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References

- 1 Stone S, Lange LS. Syncope and sudden unexpected death attributed to carbamazepine in a 20 year old epileptic. *J Neurol Neurosurg Psychiatry* 1986;**49**:1460-1.
- 2 Meyrignac C, Berges C, Benhothman A, et al. Bradycardie sinusale et bloc sino-auriculaire induits par la carbamazepine. *Presse Med* 1983;**12**:577.
- 3 Blumhardt LD, Smith PEM, Owen L. Electrocardiographic accompaniments of temporal lobe seizures. *Lancet* 1986;i:1052-6.
- 4 Iani C, Fusco L, Faedda MT, Perugino U, Manfredi M. Cardiac frequency changes during absences and partial complex seizures. Second International Conference on Ambulatory Monitoring in Epilepsy and Sleep Disorders. Telfs, 21-25 January 1987.

Position sense in a damaged knee

Sir: I was intrigued to read Dr Swash's article, "Position sense in a damaged knee",¹ as my experience has been completely different. I underwent a double right meniscectomy by open arthrotomy some 30 years ago, after an injury followed by several episodes of locking and effusions. Recovery was uneventful, except for loss of about 10° of terminal flexion and discomfort in trying to squat.

Neurologically I had a 3 cm patch of paraesthesia over the antero-lateral tibial plateau, which, over the years, has dulled down to a curious mix of hypoesthesia, hypo- and hyper-algesia on direct testing, but otherwise is no longer noted (neglect? habituation? tolerance?). I have never had any instability, or problems in gait, using steps or other activities, in the light or dark.

I would think that the newer operations, leaving smaller scars, would inflict less damage. Perhaps there is an aging component? Less disturbance may occur when the joint and surrounding nerves are attacked at a younger age, or compensatory mechanisms may be rapidly established.

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Reference

- 1 Swash M. Position sense in a damaged knee. *J Neurol Neurosurg Psychiatry* 1986;**49**:100.

Pseudotumour cerebri with amiodarone

Sir: Fikkers, *et al* reported a case of pseudotumour cerebri felt to be induced by amiodarone.¹ However, in their case there were several medication changes at one time and therefore the exact relationship between the discontinuation of the amiodarone and the resolution of the pseudotumour could not be definitely established. We have recently had a remarkably similar case which we would like to report.

A 51 year old man had been treated for five months with gradually increasing doses of amiodarone for his refractory ventricular arrhythmias. The dose at the time of admission was 800 mg/day. He had also been taking diltiazem 360 mg/day, naproxen 1 gm/day, and isosorbide dinitrate 80 mg/day for several months prior to beginning the amiodarone therapy. On admission for atypical chest pain, he was noted to have a grade II papilloedema bilaterally which had not been noted on a routine neurologic consultation for tremor one month prior. The general physical examination showed moderate obesity, mild bibasilar rales and a mild resting and action tremor. The neurological examination was unremarkable except for the eyes. Electrocardiogram showed normal rate and rhythm, with a chronic right bundle branch block. Ocular examination revealed mild corneal deposits O.U. and the above mentioned papilloedema. Visual acuity and fields were normal.

C.T. scan of the brain with and without iodinated contrast was normal except for somewhat smaller ventricles than would be expected for the age of the patient. Magnetic resonance imaging of the head was normal. Lumbar puncture showed an opening pressure of 235 mm of water with the patient supine, mildly elevated protein (0.61 g/l), normal glucose (3.3 mmol/l), and 1 lymphocyte/mm³. Routine bacterial and TB cultures were negative. Routine blood and urine tests were normal. Because of the patient's continued complaints of tremor, restlessness, and insomnia, in addition to the close chronological association between the amiodarone therapy and onset of the pseudotremor, the amiodarone was discontinued and tocainide was substituted. The medications otherwise remained the same. Over the next month, serial taps revealed a gradual resolution of the increased ICP and the increased protein, beginning with a drop in pressure to 190 mm of water 5 days after discontinuing the amiodarone. At three month follow-up the papilloedema had resolved.

We agree with the previous authors that

amiodarone would appear to have caused the pseudotumour in both cases, again because of the development of the pseudotumour shortly after the onset of therapy and its resolution after it was discontinued. However, the implication is stronger in our patient since the amiodarone was the only medication changed.

Previous reviews of the neurological side effects of amiodarone have not reported this side effect of amiodarone therapy.² The visual side effects common to pseudotumour and as reported in Fikkers's case make awareness of this side effect vitally important to the Neurologic consultant.

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References

- 1 Fikkers B, Bogousslavsky J, Regli F, Glasson S. Pseudotumour cerebri with amiodarone. *J Neurol Neurosurg Psychiatry* 1986;**49**:606.
- 2 Nygaard TW, Sellers TD, Cook TS, DiMarco TP. Adverse reactions to antiarrhythmic drug therapy for ventricular arrhythmias. *JAMA* 1986;**256**:55-7.

Book reviews

Kindling 3. Edited by Juhn A Wada. (Pp 576; \$81.50.) New York: Raven Press, 1986.

As the title indicates, this volume is the third of its kind. The volumes are all edited by Juhn Wada and derive from conferences which took place in Canada in 1975, 1980 and 1985. This one contains 28 chapters in camera-ready format (with *Discussion* reported verbatim) and a 46 page kindling bibliography. What progress have the kindling fraternity made in the last five years?

As in the previous volumes there are novel tantalising findings that may hold the answer to the mystery. Is the depletion of calcium-binding protein in the dentate granule cells and their projection areas (described by Miller, Baimbridge and Mody) the crucial clue? Indeed the fascination of these volumes has been the sense of a detective thriller; who induced the epilepsy

and how? The forensic approach has provided numerous promising reports. These initially implicated the catecholamines (with noradrenaline the prime suspect). Subsequently GABA and the associated benzodiazepine receptor appeared to be involved. Various peptides have come under close scrutiny including the enkephalins and AVP. Now rumours concern the excitatory amino acids, both in terms of presynaptic release and post-synaptic receptors. However, reports from the scene of crime officer have caused confusion. The crucial change did not occur at the obvious site (that is, the site of the intervention producing electrical or chemical kindling, for example the amygdala) but at some distant synapse or synapses (for example in the hippocampus, cortex or substantia nigra). As with most detective mysteries the reader becomes intrigued or frustrated according to the logic or lack of clarity of the narrative. It must be confessed that some of the witnesses are making vague and evasive statements. The discussion sections keep the reader alert (who are Chuck and Pete and Mac? Will the imprecisely described experiments that they plan to do contribute to the solution to the mystery?) Let us hope that the next volume is the final one, and that all the threads are brought ingeniously together.

For those who wonder what purpose this fictional genre serves there is a very well argued chapter by Engel and Cahan on the relevance of kindling to human partial epilepsy.

BS MELDRUM

Tumors of the Skull Base. Extra- and intracranial surgery of skull base tumors. Edited by H Scheunemann, K Schurmann, J Halms. (Pp 256; DM148.00.) Berlin: Walter de Gruyter & Co, 1986.

This volume marks the deliberations at the Second International Meeting of the Skull Base Study Group. Neurosurgeons, otorhinolaryngologists, maxillofacial, plastic and reconstructive surgeons, as well as ophthalmologists and neuroradiologists, have contributed to the symposium and to this volume. Professor Schurmann in his preface has pointed out that no one specialist deals with lesions of the skull base, and it is only by a multidisciplinary approach that advances in understanding and treatment will be made. With improved imaging techniques (CT Scan and magnetic resonance imaging) and new micro-surgical techniques

as well as laser and CUSA equipment, many conditions previously believed to be inoperable have been treated successfully by a multi-disciplinary surgical team. In concept a skull base study group is most attractive; their first meeting was in 1973. This report is of the meeting in Mainz 1984.

The book is divided into sections: (1) Anatomy and neuroradiology of the skull base, (2) Anterior skull base tumours, (3) Middle and posterior skull base tumours.

One has the impression that the actual symposium must have been a most profitable occasion for all those involved. Sadly, the book will provide little new information for those who would wish to benefit from the combined deliberations at the symposium. Many of the "chapters" are less than 1,000 words, and do not really tell the reader how to perform the operation (doubtless this was explained in great detail during the symposium). There are a few chapters, however, which stand out above the mists of mediocrity. The anatomical chapter "Topographical anatomy of the skull base and adjacent tissues" is in the usual exquisite detail which we have come to expect from Lang's writings. The contribution by Sekhar and Samii on "Petrioclivar and medial tentorial meningiomas" is useful. Individual case reports, unless they provide a major technical lesson, do not contribute to this volume. Three hundred words describing a fairly straightforward case of bi-frontal meningioma or five hundred words describing surgical reconstruction of the eustachian tube or blindness caused by Schwannoma of the olfactory nerve do not contribute materially to the book. The preface implies a wide international contribution to the Society; in fact 33 of the 56 contributors are German, with strong supporting roles by Dutch and French.

In summary, I am sure that the International Study Group is the way forward in understanding and treatment of the diseases of the skull base. Unfortunately, I do not believe that this volume lives up to the promise in the preface.

H ALAN CROCKARD

Progress in Neuropathology Vol 6. Edited by Harry M Zimmerman. (Pp 304; \$98.50.) New York: Raven Press, 1986.

Given that the stated aim of this sixth and latest volume in the progress in neuropathology series is the presentation of new

concepts and information in neuropathology, there should be ample excitations for even the most jaded of pathologists. Unfortunately the result is far duller than one might have expected. It is not the assembled authorship, who have ample claims to distinction. Rather it is the choice of subjects which disappoints.

Once again we are presented with the neuropathology of Parkinson-dementia complex of Guam, now rapidly dwindling incidence, the immunology of EAE, the immunocytochemistry of neurofibrillary tangles, and glioma tissue culture. Many of the detailed and meticulously referenced accounts would sit more comfortably in standard textbooks than in a volume of recent advances. Lost opportunities abound: advances in the morphometry and neurochemistry of Alzheimer's disease, neurotransmitter research, the fast expanding peroxisomal diseases, and the increasing influence of neuro-imaging techniques and gene probes which will profoundly alter the practice of neuropathology, to name but a few.

There are of course some bright spots: exciting and thought provoking essays on glucocorticoids and their role in the pathogenesis of hippocampal lesions, the influence of cytoplasmic microtubules in cell proliferation, and an essay on tumour invasion and metastasis. There is also a timely critique of the so-called neuron-specific enolase now found widely in extra-CNS tissues, a warning to us all in this age of immunocytochemistry.

The muscle chapters are particularly disappointing. Congenital myopathies take the form of a catalogue, including some excessive rarities, yet ignoring entirely the important and burgeoning subject of mitochondrial cytopathy. Previously unpublished, and (as it turns out) non-specific, muscle changes in fatal catatonias provide the coda to this book.

All in all, I doubt whether the individual purchaser will be attracted by the relatively high price and poor prose style. Literature seekers may request it from libraries, but I doubt whether they will be encouraged to stray far from their original objective.

B N HARDING

Emotions — Neuronal and Chemical Control. Edited by Y. Oomura. (Pp 446; £105.10.) Basel: S Karger AG, 1986.

Occasionally, genuine surprise accompanies