extensive block cannot be excluded when such large volumes of local anaesthetic solution are employed. Again, this has not been a problem and we believe that it is the probable malplacement of the catheter in the antero-lateral space and resultant poor block that provides some protection against such extensive blocks. Finally, we would caution against the use of this technique in patients who have experienced accidental dural puncture during the placement of the epidural catheter. There is the potential for transdural passage of the injected chloroprocaine and decreased neurotoxicity of the new preparations has not yet been affirmed.

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Propofol bradycardia

To the Editor:

We read with interest the recently published review of reflex bradycardia during surgery.¹ Recent reports implicating propofol with bradycardia suggested to us the following comments.

Propofol is a new alkyl phenol intravenous anaesthetic agent now available in an aqueous solution of ten per cent soybean oil, 2.25 per cent glycerol and 1.2 per cent purified egg phosphatide. In contrast to other IV anaesthetics, propofol does not depress baroreflex sensitivity directly but may produce an increase in vagal tone and/or a decrease in sympathetic tone by central mechanisms.² Severe bradycardia and arrhythmias have been described after propofol,^{3–6} either with fentanyl and its congeners,^{4,5} or with succinylcholine,³ vecuronium^{4,5} or neostigmine.⁶ All these drugs may be responsible for cholinergic effects and it would seem advisable to recommend that an anticholinergic drug be administered whenever propofol is given in combination with potential cholinergic agents or betablocking drugs.⁶ Mechanisms of the bradycardia with propofol remain obscure. They may be produced by a decrease in sensitivity of the baroreflex control of the heart or by a direct effect on sinus

activity.⁷ The role of rapid injection of the solvent vehicle is also possible. In experimental fat embolism, bradycardia and even atrio-ventricular block were observed and associated with right coronary ischaemia.⁸ Severe intralipid-induced transient sinus bradycardia has been observed.⁹ Furthermore, in a dog model of ischaemia, induced by partial occlusion of the left inter-ventricular coronary artery, IV intralipid decreased regional myocardial blood flow in the subendocardial and subepicardial layers,¹⁰ perhaps because of increased blood viscosity.

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REPLY

Dr. Freysz et al. rightly point out that propofol (Diprivan®) may be added to the list of anaesthetic drugs which may result in bradyarrhythmias, particularly when used in association with cholinergic stimulating drugs like succinylcholine or neostigmine. Baraka¹ has suggested that propofol may lack the central vagolytic properties which the barbiturates possess. Based on the study of a pharmacologically denervated dog, Colson et al.² make the stronger suggestion that propofol may slow the heart directly. The occasional bradyarrhythmias experienced with propofol suggest that it would generally be wise to use anticholinergic premedication where fast heart rates are not problematic, especially in vagally stimulating procedures such as laparoscopy.³

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Intra-arterial verapamil to reverse acute ischaemia of the hand after radial artery cannulation

To the Editor:

A 67-yr-old male presented for left lower lobectomy for lung cancer after a history of progressive dyspnoea, cough and haemoptysis of six months' duration. Premorbid history noted heavy cigarette and alcohol use as well as Raynaud's phenomena involving both hands. Preoperative evaluation included a modified Allen's test and Porch test¹ using pulse oximetry to assess the collateral circulation of the hand. Both tests were negative. Intraoperatively, an Arrow #22 percutaneous catheter was inserted into the left radial artery atraumatically. Upon arrival in the ICU postoperatively, the patient developed acute ischaemia of the left hand shown by decreased arterial saturation involving all digits. The radial arterial line was aspirated and no air or clot was noted. Heparin 100 U was initially flushed through the radial arterial line with

no improvement. Verapamil 1 mg diluted in 3 ml normal saline was flushed through the arterial-catheter. Within five minutes, reactive hyperaemia was noted which correlated with concomitant increases in arterial saturation of the involved digits. It was concluded that arterial spasm was the likely cause of the ischaemia in view of the history of Raynaud's phenomena. This case demonstrates the effectiveness of intra-arterial verapamil in the treatment of acute arterial ischaemia of the hand secondary to Raynaud's phenomena. It also demonstrates the ability of the PORCH test in detecting but not predicting ischaemia.

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Eligibility of Canadians for the ASA overseas teaching program

To the Editor:

When the Overseas Teaching Program (OTP) sponsored by The American Society of Anesthesiologists (ASA) and the Foundation for Anesthesia Education and Research (FAER) was inaugurated in January, 1990,¹ criteria of eligibility for those wishing to serve as volunteers teaching anaesthesia in an undeveloped country were described as including membership in the ASA, certification by the American Board of Anesthesiology (ABA), and residence in the USA. These criteria need amplification. Applicants do have to be members of the ASA, but that includes affiliate members. Also, certification as a qualified specialist in anaesthesia can be either by the ABA or by an equivalent certifying body. Finally, residence must be in North America, not just in the USA. Many Canadian anaesthetists are thus eligible to serve as OTP volunteers.

The what, why, how and where of OTP are described in the 1990 announcement of the program.¹ Suffice it to say that OTP's objective is to contribute to the quality of patient care in developing countries by increasing the