Matters arising

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Position sense in a damaged knee

Sir: I was intrigued to read Dr Swash’s arti-
cle, “Position sense in a damaged knee”, as
my experience has been completely different.
I underwent a double right meniscectomy by
open arthroscopy some 30 years ago, after an
injury followed by several episodes of lock-
ing 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-
thesis and hyper-algesia on direct testing,
but otherwise is no longer noted (neglect?
habitation? 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 dam-
age. 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 pseudo-
tumour cerebri felt to be induced by ami-
odarone.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 admis-
sion 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 atyp-
ical chest pain, he was noted to have a
grade II papilloedema bilaterally which had
not been noted on a routine neurologic con-
sultation for tремor one month prior. The
general physical examination showed mod-
erate obesity, mild bibasilar rales and a mild
resting and action tremor. The neurologi-
ical 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 pres-
sure 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 pseudo-
tumour, the amiodarone was discontinued
and tocamidine was substituted. The medica-
tions 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 pseudo-
tumour 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 Fikker’s case make aware-
ness of this side effect vitally important to
the Neurologic consultant.

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Book reviews

Kindling 3. Edited by Juhn A Wada. (Pp

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 1983. This one contains 28 chapters in
camera-ready format (with Discussion
reported verbatim) and a 46 page kindling
bibliography. What progress have the kin-
dling 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 fascina-
tion of these volumes has been the sense of
a detective thriller; who induced the epilepsy...