NON-FATAL PARADOXICAL AIR EMBOLISM

Sir,—Cerebral air emboli are usually fatal or may cause serious neurological conditions. The risk of such an embolus during insertion of a central venous catheter (CVC) is well recognized [1]. We report a patient in whom an embolus occurred when a three-way tap on a CVC became accidentally disconnected.

The patient was a 33-yr-old man with acute myeloid leukaemia who was receiving treatment via a permanently located CVC in the right internal jugular vein. The leukaemia was in good remission and the patient was cutting the lawn when he suddenly felt unwell, with sweating, dizziness and palpitations. The patient’s wife heard a buzzing sound under his shirt and thought it was a bee. He then collapsed and had convulsions accompanied by blood streaming from the CVC.

On reaching the casualty department, it was impossible to establish verbal contact with the patient. He had flexion spasms of all extremities every 5–10 s, more marked on the left. Both pupils were dilated moderately, equal and reactive to light. Bilateral foot clonus was observed and his heart rate was 140 beat min⁻¹. Arterial blood-gas analysis revealed slight hypoxia with $P_{A}O_2$ 8.6 kPa, and $S_{a}O_2$ 90%.

The patient was treated with oxygen, a left lateral tilt, Diazemuls 45 mg i.v. and phenobarbitone 200 mg i.v. The seizures stopped, but no response could be obtained from the patient. After observation for 5 h he awoke suddenly and was neurologically completely intact.

Venous air embolism with CVC catheters can occur when a subject is in an upright position. This has been demonstrated in 30–40% of patients who undergo neurosurgery in the sitting position [1]. The surest method of determining the diagnosis is by Doppler ultrasound [2]. Our patient presented with the signs of a cerebrally-triggered attack: air may have entered the cerebral circulation, possibly via a patent foramen ovale, which is present in 20–30% of the population. Previous instances of paradoxical air embolus via the foramen ovale have been described [3,4], and a pressure gradient of only 4 mm Hg is necessary to produce a right-to-left intracardiac shunt [5]. It has been shown in animals that the presence of air alone in the right atrium is enough to cause such a shunt [6], and this is seen especially when a bolus of air is involved [7].

Cerebral air emboli often result in serious neurological conditions [4], but in this patient there were no sequelae. Should brain damage occur, the treatment is limited. One patient has been described [8] in whom treatment with benzodiazepine-induced coma, slight hypothermia and hyperventilation were thought to have been effective. Hyperbaric oxygen treatment has also been described [9]. In the present patient complete recovery occurred without treatment.

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REFERENCES

SEDATION FOR ENDOSCOPY

Sir,—We note with concern the paper by Boldy and co-workers on sedation for endoscopy [1]. In the results section, the authors state that “There were no cardiorespiratory problems in either group.” There is no mention of any monitoring used in this study or at what times measurements or observations were made.

Cardiorespiratory changes during sedation for endoscopy are well known. Several studies have shown ECG changes during sedation and insertion of an endoscope [2–5], and significant reductions in oxygen tension have been demonstrated after introduction of the endoscope alone, after sedation with a benzodiazepine and after a combination of benzodiazepine and opioid [6–11]. Midazolam alone has been shown to cause marked respiratory depression [12].

We have completed a study of cardiorespiratory changes in 20 patients undergoing prolonged endoscopy. Data were recorded continuously by an Atari 1040 ST micro-computer, from ECG lead CM5, a pulse oximeter (Ohmeda Biox 3700), and a non-invasive arterial pressure monitor (Datascope). Sedation was provided by pethidine 25–50 mg and midazolam titrated to effect (mean dose 8 mg). The study commenced before administration of the sedative and continued for the first 1 h of recovery.

Oxygen saturation decreased in all patients (min 82%, SEM 12.5%), remained so for the duration of the examination and persisted into the recovery period. At the end of the study, saturation had not returned to baseline in 11 patients. An $S_{a}O_2$
of 42% was recorded in one patient. Sixteen of the 20 patients developed tachycardia. Ten patients developed supraventricular ectopic beats, ventricular ectopic beats or both. ECG changes resolved during the recovery period. A significant correlation was found between the occurrence of S-T segment depression and hypoxia (r = 0.818, P < 0.00005). No correlation was found between S-T segment depression and arterial pressure, heart rate or rate-pressure product.

From the results of our study and those of others, cardiorespiratory monitoring would appear to be mandatory during upper gastrointestinal endoscopy, especially if opioid analgesics are administered, hypoxia exists already or the patient is in a high risk group.

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REFERENCES