somewhat like a soap serving to saponify the agent and increase blood solubility. Concurrently the local concentration of the agent is reduced lowering the maximum tissue levels as well as the potential for aggregation of the agent into macrobubbles. This is a similar system to propofol when emulsified in intralipid.

It is my opinion that the case reports and studies involving the toxicity of the iv injection of pure volatile agents are better explained as a pathophysiological effect of the volatiles on the tissues rather than a physical effect of an embolism. In all the reports there is a period of initial recovery, followed by deterioration hours to days later. Clinical manifestations include progressive pulmonary shunting, elevated pulmonary vascular resistance, shock, pulmonary oedema and haemorrhage followed by systemic shock and multisystem organ failure. This is the chronology and clinical description of an ARDS as opposed to a massive embolism. The subpleural distribution of the pulmonary infarcts seen in the reports may be due to the microembolization of the volatile agent in liquid form. Were a gas embolism the main cause of pathophysiology one would expect severe initial symptoms that would improve with time.

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REFERENCES

1 *Stemp LI.* Intravenous injection of liquid halothane. Anesth Analg 1990; 70: 568.
2 *Dwyer R, Coppel DL.* Intravenous injection of liquid halothane. Anesth Analg 1989; 69: 250–5.
3 *Biber B, Martner J, Werner O.* Halothane by the I.V. route in experimental animals. Acta Anaesthesiol Scand 1982; 26: 658–9.
4 *Kawamoto M, Suzuki N, Takasaki M.* Acute pulmonary edema after intravenous liquid halothane in dogs. Anesth Analg 1992; 74: 747–52.
5 *Sandison JW, Sivapragasam S, Hayes JA, Woo-Ming MO.* An experimental study of pulmonary damage associated with intravenous injection of halothane in dogs. Br J Anaesth 1970; 42: 419–24.

Phaeochromocytoma and calcium channel block

To the Editor:

I read with interest the recent publication of Munro *et al.* entitled: "Calcium channel blockade and uncontrolled blood pressure during phaeochromocytoma surgery."[1] The authors present a case in which diltiazem proved to be ineffective in controlling blood pressure during resection of a phaeochromocytoma. While I agree that diltiazem is not a particularly good choice in this setting, I would suggest that the authors should not conclude that all calcium channel antagonists are ineffective just because diltiazem failed. The cardiovascular responses of the calcium channel antagonists are quite varied. Diltiazem should not be expected to be effective in this setting as its primary cardiovascular effects include negative inotropic and dromotropic activity.

Proye *et al.* have demonstrated the efficacy of a different calcium channel blocker, nicardipine, in controlling blood pressure in a series of ten patients with phaeochromocytoma.[2] Nicardipine was administered orally (60 to 120 mg · 24 hr^{-1}) preoperatively to control blood pressure and then intravenously (2.5 to 7.5 mg · kg^{-1} · min^{-1}) during surgical resection. Although the authors theorized that calcium channel blockers might block the release of catecholamines from the tumour, considerable elevations in both epinephrine and norepinephrine levels occurred intraoperatively, suggesting that nicardipine's effect was related to its peripheral vasodilatory properties and not a blockade of catecholamine release.

While I would not argue that the usual practice includes alpha antagonists and nitroprusside, newer agents such as nicardipine may be effective. With the addition of newer calcium channel blockers with very different cardiovascular effects, it seems particularly important to differentiate between the different agents available. Unlike diltiazem, nicardipine's actions include primarily direct arterial vasodilatation with limited effects on inotropic or chronotropic function.

Although the clinical experience with nicardipine is still somewhat limited, its initial use in anaesthetic practice suggests that it will be an effective and safe agent to control perioperative blood pressure in many different situations. It may have several applications in anaesthetic practice as well as certain advantages over more commonly used agents such as sodium nitroprusside and beta adrenergic antagonists.[3-4]

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REFERENCES

1 *Munro J, Hurlbert BJ, Hill GE.* Calcium channel blockade and uncontrolled blood pressure during phaeochromocytoma surgery. Can J Anaesth 1995; 42: 228–30.
2 *Poeye C, Thevenin P, Cecat P, et al.* Exclusive use of calcium channel blockers in preoperative and intraoperative control of pheochromocytomas: hemodynamics and free catecholamine assays in ten consecutive patients. Surgery 1989; 106: 1149–54.
3 *Tobias JD.* Nicardipine: applications in clinical anesthesia. J Clin Anesth (in press).
4 *Tobias JD, Lowe S, Deshpande JK.* Nicardipine: perioperative applications in children. Paediatric Anaesthesia (in press).