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 \( P_{O_2} \) 8.6 kPa, and \( S_{O_2} \) 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 barbiturate-induced coma, slight hypothermia and hyper-ventilation 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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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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Sir,—We read with concern the paper by Boldy and colleagues [1], which seems to highlight much of the misunderstanding concerning the use of i.v. sedation. The authors do not provide details of patient weight, making it difficult to be precise concerning doses used. If we assume that the mean male weight was approximately 70 kg, the mean dose of midazolam used in males was 0.16 mg kg⁻¹. The manufacturer's recommended dose is 0.07 mg kg⁻¹ for sedation during endoscopy—a dose found to be satisfactory by the second trial from the Hammersmith group [2] (not quoted by Boldy). As midazolam has approximately twice the hypnotic activity of diazepam, it seems unreasonable to compare diazepam 0.15 mg kg⁻¹ with midazolam 0.16 mg kg⁻¹, and hardly surprising that, after endoscopy, patients were more sedated after midazolam.

Five milligrams is the maximum dose of midazolam we use, as the average patient is too profoundly sedated to be cooperative after being given midazolam 10 mg i.v. Bell and colleagues [3,4] showed significant decreases in oxygen saturation (to less than 80%) during sedation with a benzodiazepine, exacerbated by passage of the gastroscope, and although they used larger doses (means of 6.7 and 6.3 mg) than we would recommend, these were considerably short of the doses of midazolam used by Boldy and colleagues. The addition of opioids to benzodiazepines compounds respiratory depression and hypoxia [5]. We believe that opioid supplementation has no place in sedation for routine upper gastrointestinal endoscopy.

As the peak effect of benzodiazepines given as a single i.v. injection occurs after 2–3 min, “titration” by injecting to a given endpoint cannot take this into account unless this time period is observed between each incremental dose. The pharmacokinetics of midazolam make it better suited than diazepam to sedation for endoscopy and its superior amnesic properties provide an added clinical benefit. The recent availability of flumazenil, a specific benzodiazepine antagonist, obviates Boldy's assertions that midazolam-induced amnesia is a disadvantage. Antagonism of midazolam 5 mg with flumazenil 0.5 mg i.v. prevents further amnesia, reverses sedation and returns psychomotor variables to normal within 1 min [6]. In all published trials, there has been no clinically significant residual sedation caused by the disparity between the half-lives of the two drugs (57 min cf. 1.3–2.2 h). Of course, when excessive doses of midazolam are used in individual patients, this possibility may be increased.

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