degrees C) when compared with our other patients at the time of apnoea testing.

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van Donselaar et al reply:

We completely agree with the comments made by Belsh and Schiffman. In the discussion-section of our paper we stated that the low pCO2 levels at the onset of the apnoea test might explain the insufficient levels after 10 minutes of apnoea. We also agree with their recommendation to adjust the minute volume prior to disconnection if the pCO2 is rather low. In a recent article for the journal of the Dutch Medical Association,1 we advised the following:

(1) if the pCO2 ≥ 5-0 kPa (38 mmHg)  
—ventilante with 100% O2 for 10 minutes  
—disconnect the patient for 14 minutes while giving O2 via an endotracheal tube at a rate of 6 litres/min  
—after drawing blood for blood gas determination re-connect the venti-

(2) if the pCO2 < 5-0 kPa (38 mmHg)  
—ventilante with 100% O2 for 5 minutes

continue ventilating with 100% O2 with a halved volume for 15 minutes

—disconnect the patient for 14 minutes while giving O2 via an endotracheal tube at a rate of 6 litres/min

—after drawing blood for blood gas determination re-connect the ventila-

With this method, the pCO2 will have risen to 7-98 kPa or higher in most patients, while adequate oxygenation is secured.2,3 The test must be terminated in case of ven-

tricular arrhythmias of hypotension. In our opinion blood gas determination at the end of the apnoea test is mandatory to see whether the pCO2 has reached the target value providing supramaximal stimulation of the respiratory centre. For patients with chronic lung disease we refer to the article of Rohling.4

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Sino-atrial block provoked by carbamazepine

Sir: Stone and Lange1 have reported in your Journal the occurrence of ventricular asystole followed by syncope and death in a patient treated with carbamazepine for temporal lobe seizures. They also mention the occurrence of sinus bradycardia due to carbamazepine.

The following case confirms that car-

bamazepine may cause sino-atrial block. A 54 year old female suffering since the age of 32 from complex-partial seizures with automatisms with a frequency of about 3-5 per day, had been taking carbamazepine 1200 mg/day for one year with a plasma concentration of 6-3-9-0 µg/ml. The epileptic nature of seizures was documented by simultaneous ambulatory EEG and ECG monitoring; there were no secondary cardiac arrhythmias during the epileptic attack. She was hospitalised after falling from a small ladder without loss of consciousness while housekeeping. On admission, heart rate was 36 per minute and ECG showed rare, iso-

lated monomorphous ventricular ectopic beats. Carbamazepine was discontinued. Pulse rate remained around 40 for a few hours and went back to normal the days after. During the following months the patient was unsuccessfully treated with phentoin, clonazepam and phenobarbital in combination.

Carbamazepine treatment was resumed after the initial dosage of 150 mg, gradually increased to 300 and then 600 mg over a 4-month period, under weekly ECG con-


trols. There was a considerable decrease in seizures. The patient had been taking 600 mg for 15 days when ECG evidenced a 2:1 sino-atrial block. Plasma concentration was not obtained. Discontinuation of the drug resulted in the disappearance of the arrhyth-

mia in 24 hours. Follow-up examinations on the 3rd, 7th and 14th day did not show any conduction disorder.

Stone and Lange collected nine cases of conduction disorder due to carbamazepine. We may add the present case and a case of sino-atrial block reported by Meynigard et al.2 Given the wide application of this drug the risk of cardiac complications seems low, and carbamazepine remains an excellent anticonvulsant medication.

Blumhardt et al3 have demonstrated a simultaneous EEG and ECG monitoring that temporal lobe seizures are associated with increased heart rate in 24 out of 26 patients. In a series of 16 partial complex epileptic patients monitored in our laboratory4 we have observed ictal tachycardia in 13; in two patients there was ictal bradycardia, starting 6-8 seconds from the beginning of the attack and attaining 15% and 53% of the basal frequency. Blumhardt et al3 actually suggest that antiepileptic medication may protect against the risk of sudden death in epileptic patients. The relationship between epilepsy, drugs and the heart must be therefore evaluated in the single patient. However, the need for cardiol-

ogic examination during carbamazepine prescription, emphasised by Stone and Lange,4 cannot be over stressed. The use of the drug should be cautious in patients with sick sinus syndrome or blocks at any level. Special attention must be given to elderly patients, who more frequently suffer from these diseases.

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Matters arising
Matters arising

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Ambulatory Monitoring in Epilepsy and

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- 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 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 diltilazem 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 con-
sultation for tremor one month prior.
The general physical examination showed
moderate obesity, mild bibasilar rales and a
mild resting and action tremor. The neurologi-
al 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
lymphocytes/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 tocinamide 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
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

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