PostScript

CORRESPONDENCE

Mexiletine on segmental hyperhidrosis

Ishibashi *et al*¹ reported the excellent efficacy of mexiletine for the treatment of segmental hyperhidrosis in two patients (who had syringomyelia and cavernous haemangioma of the spinal cord, respectively). They presented the decrement in the patients' sweat rate by oral administration of mexiletine.¹

Previously we performed a clinical study focusing on sweating and identified 10 patients with segmental hyperhidrosis among 30 patients with syringomyelia. We followed up the patients with hyperhidrosis for 1–10 (mean 5.0) years. The amount of sweating did not change in any of them during the follow up period,² although we did not perform a quantitative analysis. Consequently, we speculated that hyperhidrosis persists for at least a year. It is possible that the course of signs in the cases reported by Ishibashi *et al* were modified by the growth or activity of spinal cord lesions. We consider it imperative that these authors describe any spinal cord lesions and how they may have shifted. However, although they did not mention the duration and time courses of the improvement in their patients, we suppose that the duration of the follow up for each patient would not have exceeded several months, judging from how the authors described their experience. In addition, even though they did not test the effects of mexiletine on control subjects or on other parts of the body in the same patients, we can be assured that the improvement in hyperhidrosis was due to the oral administration of mexiletine, on the assumption that the spinal cord tumour could not have changed in such a short time. We consider that it would be informative for clinicians if Ishibashi *et al* were to disclose the drug dosage and the time course of its effects and to describe the features of the spinal cord lesions.

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Authors' reply

We are grateful Sudo *et al*, as they allow us to clarify a point of our study that was not discussed in the paper recently published in this *Journal*.¹ They asked about the possibility of natural remission and the non-specific effect of mexiletine on sweating.

We administered 200 mg/day mexiletine or 400 mg/day carbamazepine to our patients. Both patients noticed their hyperhidrosis was relieved within two days after administration. Although we did not perform a quantitative analysis several months after treatment, the clinical improvement of hyperhidrosis persisted. In addition, the magnetic resonance images of spinal cord lesions (syringomyelia and cavernous haemangioma) in both patients were followed up for two years. During the follow up period, the spinal cord lesions did not change their size, position, and intensity on magnetic resonance imaging. Therefore, the natural course of the spinal cord lesions could not explain the improvement of hyperhidrosis during the treatment and quantitative analysis in our patients.

The sweat rate of the area of observed hyperhidrosis was decreased without a change of the absolute sweat rate on the healthy side after oral administration of mexiletine. We calculated the ratio of the sweat rate on the affected side to that on the healthy side—the ratio was 2.13 before treatment and decreased to 0.97 on day 7 after the treatment. We therefore consider that the mexiletine had an excellent effect only on the area with hyperhidrosis. Although we did not test the effects of mexiletine on control subjects, we think that the result on a healthy area of each patient was an appropriate internal controls for the evaluation of the drug's effect on hyperhidrosis.

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Patent foramen ovale, cerebrovascular risk, and complement

Nedelchev *et al*¹ report that the presence of a patent foramen ovale (PFO) is a significant risk factor for recurrent cerebrovascular events, the risk being higher in patients with more than one previous embolic event. They highlight the absence of a current proven medical treatment or prevention regimen. Cardiac right to left shunting is present in a quarter of the population. It is thus worth drawing attention to a particular subgroup of patients with PFO that may be at an even more increased risk than the authors report—sport divers, most of whom fall within the age range of the above study.

Neurological sequelae constitute 80% of decompression sickness. Not only has neuroimaging shown an increased frequency of brain ischaemic lesions in divers, but also multiple such ischaemic lesions have been found specifically in sport divers with PFO.² While PFO patency of haemodynamic significance is a risk factor that necessitates habit modification, often the radiological lesions do

not correspond well to the neurological deficits of experienced divers.

This point, coupled with the increased risk of arterialisation of venous bubbles and the paradoxical nature of bubble genesis, suggest that a PFO is a risk factor in this subgroup for the development of neurovascular disease.³ Unknown is the added risk with age that remains to former divers. A poorly understood mechanism of bubble induced complement involvement in the pathogenesis of the neurological sequelae in decompression sickness has been suggested.⁴ Similarity of such symptoms to the postcoronary bypass syndrome lends support (and hope?) to complement based neuroprotective strategy options for the future.⁵

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- 5 D'Ambrosio AL, Pinsky DJ, Connolly ES. The role of the complement cascade in ischemia/reperfusion injury: implications for neuroprotection. *Mol Med* 2001;**7**:367–82.

Authors' reply

We thank Dr Demetriades for his comments on our study. While the average person with a patent foramen ovale (PFO) may not be at increased risk for neurological events, there seem to be subgroups of patients at increased risk. PFOs with large diameters, right to left shunting at rest, or high membrane mobility and PFOs associated with atrial septal aneurysms have been identified as “dangerous PFOs” by several investigators.^{1–3} In addition, coagulation abnormalities may promote paradoxical emboli in patients with PFO.⁴ To this list, Dr Demetriades adds special occupations or sports that may be dangerous in people with PFOs, specifically divers. Playing wind instruments has also been mentioned previously.⁵

However, many problems related to PFO remain unresolved. Even in groups that are believed to be at high risk for neurological events, deciding whether and how to treat a PFO cannot be derived from evidence based medicine. Deciding how to proceed depends on the opinion of the attending physician and is not based on data from randomised studies.

The PICSS (PFO in cryptogenic stroke study) showed that secondary prevention of cryptogenic stroke in patients with PFO by using warfarin or aspirin does not result in any difference.⁶ The PC-trial is an ongoing randomised trial we initiated to compare

endovascular PFO closure versus medical treatment alone. We hope that it will provide useful information on secondary stroke prevention in patients with presumed paradoxical embolism. It is also conceivable that divers who have ever had "the bends" would benefit from PFO closure.

Recently reported data suggest links between decompression illness, migraine with aura, and right to left shunts.⁸ These observations not only extend the clinical manifestations of PFO but also bring into discussion new pathophysiological aspects of migraine. If the association between complicated migraine and PFO can be corroborated, a randomised trial on PFO in such patients may be worth while.

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Demyelination in the brain as a paraneoplastic disorder: candidates include some cases of seminoma and central nervous system lymphoma

We read with interest the report of Ayuso-Peralta *et al*,¹ which describes a 58 year old woman who presented with several neurological symptoms. Brain imaging was consistent with leukoencephalopathy, and analysis of blood and cerebral spinal fluid was unrevealing. A few months later the patient experienced further neurological deterioration and an open brain biopsy showed central nervous system (CNS) lymphoma, together with diffuse demyelination.

The authors observed that the presentation of cerebral lymphoma as a diffuse leukoencephalopathy is not frequent and they discuss

possible aetiologies of the predominant demyelination in their case. They do not mention the possibility of a paraneoplastic aetiology.

The authors reference a similar case² previously reported in the *Journal*. That report also does not acknowledge a possible paraneoplastic aetiology for prominent diffuse brain demyelination preceding the discovery of CNS lymphoma. Two other recent reports in the *Journal*^{3–4} described focal tumour-like lesions of brain demyelination that preceded the discovery of CNS lymphoma. Only one of these reports⁴ mentioned laboratory data that suggested consideration of a paraneoplastic aetiology, the presence of serum antibodies directed against myelin oligodendrocyte glycoprotein.

One report elsewhere⁵ has described a patient who had a non-neurological malignancy and seminoma and who subsequently developed a paraneoplastic syndrome simulating encephalitis associated at autopsy with multiple foci of demyelination confined to cerebral white matter. Two other reports elsewhere^{6–7} have described biopsy confirmation of large focal demyelinating lesions in the brain associated with seminoma. The authors of these three reports all strongly considered the possibility of a paraneoplastic aetiology for the brain demyelination associated with seminoma, probably because the temporal association was close and the spatial association was distant.

The associations between brain demyelination and CNS lymphoma have been close, both temporally and spatially, making considerations of aetiology more complex. Taken together, the seminoma reports and the CNS lymphoma reports have many similarities in their patterns of associated brain demyelination, raising the possibility of similar mechanisms of demyelination. Many questions concerning aetiology remain unanswered. Given the information available, we suspect a paraneoplastic aetiology in all of these cases. We feel that future reports of brain demyelination associated with CNS lymphoma should consider this possibility in their data collection and in their discussion of results.

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Solitary focal demyelination in the brain as a paraneoplastic disorder. *Med Pediatr Oncol* 1996;**26**:111–5.

Genotype predisposition to leukoaraiosis

Leukoaraiosis, which can cause symptoms ranging from a mild cognitive impairment to severe subcortical dementia,¹ is a significant public health problem. One quarter of subjects aged 65 years or over are affected by some degree of white matter changes.² Ischaemic demyelination and small vessel disease seem to be important features of the underlying pathological process of this entity.^{1,2} Age, hypertension, and a previous stroke event have been proved to be the main clinical risk factors.^{1,2} A number of genetic susceptibility factors for leukoaraiosis have been put forward, with the assumption of polygenic aetiological factors. We were pleased to read the article by Hassan *et al* in this journal.³ The authors stated that the angiotensin converting enzyme insertion/deletion (ACE I/D) polymorphism D/D genotype was a significant independent predictor for leukoaraiosis in patients presenting with classic lacunar syndromes.³ We earlier conducted large prospective studies in which we also examined the importance of the ACE D allele and other common mutations in the development of small vessel infarction and leukoaraiosis.^{4,5} Our results were consistent with the findings of Hassan *et al* and support their results from several other aspects. (1) Our stroke study confirmed the genetic heterogeneity of ischaemic stroke in that the ACE D/D genotype proved a significant susceptibility genotype for small vessel brain infarction, as did the Leiden V mutation for large brain infarction.⁴ (2) In our leukoaraiosis study, the ACE D/D genotype was found to be a significant risk factor for leukoaraiosis in combination with brain infarction.⁵ (3) We also reported that clustering of the homozygous MTHFR 677TT and ACE D/D mutations in one person can mean a moderate (about fivefold risk), but highly significant ($p < 0.0005$) risk of leukoaraiosis without infarction.⁵ These data from different approaches reconfirm the possible aetiological role of the ACE D/D genotype in leukoaraiosis relating to small vessel brain disease. The genotype differences may explain why some patients who are exposed to clinical risk factors such as hypertension, exhibit a much higher susceptibility to leukoaraiosis than other subjects with the same clinical risk factors. Besides the classic clinical risk factors, the consistently growing knowledge of the genetic background of leukoaraiosis may permit the recognition of a large population at high risk of a new type of white matter damage, and hence this may lead to a more effective prevention.

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BOOK REVIEWS

Neurochemistry of consciousness: neurotransmitters in mind

Edited by Elaine Perry, Heather Ashton, and Allan Young (Pp 346, EUR65.00). Published by John Benjamins Publishing Company, Amsterdam, 2002. ISBN 90-272-5156-8

Consciousness is a portmanteau word, full of rich and different meanings: contrast Marxian, Freudian, and anaesthesiologists' use of the term. In recent years it has also become a fashionable hunting ground for neuroscientists, who are rarely troubled by such complexities. For them, consciousness is being awake rather than asleep, being reducible to awareness. Sweeping aside centuries of philosophical debate, they ponder over whether "it" "resides" in specific anatomical brain structures, in microtubules, in patterns of neurotransmitter release, or whatever. The present book is typical of this type of cheerfully unsophisticated empiricism: its hunt for what the editors call "NCCs"—neural correlates of consciousness—focuses on neurotransmitters, hence the subtitle. However, the concern with "mind" ceases at that point; this elusive phenomenon finds no place in the book's index. The central question for the editors seems to be whether the acetylcholine or the dopaminergic system is the more likely substrate for conscious awareness. This reductionism characterises most of the chapters. That on memory, for instance, abandons even animal memory for a discussion of a physiological phenomenon called long term potentiation, and even the psychoanalyst Mark Solms, on dreams, who surely ought to have a broader perspective, confines himself to contrasting cholinergic and dopaminergic hypotheses. However, the authors are clearly writing to an editorial brief: each chapter, in a book ranging from discussions of attention and motivation through psychotropic drug mechanisms to mental retardation and autism, following a brief nod to marginally wider concerns, offers a neurotransmitter list of potential associations or correlations with "states of awareness." Within these limitations many of the chapters provide competent student friendly overviews of their themes. If the book's pretensions weren't so much larger this would be fine; as it is, those hoping for a more multilevel or theoretically informed discussion will be disappointed.

Steven Rose

Risk control and quality management in neurosurgery

Edited by H-J Steiger and E Uhl (Pp 227, EUR108.000). Published by Springer Verlag Wien, Wien, 2001. ISBN 3-211-83678-0

This is an interesting and timely publication. The book contains a compilation of material presented at an international meeting held in October 2002. It has been divided into various sections that take the reader through grouped papers and topics and finally a projection into the future. As would be expected, the material covers experience and lessons gained in other areas such as aviation and nuclear research. The authors, generally senior in status, originate from Europe, the United States, and the United Kingdom and therefore offer a diverse collection of views, opinions, and experience relevant to a very wide readership. The increasing requirements for quality assessment and competency make this a very valuable reference book for both departmental and institutional libraries. However, it certainly will be of value to individual readers. It should be recommended reading for trainees to understand the principles and the ongoing thought behind many of the practices and control measures that they will encounter and will need to participate in as their experience and seniority advance. The quality of contributions and the outline of the information do vary, as would be expected in such a compilation, but overall very few pages or chapters do not prove insightful nor provide useful information and guidelines. It will be of value to all medical disciplines, since the principles are universal and the terms of reference or yardsticks used are convertible or transferable. It is highly recommended.

J Van Dellen

Primary progressive multiple sclerosis

Edited by M Filippi and G Comi (Pp 128, EUR47.95). Published by Springer, Milan, 2002. ISBN 88-470-0167-6

The field of multiple sclerosis (MS) is awash with literature on every aspect of the disease ranging from epidemiology and genetics to pathology and treatments. It is unusual, therefore, to find a lacuna in this niche but this book seems to have found one.

Primary progressive multiple sclerosis is written to encapsulate the latest evidence on aspects of this condition, which until recently was not regarded as important in understanding demyelinating disease. Filippi and Comi have brought together all the important players in the study of primary progressive MS. Their contributions summarise the latest information on the epidemiology, genetics, immunology, pathology, imaging, and clinical trials and therapies in primary progressive MS. This book is meant to be a useful guide to the subject and does not profess to be an authoritative account. However, it occasionally is a little too brief in its explanations and definitely lacks pictures, tables, and diagrams in the early part of the book. This makes it a rather bland and dry account initially. When the diagrams and scanned images do appear in the latter parts of the book, many of them lack definition and it is not always easy to see the details that are being referred to.

All in all this is a good up to date summary of the latest news in primary progressive MS and in particular the imaging aspects of the disease, as would be expected from the interests of the editors. It would be a useful adjunct to other literature for those working in the field of demyelinating disease.

Omar Malik

Multiple sclerosis: a guide for the newly diagnosed, 2nd edn

By N J Holland, T J Murray, and S C Reingold (Pp 160, US\$21.95). Published by Demos Medical Publishing Inc, New York, 2002. ISBN 1-888799-60-9

This book is an invaluable guide for patients with multiple sclerosis (MS), as well as their friends and families. The fact that a second edition has become necessary is extremely encouraging for those involved with MS and highlights the recent therapeutic advances for this still devastating diagnosis. Most people who develop MS are desperate for information about their new disease and many turn to the internet to find this. Unfortunately, they are then faced with misleading or simply incorrect information, which can leave patients confused or disillusioned.

The authors present detailed information in the first two chapters covering the pathological processes causing the symptoms of MS and the diagnostic tests in use. Uncertainties in both these fields are explained. The next two chapters deal with treatments, including conventional and alternative or complementary therapies; the text is clear about the lack of a cure for MS but discusses all the options including steroids for acute attacks, disease modifying drugs, and symptomatic treatments. There is a whole chapter on the important issues of lifestyle—diet, rest, sexual function, pregnancy, etc.—that help patients to control their condition. A further chapter concentrates on the psychological impact of a diagnosis of MS and its effect on relationships. Employment issues are deservedly dealt with on their own, with practical advice on when and how to disclose the diagnosis and the legal implications of disclosure both at work and on application forms such as those for health and life insurance.

The latter part of the book deals with clinical and research trials in MS that will help patients to understand how trials are designed and why treatments are offered to patients with specific disease types. The many fields in which MS research is ongoing are described and the questions being asked by investigators are well presented.

The book ends with more practical advice on how to get further information about specific topics; however, this is predominantly aimed at the North American readership with emphasis on the MS societies of the United States and Canada.

In summary, this is an excellent book, which presents all the facts in a straightforward but sympathetic way. As well as the medical facts about the disease, it is full of practical advice covering all life topics, areas that are often neglected by busy physicians. It is highly recommended to all those whose lives have been affected by this disease.

Valerie Stevenson

Disordered mind and brain: the neural basis of mental symptoms

By Peter F Liddle (Pp 320, £40.00). Published by Gaskell, London, 2001. ISBN 1-901242-65-X

The premise of this book is that the key to understanding the neural basis of the major mental disorders is an understanding of the origin of five symptom clusters or dimensions common to these disorders. These are reality distortion (hallucinations and delusions); disorganisation (of thought and behaviour); psychomotor poverty and excitation; depression and elation; and anxiety. Thus, there are five chapters each devoted to a description of a specific dimension and an exposition of how it is correlated with cognitive abnormalities derived from the dysfunction of specific neural processes.

These central chapters are preceded by five chapters describing the neuroscience of brain systems thought to be involved in generating the various symptom clusters. These are brief and the literature reviews are in no way comprehensive. Nevertheless, they serve the purpose of informing the reader of the basic neuroanatomical and neurophysiological concepts that underpin Professor Liddle's approach to understanding mental illness.

The final four chapters summarise the current evidence regarding the neurobiology of schizophrenia, bipolar affective disorder, obsessive compulsive disorder, and psychopathy.

Each ends with a synthesis that integrates this with the previous account of how the symptom clusters arise.

The explanatory power of Professor Liddle's thesis concerning the neural basis of mental symptoms is stronger for some symptom dimensions, such as reality distortion, than others, such as distortion. But it is the general unifying approach that is the major strength of this book—the detail will certainly be honed over the next decade. Another strength is that this is a self contained book! It assumes no neuroscientific or medical knowledge other than the most basic. There are many excellent colour illustrations. Therefore, this book can be highly recommended to anybody interested in the disordered mind and brain.

Eileen Joyce

CORRECTIONS

Rosso SM, van Swieten JC, Roks G, *et al.* Apolipoprotein E4 in the temporal variant of frontotemporal dementia. *J Neurol Neurosurg Psychiatry* 2002;**72**:820.

Due to the style used in house for listing authors affiliations in the Letters section of the journal, the author's names have been incorrectly listed. The correct order should read as follows:

Rosso SM, Roks G, Cruts M, van Broeckhoven C, Heutink P, van Duijn CM, van Swieten JC

This also applies to:

Lünemann JD, Kassim N, Zschenderlein R, *et al.* Rhabdomyolysis during interferon- β 1a treatment. *J Neurol Neurosurg Psychiatry* 2002;**72**:274.

The correct order of the authors is: Lünemann JD, Schwarzenberger B, Kassim N, Zschenderlein R, Zipp F.

Aarsland D *et al.* Donepezil for cognitive impairment in Parkinson's disease: a randomised controlled study. *J Neurol Neurosurg Psychiatry* 2002;**72**:708–12. An error occurred in the production process in which the codes of the two lines were erroneously interchanged. The correct figure appears below:

Figure 2 Change in mini mental state examination (MMSE) score from baseline over the two treatment sequences. Values are mean (SE).