

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

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References

- 1 Van Donselaar CA, Meerwaldt JD and Van Gijn J. *J Neurol Neurosurg Psychiatry* 1986;**49**:1071-3.
- 2 Milhaud A, Riboulot M, Gayet H. Disconnecting tests and oxygen uptake in the diagnosis of brain death. *Ann NY Acad Sci* 1978;**315**:241-50.
- 3 Schafer JA, Caronna JJ. Duration of apnea needed to confirm brain death. *Neurology* 1978;**28**:661-6.
- 4 Rowland TW, Donnelly JH, Jackson AH. Apnea documentation for determination of brain death in children. *Pediatrics* 1984;**74**:505-8.
- 5 Special Communication. Guidelines for the determination of death. *JAMA* 1981;**264**:2184-6.
- 6 Belsh JM. Brain death. Policy at Middlesex General-University Hospital. *J Med Soc NJ* 1985;**82**:199-205.
- 7 Belsh JM, Schiffman PL, Blatt R. Apnea testing for the determination of death by neurologic criteria (Brain Death). *New Jersey Medicine* 1986;**83**:593.
- 8 Belsh JM, Blatt R, Schiffman PL. Apnea testing in brain death. *Arch Intern Med* 1986;**146**:2385-8.

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)
 - 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.^{2,3} 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, Van Gijn J. Het vaststellen van de afwezigheid van spontane ademhaling bij hersendood. *Ned Tijdschr. Geneesk.* 1987;**2**:65-67.
- 2 Van Donselaar CA, Meerwaldt JD, Van Gijn J. Apnea testing to confirm brain death in clinical practice. *J Neurol Neurosurg Psychiatry* 1986; **49**:1071-3.
- 3 Schafer JA, Caronna JJ. Duration of apnea needed to confirm brain death. *Neurology* 1978;**28**:661-6.
- 4 Rohling R, Wagner W, Mühlberg J, Link J, Scholle J, Rosenow D. Apnea test: pitfalls and correct handling. *Trans Proc* 1986;**18**:388-91.

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 monomorphous 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 at the initial dosage of 150 mg, gradually increased to 300 and then 600 mg over a 4-month period, under weekly ECG controls. 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 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 Meyrignac *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 al*³ have demonstrated by 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 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 overstressed. 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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