The authors reply:

Mark is right in noting that Kerkhoff *et al* have mentioned this phenomenon of contralateral bisection bias in hemianopia. We will first examine the data provided by Kerkhoff *et al*, then peruse the literature he quotes, to answer Mark's question.

Firstly, two of the three papers quoted by Mark do not provide data on hemianopic bisection bias. In one,¹ the phenomenon is mentioned in a single sentence in the methods section; in the other,² a German review contrasting hemianopia and hemineglect published last year, there is a short discussion about bisection without experimental data. The third paper studied bisection of vertical or horizontal lines in a series of six patients³: however, all had bilateral cerebral damage and complex bilateral field defects, not hemianopia. Extrapolating from such unusual patients to those with more common unilateral hemianopia is not always appropriate.

Kerkhoff *et al* state that others have made this observation before.¹⁻³ He quotes two works from the English literature. One is the paper by Schenkenberg *et al*.⁴ This report analysed by side of brain damage without respect to hemianopia. Furthermore, hemianopic bias cannot be deduced from their tabulated data: there is bisection data for only six patients with hemifield defects without neglect, five with left hemispheric lesions, and the type of field defect is not stated. An informal *t* test on the small sample of five patients with left hemispheric lesions shows no difference from the controls.

Another citation is a monograph by Teuber *et al*.⁵ This was also puzzling, because line bisection did not seem to be reported in the methods or results. We believe that Kerkhoff *et al* may have been referring to patients with lateral or vertical shift of fixation ("pseudo-fovea", p 79). It does not necessarily follow that a shift of fixation leads to a shift in bisection judgments, though. Teuber *et al* do state that this might occur (p 82), and refer to another work of theirs,⁶ a case study of a man with a right occipital wound and left hemianopia. However, his bisection errors were biased rightward, as with neglect, rather than leftward, as we find with hemianopia.

Kerkhoff *et al* are more accurate in noting precedents in the older German literature. We became aware of these when we had a German monograph of Werth translated recently.⁷ Axenfeld⁸ does seem to be the first to make this finding, in his report of a man with left hemianopia and leftward bisection errors. Apart from precedence, though, few conclusions are permitted from a single case study without controls.

The most important work we discovered is that by Liepmann and Kalmus.⁹ These authors documented contralateral bisection bias in 10 patients with homonymous hemianopia and one with bitemporal hemianopia. Interestingly, they noted that this bias was not present with very small or very large lines. They dismissed Axenfeld's hypothesis that the error was related to a coexistent gaze palsy,⁸ but speculated that it was related to increased numbers of saccades and fixations into the blind hemifield, an explanation which resembles the idea that the bias may be secondary to an adaptive shift of attention. (Note, though, that we and others have provided evidence that the spatial representation within normal hemifields can itself generate this type of bias.^{7,10})

We appreciate the opportunity to publicise the prior observations of Axenfeld⁸ and Liep-

mann and Kalmus,⁹ and the prescience of the latter's speculations. Among our colleagues, "hemianopic bisection error" does not seem to be well known. We would also suggest that its scientific foundation in the German literature could be improved. We have provided better evidence for both the existence of the bias and the conclusion that it is caused by hemianopia, with the use of statistical analysis, normal controls, and non-hemianopic brain-damaged controls, none of which were used in the older German work. We hope that our brief initial report and this correspondence increase awareness of "hemianopic bisection error".

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Myoclonus of peripheral origin

We read with interest the recently published article by Assal *et al* on "Post-traumatic stimulus suppressible myoclonus of peripheral origin".¹ They presented a patient with painless "myoclonus" of the first dorsal interosseous muscle of the foot that was abolished by local anaesthetic block. Electromyography showed bursts of rhythmic spontaneous grouping of motor units of 200 to 400 ms duration occurring at 1.6 Hz frequency. We take issue with the usage of the term "myoclonus" for the abnormal movements described. Myoclonus is a brief, shock-like, involuntary movement with EMG activity usually lasting less than 200 ms (often even 50 ms or less).² Myoclonic discharges are usually arrhythmic and irregular, with the exception of segmental (brainstem or spinal) myoclonus which presents with rhythmic, long EMG bursts (>100 ms) involving several spinal or body segments. Descriptions of the electrophysiology of peripheral myoclonus are scarce. In our experience, the EMG bursts in peripheral myoclonus can be >100 ms in duration (rarely more than 200 ms), but are not truly sinusoidal, are irregular in frequency and duration, and have a very variable interburst interval. By contrast, in the case presented by Assal *et al*, the EMG bursts appear sinusoidal, regular in frequency, and

come in trains of EMG activity that are interrupted by periods of silence. Furthermore, even in rhythmic myoclonus with long EMG bursts, the movements still appear brief and shock-like. In contrast, the description by Assal *et al* of the "myoclonus" in their case was continuous and rhythmic. It also seems unlikely that EMG discharges of 400 ms duration will appear brief or shock-like. We agree that the EMG bursts resemble that of the painful legs and moving toes (PLMT) syndrome, which is often associated with peripheral nerve or nerve root lesions.³ A variant of PLMT, however, can have no associated pain and is referred to as the painless legs and moving toes syndrome.⁴ The EMG findings presented by Assal *et al* may very well represent myokymia. Myokymic discharges fire rhythmically at 1-20 Hz and in trains that last a few seconds followed by a short period of silence.⁵ Myokymia is often seen in peripheral nerve disorders, is abolished by local nerve blocks, and is thought to represent ectopically generated potentials in demyelinated nerve fibres. A myokymic EMG pattern has also been described in PLMT due to hypertrophic mononeuritis.⁶

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The authors reply:

We thank Evidente and Caviness for their interest in our paper. Their comments on the descriptive term we used to describe the abnormal movements of the second toe of our patient are well taken. We already partially considered this issue in our discussion.¹

We do not agree that "myokymia" would adequately describe these movements for the following reasons. Clinically, there were no quivering nor undulating movements of the skin overlying the muscle, and, although myokymia may sometimes lead to a movement of the fingers or toes, this movement is of very limited magnitude, in our experience not nearly as gross as that seen in our patient.^{2,3} Electrophysiologically, EMG myokymic discharges concern single motor units⁴ whereas many motor units fired synchronously in our patient. In some conditions myokymia may be related to the discharge of numerous single or grouped fasciculation potentials,⁵ but this activity does not manifest gross rhythmic muscle contractions either.² Moreover, when myokymia have a peripheral origin, the ectopic activity originates from the motor nerve supplying the contracting muscle^{2,3,6} whereas we showed that this was not the case in our patient.¹

The particular condition in our patient was that the abnormal impulse generator seemed to be located on a sensory nerve (branch of

the deep peroneal nerve), distinct from the nerve (tibial nerve) supplying the contracting muscle. Therefore, a spinal cord relay had to be implicated. This might be an explanation for the rhythm and long duration of the myoclonic bursts seen since, as mentioned by Evidente and Caviness, spinal myoclonus may present such characteristics. This last hypothesis reinforces our impression that myoclonus, already used by others in similar conditions,⁷⁻⁹ was the most adequate term to describe the single painless moving toe of our patient.

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BOOK REVIEWS

Diagnosis and Management of Parkinson's Disease. Edited by CHERYL H WATERS. (Pp 240, US\$17.95). Published by Professional Communications, New York, 1998. ISBN 1-884735-33-9.

This is a new pocket size handbook on Parkinson's disease with 11 chapters covering all aspects of this disorder ranging from definition and classification to newer surgical therapies. It is well written and easy to read, although its format means that some of the pictures and figures are so small that they are difficult to interpret. However, the simple chapter layout and the marking out of them on the margins of the page does make it easy to use. The book makes good use of graphs and tables, although some of the tables are so overinclusive that it is hard to know their value in terms of what is rare and what is not.

The text is concise and clear although there are controversial statements that I would take issue with—for example, the existence of paraneoplastic parkinsonism and the notion that the intralaminar nuclei of the thalamus are part of the basal ganglia. Conversely other controversial issues are not discussed and

these include the cognitive aspects of early Parkinson's disease, the priming effect of levodopa in the development of dyskinesias and thus the treatment of young onset Parkinson's disease. Furthermore, the book rather overstates some of the trial data on various drug therapies which means that the best management of patients with Parkinson's disease can sometimes be hard to extract. Indeed the book is clearly written for the American physician, and so there is essentially no mention of lisuride, apomorphine, or cabergoline whereas pramipexole has a very high profile and selegiline receives little adverse publicity. Furthermore, the classification of multisystem atrophy and the distinction between it and striatonigral degeneration, Shy-Drager and sporadic OPCA in the United States can be confusing. This emphasis obviously makes it harder to accommodate in the United Kingdom market, and the audience for which the book is attended, although neurologists in training, unable to afford the bigger movement disorder books, would probably find this book useful.

However, as an easy to read, rapid guide to Parkinson's disease, this is a useful addition not least because of its length and cost and its attempt to deal with all aspects of the disorder equally.

ROGER BARKER

Sleep Disorders: Diagnosis and Treatment. Edited by J STEVEN POCETA AND MERRILL M MITLER. (Pp 234, US\$99.50). Published by Humana Press, New Jersey, 1998. ISBN 0-89603-527-1.

This book is much shorter than most that have recently been published on sleep disorders and is all the more useful for this. The authors have been selective in the topics that they have covered and have emphasised the management of sleep disorders in primary care. Well recognised problems such as insomnia are included, and the book also deals with common but often overlooked conditions such as the restless legs syndrome and periodic limb movements in sleep. The chapter on pharmacological aspects of sleep deals mainly with hypnotic drugs and their use in insomnia and the place of stimulant drugs, especially amphetamines, in conditions causing excessive daytime sleepiness. One of the most original and most useful chapters is that which discusses sleep disorders in children. The overlap of medical, psychological, family, and social factors which are all important at this age are carefully dissected out.

The book emphasises how patients present to medical attention, and how to assess the various symptoms of sleep disorders. The chapter on sleep disorders as preventive medicine is interesting and most of the chapters are enhanced by the inclusion of case studies. These are well chosen examples of clinical histories which exemplify how, and also how not, to manage sleep complaints. The tables, graphs and recordings from sleep monitoring records also make this book easier to read.

The authors cover each topic to a level which is sufficient for the non-specialist in sleep disorders, although some sections are rather superficial, and, as is almost inevitable in a multi-author book, different chapters assume different levels of knowledge and understanding by the reader. The inclusion of

a brief overview of the topic of some in the chapters would have been helpful. The discussion of excessive daytime sleepiness, for instance, concentrates on narcolepsy and gives little coverage of the many other disorders causing this symptom. Overall, however, this is a useful and informative book which will be helpful to both medical and non-medical staff who are involved in handling sleep problems.

JOHN SHNEERSON

Congenital Muscular Dystrophies. Edited by Y FUKUYAMA, M OSAWA, AND K SAITO. (Pp 432, NLG 420.00). Published by Elsevier Science, The Netherlands, 1997. ISBN 0-444-82487-1.

This book describes in detail the clinical features and latest research findings on congenital muscular dystrophy. The book follows on from an International Symposium on Congenital Muscular Dystrophies which was held in Tokyo in July 1994. The book has many positive features but also some less good features, so common in edited multi-author texts.

On the positive side, the book gives a very comprehensive and authoritative review of the clinical features of the various types of congenital muscular dystrophies. I particularly enjoyed reading (and learning) about the different clinical phenotypes from clinicians who clearly have seen many cases. The fascinating association of the muscular dystrophy with CNS abnormalities both clinical and radiological, are well described.

The chapters describing the morphological features in both muscle and brain are also comprehensive and helpful to those trying to establish a diagnosis. There is also an interesting chapter on the clinical management of patients, so important for those incurable diseases.

Where have the major advances in this area been. As with other muscular dystrophies, progress has been made by the finding of an absence of a structural protein, in this case merosin, in a subgroup of patients. The most common form of congenital muscular dystrophy in Japan, Fukuyama congenital muscular dystrophy, has been mapped to a specific chromosomal location and surely the defective gene will be identified. Finally, on the positive side, there is a comprehensive bibliography which was updated to March 1997—helpful for those wishing to know more.

On the negative side is a feature I find concerning with many multi-author books. Some chapters are repetitive, giving only a slightly different slant on the same problem. Also, towards the end of the book the chapters on pathogenesis I found less interesting than the chapters which tried to pull the clinical and pathological features together.

Who should buy this book? Well, it is a must for all paediatric neurologists who have an interest in neuromuscular disease, or aspiring to have an interest. For those with a general neurological interest it will be important to have access to a copy. It will also be helpful for adult neurologists, such as myself, who get a mental block at those clinical meetings where you are expected to remember the 85 varieties of floppiness. Be warned, I am now an expert and can confidently diagnose a case of Fukuyama muscular dystrophy; shame that they are only seen in Japan!

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