Correspondence to: Professor P N Leigh
n.leigh@iop.kcl.ac.uk

- 1 Shaw CE, Enayat ZE, Powell J, et al. Familial amyotrophic lateral sclerosis: molecular pathology of a patient with a SOD 1 mutation. *Neurology* 1997;49:1612-16.
- 2 Ray Chaudhuri K, Leigh PN. Genetics of motor neuron disorders. *Advances in Clinical Neurosciences* 1994;4:267-6.
- 3 Chen R, Grand'Maison F, Strong MJ, et al. Motor neuron disease presenting as acute respiratory failure: a clinical and pathological study. *J Neurol Neurosurg Psychiatry* 1996;60:455-8.
- 4 Hu MTM, Ellis CM, Al-Chalabi A, et al. Flail arm syndrome: a distinctive variant of amyotrophic lateral sclerosis. *J Neurol Neurosurg Psychiatry* 1998;65:950-1.

Winer et al reply:

We are pleased that Leigh and Shaw found our report of interest and that the evidence that the patient had from giant cell arteritis is incontrovertible.

We also agree that the clinical features were very suggestive of motor neuron disease, which was in fact the antemortem clinical diagnosis. The only disagreement is whether the patient had two diseases or just giant cell arteritis.

Histological studies of the postmortem material, however, showed evidence of arteritis in the exact distribution of the clinical deficit—that is, the cervical roots associated with diaphragmatic function which we found convincing as the sole explanation for the clinical picture.

We agree that it is not possible to exclude the rare possibility of both motor neuron disease and arteritis and will undertake the suggested staining of sections of the brain stem, spinal cord and motor cortex with ubiquitin.

JOHN WINER
ED BURTON
PETER BARBER

University Department of Clinical Neurology,
Queen Elizabeth Hospital, Edgbaston, Birmingham
B15 2TH, UK

Correspondence to: Dr J Winer

Unidirectional dyslexia in a polyglot

We read with interest the short report by Leker and Biran,¹ describing unidirectional dyslexia in a polyglot, and would like to offer alternative hypotheses for the symptomatology.

The case describes alexia while reading Hebrew, but not when reading English. This is a recognised neuro-ophthalmic phenomenon, which we have encountered in our practice in patients reading the Urdu language, another script written right to left.

The extent of reading difficulties in patients with hemianopia is well described.² Patients with a right hemianopia cannot see letters following those already deciphered. Patients with a left hemianopia can read without difficulty, until they have to refixate the beginning of the next line which is in the hemianopic field, and their place in the text is then lost. It would be interesting to know if this was the case with this patient while reading English. Turning the page through 90° allows reading in the intact hemifield, and is standard advice to all patients with hemianopia.² Also by turning a page through 180° the Hebrew text would have been readable and comprehensible, as this would allow horizontal reading in a left to right direction.

The process of reading consists of repetitive small saccadic eye movements alternating with periods of fixation. Semantic identifica-

tion of word strings occurs during these pauses between the saccades. At the end of a line, a large saccade in the opposite direction, to the beginning of the next line occurs and the pattern of small saccades is repeated.³

Horizontal saccadic eye movements for reading are generated to peripheral visual stimuli (letters following those already interpreted). If the stimulus is in the hemianopic field, there is a primary inability to initiate the saccade (saccades can be generated without a target, but tend to be inaccurate). Also, the parietal eye fields are involved in horizontal saccadic eye movements, and may be implicated in the reported case given the anatomical location of the haematoma. Parietal eye field neurons discharge while executing visually guided saccades, and during learned saccadic tasks, which may be important during reading,⁴ and parietal lesions are known to cause saccadic defects. Vertical saccades are mediated via a separate system.

In true alexia, reading error results from an inability to interpret written material. The anatomical basis of alexia without agraphia is well described. This usually occurs in association with a right hemianopia, colour anomia, cerebral dyschromatopsia, and prosopagnosia, and is seen in left occipital lesions with damage to the contralateral visual association fibres which cross in the splenium of the corpus callosum.⁵ Reading pathways are well defined from visual cortex to auditory cortex via the dominant left angular gyrus, and such lesions have essentially isolated the angular gyrus from visual input. We think that the reported case describes "pseudoalexia" or "hemianopic dyslexia",¹ where a fixation splitting hemianopia causes difficulty reading by obscuring parts of words.

Visual hallucinations are common in patients with hemianopias despite being rarely mentioned,⁵ and in this case represent a release phenomenon, as ictal activity and pharmacological causes were excluded. They are a prognostically favourable sign.² Palinopsia is also seen in hemianopic fields. It would be interesting to know what palinoptic imagery was seen in this case, and under what circumstances. Was it perseveration of the hallucinations, or "blindsight" imagery of objects in motion being detected in the blind hemifield (the Riddoch phenomenon).

The "fluttering" described in the intact visual field of the reported case may reflect the "bilateral effect" seen from unilateral occipital lesions, which affect the primary visual and extrastriate cortex. Such damage is thought to alter interhemispheric connections along their presplenial course and cause disturbance of visual attention.² The effects are usually subtle relative to the hemianopia but often functionally significant.

We think that all the symptoms in the reported case can be accounted for within the realms of established knowledge, without postulating the existence of novel language associated neuronal networks in the right hemisphere.

MOIN D MOHAMED
SAMER M ELSHERBINY

Department of Ophthalmology, Bradford Royal
Infirmary, Duckworth Lane, Bradford BD9 6RJ, UK

PETER J GOULDING

Department of Neurology, St James's University
Hospital, Beckett Street, Leeds, LS9 7TF, UK

Correspondence to: Dr Moin D Mohamed

- 1 Leker RR, Biran I. Unidirectional dyslexia in a polyglot. *J Neurol Neurosurg Psychiatry* 1999;66:517-19.

- 2 Miller NR, Newman NJ. Topical diagnosis of lesions of the optic radiation. In: Miller NR, Newman NJ, eds. *Walsh and Hoyt's clinical neuro-ophthalmology*. Baltimore: Williams and Wilkins 1998:371-5.
- 3 Sharpe JA. Neural control of ocular motor systems. In: Miller NR, Newman NJ, eds. *Walsh and Hoyt's clinical neuro-ophthalmology*. Baltimore: Williams and Wilkins 1998:1107-19.
- 4 Feldon SE, Burde RM. The ocular motor system. In: Hart WM, ed. *Adler's physiology of the eye*. St Louis: Mosby, 1992:176.
- 5 Lessell S, Lessell IM, Glaser JS. Topical diagnosis-retrochiasm visual pathways and higher cortical function. In: *Duane's clinical ophthalmology*. Philadelphia: Harper and Row, 7:17-19.

The authors reply:

We thank Mohamed, Elsherbiny, and Goulding for their insightful comments regarding our paper.¹ We also think that the essence of this case is the distinction between a peripheral and a central processing defect causing the lexical phenomenon encountered by our patient. Mohamed et al suggest saccadic dysfunction as a mechanism for explaining the unidirectional dyslexia in our patient. However, whereas such a mechanism can explain right to left dyslexia when reading sentences, our patient had the same language abnormalities when reading isolated words when such saccadic mechanisms are not as important. We agree with Mohamed et al that visual hallucinations in the hemianopic field represent a favourable sign as indeed exemplified by the favourable outcome in our patient. Furthermore, we concede that the fluttering motion probably contributed to our patient's lexical difficulties. As pointed out by Mohamed et al such motion results from alterations in interhemispheric (occipital) connections.² This favours a more central processing abnormality. Moreover, the persistence of the unidirectional dyslexia while the visual fields returned to normal, as well as the dissociation between reading Yiddish (written in Hebrew letters from right to left), which was preserved, to reading Hebrew which was abnormal both support a higher hierarchical disorder.

Last but not least, the role of the right hemisphere in language functions as suggested by our case is supported by recent observations on aphasia recovery after stroke.³

R R LEKER
I BIRAN

Department of Neurology, Hadassah University
Hospital, Ein Kerem, P.O. Box 12000, Jerusalem
91120, Israel

Correspondence to: Dr R R Leker
leker@cc.huji.ac.il

- 1 Leker RR, Biran I. Unidirectional dyslexia in a polyglot. *J Neurol Neurosurg Psychiatry* 1999;66:517-19.
- 2 Miller NR, Newman NJ. Topical diagnosis of lesions of the optic radiation. In: Miller NR, Newman NJ, eds. *Walsh and Hoyt's clinical neuro-ophthalmology*. Baltimore: Williams and Wilkins 1998:371-5.
- 3 Ola AS. Recovery from aphasia: activating the "right" hemisphere. *Ann Neurol* 1999;45:419-20.

Dyslexia in Hebrew

This letter concerns the very interesting short report about unidirectional dyslexia in a polyglot by Leker and Biran.¹ We wonder if the authors would offer an explanation for the fact that at a time the patient still had "minor deficits when reading Hebrew" (page 518) he made no mistakes when reading Yiddish?

The languages are different to be sure, but the script and the right to left reading are nearly the same. Does this support the hypothesis that "the flaw lies within the central multimodal, word processing neuronal network"? The ability to read Hebrew vertically would still be unexplained.

Did the authors examine mirror reading in English and Hebrew? If so, what were the results?

MONROE COLE
ALAN J LERNER

Department of Neurology, University Hospitals of
Cleveland, 11100 Euclid Avenue, Cleveland, OHIO
44106, USA

1 RR Leker, I Biran. Unidirectional dyslexia in a polyglot. *J Neurol Neurosurg Psychiatry* 1999;66:517-19.

The authors reply:

We thank Cole and Lerner for their comments. We think that a higher hierarchical damage is supported by the discrepancy between the relatively preserved reading ability of our patient in Yiddish compared with Hebrew.

This discrepancy favours the existence of separate neuronal networks activated when reading in different language paradigms. The preserved vertical reading is suggestive of either a coexistent lower hierarchical damage or of a different pathway connecting primary receptive neurons with associative cortices when interpreting vertically presented information. We agree with Cole and Lerner that testing mirror reading might have yielded interesting information, but unfortunately we did not examine that at the time.

R R LEKER
I BIRAN

Hadassah University Medical Center, Jerusalem, Israel

CORRECTION

Khudados E, Cody FWJ, O'Boyle D. Proprioceptive regulation of voluntary ankle movements, demonstrated using muscle vibration, is impaired by Parkinson's disease.

J Neurol Neurosurg Psychiatry 1999;67:504-10. In this paper the address of Etedal Khudados should read Department of Neurological Surgery, Manchester Royal Infirmary, Manchester M13 9WL, UK.

BOOK REVIEWS

MRI of the Brain. Normal Anatomy and Normal Variants. Edited by PATEL and FRIEDMAN. (Pp 480, £61.00) Published by W B Saunders Co, London, 1997. ISBN 0-7216-6945-X.

This book presents the normal anatomy of the brain as seen on MRI studies in a very didactic, well presented, and novel manner. Images are well chosen, and are of high quality. The authors also attempt to relate some of the anatomical structures to their functions and dam-

age of relevant areas to disease processes. The book reflects the hard work and dedication of the authors in pursuing a good radiological-anatomical correlation, something which although being crucial for neuroradiology, is sometimes forgotten or taken for granted!

The book is divided in 19 chapters, 18 of them covering different anatomical regions of the brain. The last chapter presents some anatomical variants, as well as pitfalls on MRI, which should not be confounded with real lesions. In general, the book will be useful for the training neuroradiologist, and also for all of us dealing with neuroradiology and MRI and having to exercise our anatomical knowledge on a daily basis! Although the authors base the book's structure on classic anatomy, sometimes putting too much emphasis on anatomical classifications that may not be too useful for neuroradiologists nowadays, it results quite enjoyable, easy to read, and a useful teaching tool to have.

Those chapters dealing with the vascular anatomy on MRA are specially useful, in my opinion, since MRA is playing an increasingly more important role in neuroradiology, replacing conventional angiography for many clinical indications.

BEATRIZ GOMEZ-ANSON

Neurosurgical Treatment of Movement Disorders. Edited by ISABELLE M GERMANO. (Pp 276). Published by AANS, Illinois, 1998. ISBN 1-879284-58-8.

This volume is one in the series *Neurosurgical Topics* published by the American Association of Neurological Surgeons. It provides an excellent introduction to what is a fast moving and increasingly important area of clinical neuroscience.

The book deals with neurosurgery for excessive or insufficient movement, mainly in the context of Parkinson's disease but also covering hemifacial spasm, torticollis, and dystonia. It is divided into five sections. The first is an overview of classification, clinical features, neuropathology, and neuroimaging. The second describes in detail indications, techniques, and results of ablative surgery while the third covers restorative procedures by neurostimulation or by neural grafting. The last two sections cover ongoing technical developments in surgery for Parkinson's disease, and surgery for hemifacial spasm, spasmodic torticollis, and dystonia. Nearly 40 contributors have contributed a total of 19 chapters, all of which are clearly written and well referenced. Two of the authors appear under the misapprehension that Parkinson wrote in 1917 rather than 1817. However, this publication provides a comprehensive and very practical guide not only for neurosurgeons but also for neurologists and other clinical neuroscientists.

DAVID THOMAS

Transcranial Doppler Ultrasonography. Edited by VIKEN L BABIKIAN and LAWRENCE R WECHSNER. (Pp 409, £100.00). Published by Butterworth Heinemann, Oxford, 1999. ISBN0 7506 9969 8.

In the 17 years since Aaslid's first description of transcranial Doppler ultrasonography (TCD) the technique has developed from a research tool to a routine clinical investigation, at least in some circumstances. This transition has been especially rapid in the 6 years since

the first edition of this book was published in 1993. This second edition has grown in volume by 180 pages and now comprises 24 chapters organised into sections covering basic techniques and physics of Doppler ultrasound, cerebrovascular physiology, evaluation of cerebrovascular disease, monitoring for microemboli, intraoperative and intensive care monitoring, transcranial duplex imaging, paediatric applications, and the role of contrast media. To some extent the first edition of this book seemed to play second fiddle to the volume of Newell and Aaslid—*Transcranial Doppler*. Now however, with the introduction of several new chapter authors, this book is likely to occupy the prime place on the shelf of any clinician or technologist with an interest in transcranial ultrasound.

Any book written by 42 authors is bound to have some overlap in content but the current edition is well edited and the illustrations (many in colour) are of high quality. Each chapter is comprehensively referenced. Particularly welcome to this edition is the chapter by David Evans, Professor of Medical Physics at Leicester University. The ability, or otherwise, of TCD devices to count embolic phenomena accurately and to distinguish particulate from gaseous emboli has been a point of some contention amongst workers in this field. Thus it is with some relief that here we have a physicist of some authority in the field of Doppler ultrasound trying to ensure that clinicians understand the limitations of the technique before making potentially inaccurate assumptions.

Statements regarding the value of TCD monitoring during carotid endarterectomy are now all the more concrete given the work which has been published during the mid-1990s regarding this application, but much uncertainty remains over the exact clinical utility of TCD in other settings—for example, monitoring "vasospasm" or the patient with head injury. The relevant chapters provide a balanced summary of the available evidence. The section on colour coded transcranial imaging has enlarged significantly since the first edition and further technology such as power Doppler, three dimensional reconstruction, and echo contrast agents are comprehensively discussed. These technologies are rather at the stage at which TCD found itself a decade ago—that is, promising techniques awaiting a role. Quite possibly by the time the third edition of this book arrives in a few years time their clinical relevance will have been delineated.

Overall this is a welcome summary of the TCD literature. I would not rush to replace my first edition of the book with this edition as much of what is contained in the first is not made redundant by the later edition. However, for those clinicians and scientists seeking an overview of TCD and its current status this text will suffice very well.

PETER MARTIN

Russell and Rubinstein's Pathology of Tumors of the Nervous System. Sixth Edition. Two volume set. Edited by DARELL D BIGNER, ROGER E MCLENDON, and JANET M BRUNER. (vol 1 pp 615, vol 2 pp 757, £265.00 the set (HB)). Published by Arnold, London 1998. ISBN 0-340-58113-1.

The sixth edition of Russell and Rubinstein's *Pathology of Tumours of The Nervous System* has appeared in 1998, 39 years after the first