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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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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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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