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The authors reply:

We write in response to the letter of Meador. We concur that his review article discusses both positive and negative psychotropic effects of antiepileptic medication.

Meador comments on the biased nature of our patient sample. However, we think that we adequately emphasised that our study was not randomised or controlled but carried out in a clinical environment. We pointed out that some of the patients had been referred due to cognitive complaints. Furthermore we acknowledged studies which reported no cognitive effects of the drug including the valproate and topiramate study and drew attention to the conflicting findings. We agree that prospective randomised controlled trials are the way to minimise selection bias. However, they are not the be all and end all. Bone marrow aplasia with felbamate and visual field constriction with vigabatrin treatment were not found in randomised controlled trials but by the careful clinical study of patients. This is an analogous situation. Randomised controlled trials are not without their own biases as most will be sponsored by the pharmaceutical industry and it would be naïve to conclude that this does not influence the presentation of the results.

We agree with Meador that the adverse effects of topiramate reported are likely to occur in a minority of patients treated with the drug. However, we think that this may represent a clinically significant number of patients, particularly in those attending tertiary referral centres. Negative effects, however small the numbers, are worthy of reporting and of further exploration. The question our findings raise is not does topiramate have adverse effects but rather why does it have adverse effects in some people?

We agree with Meador's final point and indeed this was one of the intended take home messages of our paper. This is why we chose to submit to a journal with a broad readership who would have much less experience with topiramate. We hoped that our paper would draw attention to a group of patients who should be prioritised for neuropsychological monitoring and highlight the type of measures that could be employed showing that an extensive assessment is not necessary.

We, however, do not think that the cognitive changes experienced would be obviously attributed to topiramate treatment. Most patients in the study were not referred because of cognitive complaints. Six were being seen as part of their presurgical assessment and indeed were not considered good candidates due to their neuropsychological test profiles. For some the cognitive complaints did not occur in association with the introduction of topiramate or with any change in dosage and did not seem to develop until they had been on the drug for several months. For such patients, particularly those with left hemispheric pathology, increases in word finding problems and other

verbal difficulties are more likely to be attributed to the underlying pathology and ongoing seizures than to a drug effect. Topiramate is a useful antiepileptic drug but it may lead to adverse cognitive changes and we need to be alert to this.

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Postoperative hearing loss due to venous congestion at the inferior colliculus, or cochlear dysfunction?

I read with interest the article by Strauss *et al* on postoperative contralateral hearing loss which developed on the third day after microvascular decompression for trigeminal neuralgia. They attributed the symptoms to venous congestion at the ipsilateral inferior colliculus after dissection of the pontotrigeminal vein, which was documented by MRI. Symptoms resolved partially after intravenous rheologic medication for a total of 19 days. The authors' explanation for the delayed postoperative hypacusis, however, merits further discussion. Strauss *et al* provided preoperative and postoperative recordings of brain stem auditory evoked potentials: postoperatively, after stimulation on the operated side, ipsilateral waves I through V, and contralateral waves II through V are all clearly identifiable, contrasting with stimulation on the non-operated side, depicting only a small wave V bilaterally, but no other components. This pattern suggests a left sided lesion involving the generator of wave I—presumably the auditory nerve near the cochlea—and is also consistent with the patient's pancochlear hearing loss.²⁻⁴ By contrast, brain stem lesions—unless damaging the cochlear nucleus—are usually not associated with pure tone hearing loss, but rather with abnormal auditory localisation or interaural time discrimination,^{2,5} as auditory impulses are conveyed bilaterally in the brain stem.^{2-4,6} Furthermore, a brain stem lesion causing profound hearing loss is likely to produce also contralateral wave IV/V abnormalities after stimulation on the non-affected side, but even the severest brain stem lesions, such as in evolving brain death, do not affect wave I.¹ The vascular supply of the mesencephalic brain stem differs from that of the inner ear, the first being fed by mesencephalic arteries via the posterior cerebral or superior cerebellar artery, and drained through the superior petrosal vein; the second being supplied by the more caudally originating labyrinthine artery via anterior—or occasionally posterior—inferior cerebellar artery, and drained by the labyrinthine vein through the posterior part of the superior petrosal or transverse sinus, and the internal jugular vein.⁶ Although the patient's hearing may have been partially affected by the documented mesencephalic lesion, hearing loss may in fact be more likely caused by concomitant cochlear dysfunction. An ischaemic lesion seems probable, presumably postoperative vasospasm, or—less likely—unrelated embolism. In either situation, rheologic treatment may have been benefi-

cial, as well as in venous congestion of the inferior colliculus. Concomitant cochlear dysfunction should have been considered as a cause of hearing loss in this patient, or ruled out by further examination.

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Strauss replies:

We appreciate Kofler's comment on our paper and his interest in this unusual and still poorly understood clinical picture. We agree that the brain stem auditory potentials (BAEPs) after contralateral stimulation do not clearly point to a lesion of the ipsilateral colliculus; however, to our knowledge the neurophysiology of auditory pathways within the brain stem is not yet fully understood. For example, in our series of more than 300 cases of acoustic neurinoma monitoring we have made the observation that the contralateral wave V is much more pronounced compared with the ipsilateral wave V. The advantage of this case report is the preoperative and postoperative radiological and clinical documentation. The delayed onset of symptoms several days after the surgical procedure, the lack of effect of calcium blocker therapy—actually the patient's pure tone audiogram and speech discrimination deteriorated under nimodipine treatment—and the hearing improvement after heparinisation do not suggest vasospasm as the underlying pathophysiological mechanism. The literature on this rare yet important phenomenon of contralateral hearing loss after cerebellopontine angle surgery is purely speculative. By contrast this case report follows a straight course, which started at surgery with dissection of the pontotrigeminal vein, followed by a delayed contralateral hearing loss, and ended with a lesion of the ipsilateral colliculus. This lesion was not documented on preoperative MRI. Taking these findings into consideration, together with the neurophysiological findings of BAEPs in a still not fully understood auditory pathway within the brain stem, the "isolated vasospasm theory" seems unlikely.

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BOOK REVIEWS

Behaviour and mood disorders in focal brain lesions. Edited by JULIEN BOGOUSLAVSKY and JEFFREY L. CUMMINGS. (Pp554, £49.95). Published by Cambridge University Press, Cambridge, 2000. ISBN 0-521-77482-9.

This book has arisen because of the comparative dearth of studies examining the emotional and behavioural consequences of focal brain lesions and, if nothing else provides a useful compendium of this literature in 18 chapters written by experts in the field. The first three chapters set the scene by providing a conceptual overview and discussions of methodological issues. The rest are devoted to specific topics and generally take the form of either examinations of the evidence for the localisation of specific neuropsychiatric syndromes (depression, mania, obsessive compulsive disorder, psychosis) or reviews of the effects of lesions of particular structures (thalamus, basal ganglia, frontal lobe, temporal lobe). In their overview, Cummings and Bogousslavsky group the disorders of mood, thought, affect, and motivation considered in this book under the rubric "fundamental dysfunctions" which they point out are associated with interruptions of limbic and frontosubcortical circuitry. This diffuse nature of these circuits is reflected in the finding that lesions of disparate areas can produce, for example, psychotic phenomena and that lesions in specific structures—for example, thalamus or basal ganglia, can result in a range of behavioural and emotional abnormalities. It is thus often difficult to make strong inferences from this literature about the neural basis of emotion and behaviour. Indeed some chapters are essentially categorised lists of studies without much in the way of theoretical underpinning. Several chapters, by Habib (disorders of motivation), Eslinger and Geder (behavioural and emotional changes after focal frontal damage) and Tranel (neural correlates of violent behaviour), stand out. These stray from localisation and bring in evidence from basic neuroscience and human functional neuroimaging to inform and interpret fascinating case histories in terms of a functional neuroanatomy of emotion and behaviour.

EILEEN JOYCE

Update in neurology for general practitioners. By P. ORTON, D. BATEMAN, G. FULLER, P. NEWMAN, D. PARK, R. SHAKIR, and G. YOUNG. (Pp 218 + 2 CD—ROM, £80.00) Published by University of Bath/British and Spine Foundation/Royal College of General Practitioners, Bath, 2000.

The seven authors of this text are to be congratulated on their innovative approach and the freshness they bring to the evaluation of the common neurological problems encountered in primary care. This text and the two accompanying CD ROMs represent a cooperative venture between the British Brain and Spine Foundation, the Royal College of General Practitioners and the University of Bath.

The text addresses the commonly encountered problems of headache, sensory disturbance, cerebrovascular disease, dementia, confusion, movement disorders, fits and faints, and back, leg, and arm pain. The approach is a simple one—namely, a definition of the condition or conditions, who gets it? What causes it? How does it present? What is the differential diagnosis? How is it investigated and managed? This pragmatic approach pays special attention to the prevalence of each disorder in the community and contains "red flags" where there is diagnostic doubt and uncertainty. The two accompanying CD ROMs outline learning outcomes, case histories, self assessment questionnaires, and case pathways with respect to investigation, management, and prognosis. Each such case is linked with the text providing assignments in clinical audit, comment, documentation of data, and "setting standards". Incorporated in all of this are useful components such as data collection sheets for specific symptoms and summary sheets to aid and improve documentation.

I found this an interesting book and enjoyed finding my way around the CDs, which were easy to use. I would imagine that this would be a valuable aid for the busy general practitioner and should help set standards in primary care that would aid patients and ensure thorough evaluation before referral. The only thing missing was guidance demarcating between evaluation in the primary care setting from the need to refer on for a specialist opinion. All of us occasionally think, under moments of work overload, that our colleagues in primary care could be a little more circumspect in whom they refer (although the truth of the matter is that most referrals are thoughtful and necessary). A text such as this with no obvious competitor, is long overdue, written by neurologists who, along with their general practitioner colleague, have adopted a common sense approach to their task. I would strongly recommend this book with its accompanying CD ROMs to my general practitioner colleagues in the belief that it would serve to demystify our specialty. Perhaps in future editions the authors might consider incorporating referral guidelines that would be a useful supplement to a text that, for example, takes general practitioners through from the suspicion of brain tumour to the process of biopsy and subsequent radiotherapy. While such data informs and allows the practitioner to explain to his or her patient what they might expect to encounter in the process of investigation and treatment, it does not clearly define who does what and when.

In conclusion, I recommend this innovative text, which, although directed primarily at our general practitioner colleagues, contains something for all on the simple approaches of evaluation, the importance of documentation, and the value of using an interactive CD ROM as an educational tool. I think that this text goes a long way not just to educate and entertain, but also remove from primary care, the notion that neurology is a difficult and uncertain area.

IAN BONE

Rehabilitation of visual disorders after brain injury. By J. ZIHL (Pp 187, £29.95). Published by Psychology Press, Hove, 2000. ISBN 0 86377 898 4.

This is the second series on neuropsychological rehabilitation fostered by Barbara Wilson and Ian Robertson (the first being *Neural repair, transplantation, and rehabilitation* by R. Barker and S. Dunnett). The renewed interest is welcome. Josef Zihl is one of the first clinical scientists, together with collaborators such as von Cramon, to have put a concentrated effort into rehabilitation of visual disorders, spanning a period of 2 decades. Much of this pioneering work lies in Germany, stemming from an older tradition going back to Poppelreuter, and the current book should make the developments better known outside that country. It remains something of a curiosity that visual rehabilitation is scarcely pursued in Britain and the United States, especially given the seminal animal work (by Cowey in Britain and Mohler and Wurtz in the United States) clearly demonstrating the potential capacity for recovery after V1 lesions in the monkey, with practice regimes. Indeed, Zihl's own work acknowledges the influence of these animal studies on his own research.

The Series preface describes this work as a "modular handbook". Handbook it is not. Rather it is a compact and relatively short account that is more like a progress report, full of interesting approaches described in mid-stream of the research, much of it still to be completed. It is broad ranging, the largest and most advanced section (about half of the book) being on visual field disorders, but progressing to visual acuity, colour vision, visual space perception (including Balint's syndrome), visual agnosia, and a final brief section on the special problems of a central scotoma. Each section starts usefully with evidence of spontaneous recovery, which is the baseline from which rehabilitative efforts must be assessed. Such efforts are almost entirely based on intensive practice (what I have called "grindsight"). Zihl is very cautious, lacing the evidence with statements such as "our observations produce preliminary evidence that spatial contrast sensitivity can, in principle, be improved by specific and systematic practice. However, the limited number of cases does not allow definite conclusions to be drawn". (p 96). He is also careful to distinguish between measurable effects in the laboratory and their "ecological value" to the subject in terms of any benefit in everyday life. In some regards he is over-cautious—for example, in his speculation (with von Cramon) "that recovery of visual field defects can only be expected with complete striate cortex injury" (p 34). Of course, complete V1 lesions are almost always accompanied by additional damage, when animal work demonstrates a reduced residual capacity. But a complete but restricted V1 removal in the monkey still allows further recovery to take place.

Anyone wishing to find practical approaches for helping patients across a range of visual disorders will find this a useful, humane, and dedicated book. He or she will have to work through a compact monograph and share Zihl's experience with him, rather than find a collection of shorthand recipes. It will be rewarding work.

L. WEISKRANTZ

An odd kind of fame. Stories of Phineas Gage. Edited by MALCOLM MACMILLAN (Pp 552, £24.95). Published by Massachusetts Institute of Technology, USA, 2000. ISBN 0 262 13363 6.