van Donselaar et al. report:

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 discontinuation 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)
   --preventilate 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 ventilator.
2) If the pCO2 < 5·0 kPa (38 mmHg)
   --preventilate 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 ventilator.

With this method, the pCO2 will have risen to 7·98 kPa or higher in most patients, while adequate oxygenation is secured.2-4 The test must be terminated in case of ventricular 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

References
1 Van Donselaar CA, Meerwaldt JD, and Van-Gijn J. J Neurol Neurosurg Psychiatry 1986;49:1071-3.
7 Belsh JM, Schiffman PL, Blatt R. Apneea testing for the determination of death by neurologic criteria (Brain Death). New Jersey Medicine 1986;83:593.

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 carbamazepine 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, isolated monomorphic ventricular ectopic beats. Carbamazepine was discontinued. Pulse rate remained around 40 for a few hours and went back to normal the day after. During the following months the patient was unsuccessfully treated with phenytoin, 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 an 4-month period, under weekly ECG controls. There was a considerable decrease in seizures. The patient had been taking 600 mg 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 arrhythmia 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 Meyrink and al.2 Given the wide application of this drug the risk of cardiac complications seems likely and carbamazepine remains an excellent anticonvulsant medication.

Blumhardt et al.3 have demonstrated 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 or epileptic patients monitored in our laboratory 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 al.3 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 cardiologic examination during carbamazepine prescription, emphasised by Stone and Lange,1 cannot be overestimated. 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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References


Position sense in a damaged knee

SIR: I was intrigued to read Dr Swash’s article, “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 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 bi-basilar 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 pseudotumour, 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 Fikker’s case make awareness of this side effect vitally important to the Neurologic consultant.

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References


Book reviews


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...