Correspondence

Air embolism during liver transplantation

To the Editor:

Liver transplantation is being done with increasing frequency. Although the anaesthetic considerations have been reviewed, the possibility of air embolism has not been mentioned.^{1,2} We recently looked after a patient whom we believe suffered an air embolus during the release of the clamps from the portal vein and the suprahepatic inferior vena cava.

A 29-year-old woman was undergoing liver transplantation for terminal chronic active hepatitis. Anaesthesia was induced with fentanyl, thiopentone and succinylcholine and maintained with nitrous oxide 50 per cent in oxygen, fentanyl, isoflurane and pancuronium. Due to technical problems, we were unable to establish a central venous catheter other than a large bore, short (2.7 cm) #14 gauge catheter in the right external jugular vein. At the time the clamps were removed from the portal vein and the suprahepatic inferior vena cava (IVC), the patient became acutely cyanosed, tachycardic and hypotensive. For approximately 30 minutes she remained in this state, despite the withdrawal of nitrous oxide and isoflurane and the addition of intravenous calcium chloride, dopamine and dobutamine as well as positive end expiratory pressure. Fifteen minutes after the start of this acute deterioration the serum potassium was $4.6 \text{ mmol} \cdot \text{l}^{-1}$ (normal $3.5\text{--}5.0 \text{ mmol} \cdot \text{l}^{-1}$) and arterial blood gases on F_1O_2 1.0 were pO_2 25 mmHg, pCO_2 55 mmHg, pH 7.22, bicarbonate $22 \text{ mmol} \cdot \text{l}^{-1}$ and base excess $-7 \text{ mmol} \cdot \text{l}^{-1}$. The serum calcium remained unchanged from values obtained prior to the crisis at $1.86 \text{ mmol} \cdot \text{l}^{-1}$ (normal $2.12\text{--}2.62 \text{ mmol} \cdot \text{l}^{-1}$). The CVP which had previously been $10 \text{ cm H}_2\text{O}$ rose to greater than $35 \text{ cm H}_2\text{O}$. As we did not think of air embolism until this crisis had nearly completely resolved and because the chest was prepared and draped up to the fourth intercostal space, we were unable to hear any of the characteristic cardiac signs

of an air embolus. The patient returned to her previous condition during the following half hour. No air was aspirated from the external jugular venous catheter. On awakening, she had no neurological deficits and made a good recovery.

In order to reduce the amount of perfusate flushed into the systemic circulation, the liver is flushed with saline. This enters through the partially completed portal venous anastomosis and exits through the infrahepatic IVC from which the clamp is temporarily removed and then reapplied. The portal vein anastomosis is completed and the clamps are removed from the already anastomosed suprahepatic IVC and the portal vein. The anastomosis of the infrahepatic IVC is then completed. In this case the clamp was inadvertently not reapplied to the infrahepatic IVC. When the suprahepatic IVC and the portal vein were unclamped, we believe that air was entrained into the venous system.

Acute cardiac depression does occur with washing out of the perfusate.³ This may be related to acute hyperkalemia or hypocalcemia. The rapid termination of the above reported event and the lack of change in the electrolytes suggests that this was the case. While we obviously have only circumstantial evidence to support our hypothesis, we believe that the risk of air embolism is real and that anaesthetists and surgeons should be cognizant of this possibility so that appropriate monitoring and therapeutic techniques are available. We routinely monitor right atrial, pulmonary artery and pulmonary capillary wedge pressures, end-tidal carbon dioxide content, and heart sounds by means of an oesophageal stethoscope. A precordial doppler is not practical because the patient is prepared and draped to above the nipple line in case a sternal split is necessary for adequate exposure.

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Bradycardia induced by repeated dose of succinylcholine

To the Editor:

We read with interest the article by Nigrovic¹ concerning his hypothesis on the mechanism of adverse haemodynamic reactions to succinylcholine. When Nigrovic discusses bradycardia following a repeated dose of succinylcholine, he points out the study of Mathias *et al.*² who concluded that pre-curarisation with a non-depolarizing drug such as d-tubocurarine can prevent this phenomenon, an opinion usually accepted by the anaesthetist.³

We would like to emphasize that this prevention of bradycardia is not the case. We recently did a double blind study which included 24 women, ASA physical status I, in which we examined the efficacy of diazepam in preventing bradycardias induced by a second dose of succinylcholine (1 mg·kg⁻¹). The patients were randomly divided into three groups of eight patients each according to the pre-treatments received: G-I, intravenous diazepam (0.05 mg·kg⁻¹), G-II, d-tubocurarine (0.06 mg·kg⁻¹) and G-III, placebo (NaCl 0.9 per cent). Four cases of sinus arrest (3.2 to 14 sec) (G-I = 1, G-II = 2, G-III = 1) and two of bradycardia of less than 50 beats per minute (in Group III) followed the reinjection of succinylcholine. In fact, succinylcholine reinjection is a dangerous procedure and neither

intravenous d-tubocurarine nor intravenous diazepam provide adequate protection.

In view of the literature on this particular side effect of succinylcholine reinjection, we feel that there is at present no protection against this potentially harmful side-effect, except for the possible protective effect of glycopyrrolate.⁴

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REPLY

Thank you for the opportunity to answer the interesting letter of Drs. Martin and Léna.

When commenting on the explanation of Mathias *et al.*¹ about the mechanism by which non-depolarizing muscle relaxants alter cardiac effects of a repeated dose of succinylcholine, I pointed out² its shortcomings. It is my belief that this explanation cannot be the sole answer for the clinical observations of bradycardia or bradyarrhythmia.

On the other hand, published clinical reports, e.g., McLeskey *et al.*,³ and the data reported by Martin and Léna, show that "precuarization" does not reliably protect against the adverse cardiovascular effects seen on repeated administration of succinylcholine. These findings, however, can be easily interpreted in terms of the proposed hypothesis.² Inactivation of the nicotinic receptors on sympathetic terminals by (a) the previous administration of succinylcholine (see Fig. 2 in²) and/or (b) precuarization, leaves the succinylcholine-induced activation of the cardiac muscarinic receptors, with its