

PostScript

CORRESPONDENCE

Potentially misleading extratemporal lobe lesions

We read with interest the paper by Dr Alsaadi and colleagues on “potentially misleading extratemporal lobe lesions” in patients with symptoms of temporal lobe epilepsy.¹ Most of their successfully operated patients with extratemporal lesions also had hippocampal atrophy on the operated side, which has positive prognostic value for mesiotemporal surgery in temporal lobe epilepsy, even in the presence of extratemporal lesions.² Their material provides excellent evidence that not all lesions in an epileptic brain are epileptogenic, and in some cases epilepsy may originate from morphologically intact mesio-temporal structures in a lesioned brain. However, this situation is only one of the possible scenarios of the very complicated relations between mesiotemporal epilepsy and extratemporal lesions.

Another possibility is that mesiotemporal epilepsy is induced by secondary epileptogenesis from an extratemporal lesion. This possibility has not been clearly proven but there are reports of patients with mesiotemporal lobe epilepsy and potentially epileptogenic extratemporal lesions such as hypothalamic hamartomas, midline lesions, and retrosplenial lesions³ behaving like temporal lobe epilepsy. The poor surgical outcome when resection involves only the mesiotemporal structures and the good outcome when the extratemporal lesion is also resected may be explained on the basis of secondary mesiotemporal epileptogenesis from extratemporal sources.

A third possibility is that the temporal lobe acts as a symptomatogenic area for seizures spreading to it from an extratemporal source. In such cases resection of the temporal lobe may result in an Engel Ib outcome: auras persist but seizures stop.

The big question is how we can know whether the extratemporal lesion is epileptogenic or not—in other words, what is the chance of being successful when operating only on the temporal region, or only on the extratemporal lesion, or on both?

At present only clinical experience can help us in this regard. “Encephalomalacic lesions,” which form a majority of the authors’ extratemporal lesions, are not as epileptogenic as—for instance—the well known periventricular or diffuse nodular heterotopias. The latter types of extratemporal lesion are reported as being present in patients who did not become seizure-free after resection of the mesiotemporal structures.^{4,5} In agreement with Alsaadi *et al* and other workers,⁶ we do not consider arachnoid cysts to be epileptogenic unless they overlie a pathological cortex. Our impression is that post-traumatic lesions have an intermediate position, and the high epileptogenic potential of tumours is well known.

On the basis of similar studies it would be helpful to make an epileptogenicity list of different brain lesions. In combination with an optimal preoperative examination

schedule, this could help in preoperative decision making.

A Szűcs, G Rásonyi, J Janszky, P Halász

National Institute of Psychiatry and Neurology, 1021 Hűvösvölgyi út 116, Budapest 1021, Hungary

Correspondence to: Dr A Szűcs; szucsan@bakats.tvnet.hu

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Author’s reply

We appreciate the comments from Dr Szucs and colleagues. As not all of our patients became completely free from seizures after temporal lobe surgery, we cannot exclude secondary epileptogenesis or propagation through temporal structures in a minority of cases. Nevertheless, we expect that our outcomes would have been much worse if these mechanisms were responsible for temporal lobe epilepsy (TLE) or “apparent TLE” in most of our patients.

We agree that the suspected pathology of the extratemporal lesion is critical in determining the presurgical evaluation and the surgical plan. Many of our patients had aetiologies such as trauma that are commonly associated with multifocal brain pathology. We certainly suggest that suspicion for TLE be higher in these patients than in patients with extratemporal gliomas.

T Alsaadi

University Of California Davis, Department of Neurology, 4860 Y Street, Suite No 3700, Sacramento, CA 95817, USA; taoufik.alsaadi@ucdmc.ucdavis.edu

Brain tissue guided treatment supplementing ICP/ CPP therapy after traumatic brain injury

The recent article by Meixensberger and colleagues comparing a brain tissue oxygen guided treatment protocol with an ICP/ CPP protocol¹ presents insufficient evidence to support the conclusion that P_{ti}O₂ guided cerebral perfusion management reduced cerebral hypoxic events following traumatic brain injury. Brain tissue oxygen levels can easily be modified through changes in

F_iO₂.^{2,3} Although the authors indicated that changes in F_iO₂ were not used to raise the P_{ti}O₂, no data were presented to support this assertion. What was the F_iO₂ before and after cerebral hypoxic episodes in each group? Furthermore, despite no significant difference in CPP, P_{ti}O₂ was significantly higher on days 1, 2, 5, and 6. This could be explained by higher P_aO₂ levels in the P_{ti}O₂ guided group, yet no data on P_aO₂ were presented. To assess the influence of the P_{ti}O₂ guided cerebral perfusion management accurately, the patient’s P_aO₂ and incidence of systemic hypoxaemia must be known, because of the strong correlation between P_aO₂ and P_{ti}O₂. These data are essential to conclude that cerebral perfusion management, and not P_aO₂ management, was responsible for the reduction in cerebral hypoxic episodes.

A major difficulty in the analysis of two consecutive series of patients lies in ensuring that the groups are equivalent at baseline and controlling for confounding interventions. General intensive care management has significantly evolved in the past decade and the possibility that co-interventions unrelated to perfusion management were responsible for the reduction in cerebral hypoxic events cannot be ruled out. Furthermore, the non-consecutive nature of recruitment may have resulted in selection bias. As the authors pointed out, it is clear that randomised controlled studies are needed to evaluate properly current monitoring and therapeutic strategies in the management of severe traumatic brain injury. Although expensive and labour intensive, such trials are warranted because of the tremendous burden of this disease on patients and society.

Correspondence to: Dr D Zygum, University Department of Anaesthesia, University of Cambridge, Box 93, Level 4, E block, Hills Road, Addenbrooke’s Hospital, Cambridge CB2 2QQ, UK; dzygun@ucalgary.ca

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Dural tear and intracranial hypotension in a chiropractic patient

The report of Dr Beck *et al* that a spinal chiropractic manipulation may lead to intracranial hypotension opens a debate between internal/genetic forces versus external/epigenetic events in the aetiology of dural tears.¹ Considering that only 20% of patients with basilar fractures resulting in a dural tear experience CSF leakage,² one could question

Table 1 Evidence of connective tissue disorders in eight patients from a cohort of 69 chiropractic patients under 45 years of age

Patient no.	Age, sex	Height (cm)	Arm span (cm)	Ratio*		Clinical presentation
1	16, M	173	174.5	1.01	Median	Mild pectus excavatum, thin long fingers, scoliosis
2	24, M	188	193	1.03	>95th percentile	Mild pectus excavatum, scoliosis
3	29, M	186	195.5	1.05	>90th percentile	Pectus excavatum, loss of elbow angle, family history of Marfan
4	19, M	186	185	0.99	>90th percentile	Mild pectus excavatum, scoliosis
5	13, M	181.5	181	1.00	>95th percentile	Pectus excavatum, scoliosis
6	21, F	168	172	1.02	>90th percentile	Mild pectus excavatum
7	21, F	160	168	1.05	>90th percentile	Mild pectus excavatum, striae, long thin fingers
8	39, F	164.5	167	1.02	>90th percentile	Mild pectus excavatum, scoliosis

*Marfanoid habitus is considered when ratio of arm span to height is greater than 1.03.

how an external force from a physiological movement of the neck, which when delivered by hand generates only a minute fraction of the forces needed to fracture a bone, could tear a “healthy” dura. However, internal weakness of the dura has been noted, suggesting that an underlying hereditary disorder of connective tissue (HDCT) may exist in patients with CSF leakage.^{1,3} Dr Beck accurately reports that microfibrillinopathy is quite common in these patients, yet we are left with a quite tertiary or “general” examination to eliminate connective tissue disorders.¹

The most widely used method to test the hypermobility of joints is to test whether the patient can perform a series of nine manoeuvres (Beighton score), which is included in the revised (Brighton 1998) criteria for the diagnosis of benign joint hypermobility syndrome (BJHS)/Ehlers-Danlos Type III (EDS III).³ I used the Beighton and Brighton hypermobility index in 118 consecutive patients that entered my clinic over a 5 month period. Of the 69 patients under 45 years of age (34F; 35M), 39% demonstrated BJHS (53%; 26% respectively) an extremely high percentage compared with that in other specialties. Furthermore, as summarised in table 1, I was able to identify eight patients with characteristics of Marfan syndrome, a condition that carries the highest risk for dural weakness.^{1,3,5} Interestingly, four of the patients with marfanoid habitus did not demonstrate BJHS. None of these patients suffered negative reactions from chiropractic manipulations.

Chronic musculoskeletal pain is a common manifestation of HDCT patients, with back or neck pain dominating the lives of many patients with Marfan and Ehlers-Danlos syndromes,^{3,5} leading them to seek care from specialists such as chiropractors. I suggest that as there exists within the chiropractic patient population a high prevalence of HDCT, the conclusion that external forces from a spinal chiropractic manipulation alone can lead to intracranial hypotension via a dural tear may be premature, and that patient selection from within this specific patient group, without testing of their genetic loads, certainly biases the conclusions of the studies.

Owing to the varied organ systems affected in patients with HDCT (heart, bone, ocular, skin), many different specialists tend to examine these patients.^{3,5} I propose a standardised

examination for the detection and reporting of suspected patients with HDCT that would improve cross speciality communication and improve our knowledge of the role that HDCT plays in the pathogenesis of dural tears and neurovascular accidents.

J O Di Duro

Private Chiropractic Practice, Vicenza, Italy, and Postgraduate and Continuing Education Department Faculty, New York Chiropractic College, Seneca Falls, NY, USA

Correspondence to: Dr J O Di Duro, Via Prati 14, 36100 Vicenza, Italy; jdiduro@protec.it

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BOOK REVIEWS

Botulinum toxin in painful diseases

Edited by W H Jost. Published by Karger, Basel, 2003, pp 168, €128.50. ISBN 3-8055-7500-9

It is not surprising that botulinum toxin is now an increasingly popular treatment for pain disorders including tension headache, migraine, and myofascial pain syndromes. These conditions may not be associated with muscle tension or in any event with increased EMG activity. Furthermore, many trials to date suffer from not being randomised

placebo controlled studies, patient numbers are often small, and follow up is brief. It is against this background that this new book should be read with caution. The authors from various German centres offer possible explanations why botulinum toxin may work in the absence of strikingly high abnormal muscle tone. Stimulation of substance P and enkephalin and a discrete anti-inflammatory effect have been mooted. Irrespective of the theoretical difficulties, the relative sparsity of convincing control trials results in difficulties in balance within the chapters. For example, the section on tension headache includes 12 pages on classification and conventional therapy and only 4 pages directly discussing botulinum toxin therapy. The chapter on low back pain is even more unbalanced—8 pages on background and then the statement “considering the scarce data, we must refrain from recommending BTX injections in low back pain for the time being”. The chapters concerning pain relief in cervical dystonia and spasticity are well done but these conclusions are already widely known and covered in other texts. The chapter on piriformis syndrome is an excellent review of this interesting condition, irrespective of whether botulinum toxin works or not.

For those interested in an overview of pain management with botulinum toxin my recommendation would be to read the chapter by Nurmikko in the excellent second edition of *Handbook of botulinum toxin treatment*.

N Hyman

Textbook of clinical psychiatry, 4th edition

Edited by Robert E Hales and Stuart C Yudofsky. Published by The American Psychiatric Publishing Inc, Washington DC, 2003, pp 1764, US\$229.00. ISBN 1-58562-032-7

This is an impressive textbook; experts with international reputations have contributed to its pages covering all aspects of psychiatric practice. As one might expect from an American textbook, categorical descriptions, backed up by DSM-IV, and psychodynamic principles are both given high profile. Many chapters follow a similar pattern, with the rhythm set by the boxes describing the DSM criteria for each of the diagnosis under discussion. The book even comes with a CD-ROM of DSM-IV-TR.

A psychiatrist in training will find almost all he/she needs to know in this book, but I am less confident that it will be of value to clinical neurologists. Any book that is led by DSM (or ICD) is unlikely to perform for clinicians who want a good exposition of neuropsychiatry. And this is evident here; neuropsychiatry only being represented by one chapter on delirium, dementia, and amnesic disorders and then just three separate chapters (somatoform disorders, factitious disorders and malingering, and dissociative disorders) unhelpfully parcelled out to separate sets of authors. Indeed, I found the chapter on dissociative disorders particularly poor; and nothing on pseudo-seizures!

Given that the editors are two thirds of the team that produced the excellent *Neuropsychiatry of traumatic brain injury*, I, on behalf of neuropsychiatry, was a little disappointed. In addition, when one finds later

in the book almost 50 pages devoted to hypnosis, a treatment by and large without any evidence base, as against 60 pages devoted to schizophrenia and other psychotic disorders, one wonders about the editors' sense of proportion.

S Fleming

The treatment of obsessions

By S Rachman. Published by Oxford University Press, Oxford, 2003, pp 158, £22.95. ISBN 0-19-851537-5

This practical manual on the cognitive behavioural treatment of obsessive compulsive disorder (OCD) was recently published in the *Cognitive behaviour therapy science and practice* series, edited by David Clark, Christopher Fairburn, and Steven Hollon. The author is an authority in the field of OCD who has laid the foundations of the behaviour theory of OCD and has remained doing important research on the development and treatment of the disorder ever since.

Although this book shows that he has moved towards including more cognitive elements in his treatment approach, his behavioural background shows in the lack of a coherent, developmental, cognitive behavioural formulation. As a consequence, the presented treatment techniques tend to remain patchy and arbitrary. The verbal and behavioural challenging of the meaning of obsessions could be described in more detail.

In his introduction, the author states that although considerable progress has been made in treating compulsive behaviour, this was not accompanied by comparable progress in dealing with obsessions. Although the book is aimed at filling this gap of treating "patients with pure obsessions", many of the examples in the book are of patients with compulsive behaviours and most of the techniques are equally applicable in obsessive patients with or without compulsions. The book has been clearly written, but there is considerable overlap and repetition between the different chapters. Although the author sometimes attempts to define the terms he is using, a better demarcation of concepts as intrusive thoughts, obsessions, preoccupation, and rumination; compulsions, neutralisation, safety behaviour, and avoidance; and exposure and behavioural experiments would be very helpful.

Having said that, the book offers a wealth of ideas to use in the treatment of OCD and includes a lot of practical examples and case illustrations. The author has generously made available some of the interviews, questionnaires, and information material he has used with patients. I am sure this book will prove a very helpful adjunct for all therapists who are involved in the treatment of patients with OCD.

A Speckens

NINDS at 50: an incomplete history celebrating the fiftieth anniversary of the National Institute of Neurological Disorders and Stroke

By Lewis P Rowland. Published by Demos Medical Publishing, New York, 2003, pp 321, \$44.95. ISBN 1-888799-71-4

The epicentre of neurological research, which in the 1950's was Cambridge, England, has since shifted to Bethesda, Maryland. If you want to know why, read this book about the history of the National Institute of Neurological Disorders and Stroke (NINDS), from its origin in 1950 to the present. For those unfamiliar with the organisation of the National Institutes of Health (NIH) in Bethesda, the NINDS is the "neurological institute", whose research activities overlap those of other institutes, including aging (NIA), vision (NEI), and mental health (NIMH).

At the inception of its 50th anniversary, the NINDS director asked Lewis P (Bud) Rowland, who had retired as Chair of Neurology at Columbia, to write the NINDS's history. The product is outstanding. It not only provides the hard facts, but an explanation of the behind the scenes activities that propelled and nurtured the institute's growth.

Rowland meticulously traces the evolution of the institute, which consists of extensive intramural and extramural programmes, the latter funding most of the neuroscience research in the United States. Biographies of the successive NINDS directors, the politicians, and prominent lay supporters who promoted its development, and its luminaries enliven the story. The latter bios are of the six Nobelists and five Lasker Award winners, most of who had worked in the intramural programme. Others trained in, or were greatly influenced by, the programme. A polymath with catholic neuroscience interests, Rowland provides lucid descriptions of the research that led to these awards.

An added treat is a provocative timeline, listing the "landmark" neurological and neuroscience advances, throughout the world, every year, from 1950 to 2001. Rowland developed this by collating opinions of many awards committees and prominent neurologists and neuroscientists, most outside of the NINDS.

The author, who took a sabbatical leave from Columbia to work full time in Bethesda on this project, mulled over the question of who would want to read his book. He concluded that some would immediately look at the index to see if they were listed, but hoped "that general readers will find the biographies illuminating and informative. I hope that others will find out how NINDS works, and what it has achieved. In all of this, I have tried to write in a way that is

comprehensible to non scientists, but not offensive to scientists". Rowland succeeded. Not only is it a history of the NINDS, but of neuroscience over the past 50 years.

R B Daroff

Sleep and dreaming: scientific advances and reconsiderations

Edited by Edward F Pace-Schott, Mark Solms, Mark Blagrove, and Stevan Hamad. Published by Cambridge University Press, Cambridge, 2003, pp 350, £65.00 (hardback). ISBN 0-521-81044-2

How and why the brain produces dreams during sleep is a question that has intrigued researchers for many years. Accordingly it is timely that in the 50th anniversary year of the discovery of rapid eye movement (REM) sleep this book should seek to address the state of the art role of REM sleep in memory consolidation and dreaming, as well as wider consideration of the mechanisms of dreaming.

This book consists of five papers, each written by a world expert, that are discussed by over 75 eminent sleep researchers. These commentaries are followed by a rebuttal from the five authors. The book's format is entertaining and comprehensive, providing an efficient way to get a definitive picture of the current view.

The book covers the relationship between dreaming, brain physiology, and brain biochemistry. Specifically, the role of REM sleep is explored using modelling of conscious states. The nature of the relationship between REM sleep and dreaming is also examined in detail, with arguments put forward against the traditional memory consolidation role of REM sleep. Some of the book contents were first published in a special issue of the journal *Behavioural and Brain Science* in 2000, leaving one to speculate why there is need for this book. One advantage of the book is that more recent advances in the field, such as the discovery of the orexin system, are covered in a highly informative epilogue.

The book has a multidisciplinary approach, which is both its strength and weakness; in some cases the information is so detailed that the more general reader is likely to find it heavy going. The book has been designed to appeal to "students and researchers in neuroscience, cognitive science and psychology". In reality it is extremely detailed and is likely to appeal more to the specialist audience.

In summary, this is an authoritative text that covers all aspects of the production and function of dreaming in a novel format, and is likely to appeal to those wishing to gain deeper insight into this area of research.

M J Morrell