year Book Inc, St Louis 1996. ISBN 0-8151-8792-0.

This multiauthor book is divided into eight sections. The basic principles of ultrasound are covered in the initial section. These are easy to follow even by those who have long since forgotten whatever physics they ever knew!

The second section considers the value of ultrasound in the diagnosis of adult extracranial arterial disease. It contains many practical points and considers the various pitfalls which may trap the unwary if a comprehensive ultrasound assessment is not performed. My only criticism is the lack of detail relating systolic and diastolic velocities with different degrees of stenosis. These are crucial measurements when assessing the severity of any stenosis, particularly in view of the need to know if a stenosis is more or less than 70%. This section covers only one tenth of the whole book which is surprising given this is the primary use of adult neurosonology; maybe the reviewer is biased!

The middle sections of the book review adult transcranial Doppler (TCD) and embolus detection with Doppler ultrasonography; this takes up almost a third of the book which seems a little excessive given their present clinical value. There is then a short section on echocardiography followed by a long section on paediatric neurosonology which covers a quarter of the book. The penultimate section discusses intraoperative and perioperative sonography; finally there are chapters on neurosonology in clinical practice and most importantly an appendix which discusses professional training guidelines, quality assurance, and accreditation in vascular ultrasound departments. These later issues are of the utmost importance: it is interesting to see how some countries

encourage or even insist on the certification of both individuals and laboratories before they are allowed to submit reports. The more widespread application of such standards should be supported.

This book is comprehensive and well written although at times a little uncritical; unfortunately, because it is a multiauthor text, several topics are covered more than once (for example, vasospasm, brain death). The overall balance of the book needs to be reviewed. Few clinicians, radiologists' or technicians would be interested in the book as a whole. The assessment of adult extracranial arterial disease, TCD, embolus detection, echocardiography, paediatric neurosonology, and operative examinations will be covered by a variety of different hospital departments in the UK. However, this book serves as a useful reference to the full range of conditions where neurosonology may have a role.

PETER HUMPHREY

Neurological Surgery. Fourth Edition. 5 Volume Set. Editor in Chief JULIAN R YOUMANS. (£585-00 HB.) Published by W B Saunders Co, London 1995. ISBN (5 Volume Set) 0-7216-5141-0.

This is one of the best known and used reference works in neurological surgery. It has been a pleasure to review and although I have not read each volume from cover to cover, I have used the text for advice and a second opinion on cases I have dealt with over the past few months. It has also been used by trainees in the department whose opinions have been equally complimentary. The book is amply illustrated with radiological images which have been well chosen and are reproduced with great clarity. The histological photomicrographs are less clear, mainly because they are in black and white and their relevance outside a pathological textbook is questionable.

Despite the pace at which new information is becoming available there is still an important place in neurological surgery for a large reference book that gathers together the views and experience of some of the most experienced practitioners in the field. All textbooks of this type run the risk of becoming obsolete before they are published and although there is much that is changing in neurological surgery there is also a great deal of background information and shared experience that will remain as the foundations of neurosurgical practice for many years. Whilst keeping an open mind and preparedness to change we should also be wary of too readily forgetting the lessons learned by those who have pioneered much that we now take for granted. We should learn by other people's mistakes and take care not to reinvent too many wheels. Out of approximately 340 contributors 12 are from Europe (two from the UK), seven from other parts of the world, and the rest are from the USA and Canada and so this textbook has a strong North American bias. This reflects the reality of contemporary neurosurgery where much of the innovative work is carried out in the United States. This arises because plentiful resources and superspecialisation allows for the concentration of rare and uncommon conditions in major centres which gives unrivalled opportunities for audit, research and development.

The sections of the book I have read in detail contain information which is for the most part up to date and relevant. Many of the authors have opinions which correspond to my own. Whether this reflects on the adequacy of training in the UK or is merely evidence of a closed mind is debatable. At £580 this reference work is remarkably good value, especially when compared with the price of other textbooks in specialised fields. Not only should all neurosurgical departments have a copy for their trainees but I suspect many consultants will wish to have a copy for their own personal use.

RODNEY LAING

Clinical Diagnosis and Management of Alzheimer's Disease. By SERGE GAUTHIER. (Pp 384; £69.95.) Published by Martin Dunitz Publishers, London 1996. ISBN 1-85317-308-8.

With an increasingly aged population, the social and economic impact of Alzheimer's disease on society becomes more apparent. This multiauthor one-volume text aims to provide an overview of recent scientific and clinical advances in our knowledge of this condition, although focusing on clinical diagnosis and management. The largely north American authorship comprises geriatricians, neurologists, psychiatrists, and psychologists.

It should be remembered that a diagnosis of Alzheimer's disease Alzheimer's disease can only be made histopathologically. By contrast, most in vivo studies are of dementia of Alzheimer type (DAT), which is a clinical diagnosis made on the basis of diagnostic criteria. These criteria are discussed in the introductory section, as are the pathological features.

Next, there is a detailed section on diagnosis. The chapter on clinical features is excellent, as is the one on clinical pointers to non-Alzheimer's disease forms of dementia, although the latter is limited in length. Investigations are then discussed. While formal neuropsychology and structural imaging are clinically indicated, functional imaging and electrophysiology are currently of research interest.

The natural evolution section discusses prognostic factors and how to stage the illness. The typical patterns of deterioration of cognition, mood, and behaviour and functional autonomy are also covered.

Psychopharmacology of Alzheimer's disease is covered in four chapters, ranging from antipsychotics, agents aiming at improving cognition, to those which attempt to halt or slow the disease. There are also sections on community and institutional management, and ethical and legal issues.

This text is a comprehensive one-volume account of Alzheimer's disease. Despite being multiauthor and multidisciplinary, it is well edited and has a uniformity of approach. It is broad in its scope, and each chapter is extensively referenced. Given that it is a textbook on Alzheimer's disease rather than the dementias, the brevity of the chapter on non-Alzheimer's disease dementias cannot be taken as a fault.

It would be wrong to criticise a book for not being something it did not set out to be. However, clinicians may wonder whether a textbook covering all the dementias, such as *Dementia* by Burns and Levy, might not be more appropriate to their needs.

JOHN GREENE

therapy, thus drawing together a literature spread through a wide range of journals, usually unfamiliar to the neurologists who are none the less asked to give clinical opinions.

This book is easy to use for reference. The drawings, photographs, and tables are excellent. Only a relatively short and predominantly north American bibliography is provided for each chapter and there are very few references cluttering up the text. The text is clearly written, albeit in a somewhat utilitarian style. Engaging prose can be found in Shuftell's chapter on "Headache", a topic which must be the forte of any ambulatory neurologist. He makes a cost-free therapeutic suggestion of dazzling attractiveness in recording how Dr John Graham commented to a school teacher with chronic daily headache and migraines "what a shame that your migraines have deprived your students of such a wonderful and dedicated teacher". I am not so keen to try his other suggestion of asking such a patient to describe their typical day from morning to evening. On page 307 we read that "two patterns of [amphetamine] abuse exist. Truck drivers, housewives and students employ oral preparations; . . .". Fellow Updike readers will be surprised to find later in the same chapter that American housewives are not singled out as particularly likely to abuse alcohol.

I found this a useful book to keep on my desk and look up issues which had arisen on ward rounds or in the clinic. It achieves its purpose with distinction.

MICHAEL DONAGHY

Cellular and Molecular Mechanisms Underlying Higher Neural Functions. Edited by A I SELVERSTON and P ASCHER. (Pp 328; £65.00). 1994. Published by John Wiley and Sons, Chichester. ISBN 0-471-94304-5.

This volume is one in a series of reports from workshops designed to promote interdisciplinary and international exchange of scientific ideas. Delegates are established scientists with international reputations, and this particular volume is the result of a meeting on the cellular and molecular mechanisms which may underly higher neural functions. As the editors point out, neuroscience has progressed over the past 10 years on many fronts, and at a number of levels from molecular and cellular, through systems, to behavioural. This workshop set out with the ambitious aim of trying to bridge gaps by taking a number of important scientific findings at one level and attempting to relate them to understanding at a different level. In practical terms, this was done by four separate groups which aimed to examine the following: the interface of behavioural plasticity and cellular processes, how neural circuitry can be affected by neuromodulators, how cellular mechanisms can affect long term potentiation, and the molecular mechanisms which may be involved in plasticity. The discussion was centred around a small number of key papers presented by delegates, following which each group produced a report based on a number of well-defined principles, outlining outstanding questions, and indicating future directions. For example, the first group's discussion was centred round examples of behavioural plasticity for which claims have been made of a direct relation to certain cellular processes studied *in vitro*, such as the effects of long term depression on cerebellar learning. They set out to examine whether current models are satisfactory for studying neural plasticity, whether better models might exist, and whether some mechanisms for learning are being overlooked completely.

One of the ultimate interests in studying molecular and cellular mechanisms in neuroscience is to try to understand how these processes come together to produce neural function—that is, behaviour of various sorts. For this reason, there is much of interest in

this for neuroscientists and neurologists alike. In addition, the attempt to make sense of current data derived from different disciplines is an exercise which is rarely performed in other settings, such as journals or textbooks. Unlike many volumes originating from conferences, this is not simply a collection of recent advances, but has drawn on classic background research to extract important principles about the way in which the brain may function. As such it is of wider appeal and should have a considerably longer shelf life.

ANNE ROSSER

SHORT NOTICES

Readers may be interested in

Reflex Sympathetic Dystrophy: A Reappraisal Edited by W Jaing and M Stanton-Hicks. (Pp 249; \$55.00). Published by IASP Press, Seattle. 1996. ISBN 0-931092-13-2.

Physicians' Guide to the Internet Edited by Lee Hancock. (Pp 240; £23.00). Published by Lippincott-Raven Publishers, Philadelphia. 1995. ISBN 0-397-51634-7.

Diagnosis and Treatment of Pain of Vertebral Origin Edited by Robert Maigne, (Pp 550; £85.00). Published by Williams and Wilkins, Baltimore. 1996. ISBN 0-683-05376-0.

Meningitis: 100 Maxims Edited by Karen L Roos. (Pp 208 £27.50). Published by Arnold, London. 1996. ISBN 0 304 60879 X.