Sir,—Dr Della Pupa questions the mechanism whereby reversal of hypotension can be achieved at the end of surgery when extensive neural block is still in effect. Our usual approach is to discontinue low-dose adrenaline slowly and change to epididrine with or without a fluid challenge.

This enables systolic pressure to be maintained typically in the range 100–120 mm Hg in the post-anaesthesia care unit. Epididrine has both alpha and beta effects, so that arterial pressure can be maintained without causing tachycardia.

Epididrine and fluid as a means of maintaining circulatory stability for patients with high extradural block has been an accepted practice for some time, in both surgical [1] and obstetric patients.

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REFERENCE

PROPOFOL IN ACUTE INTERMITTENT PORPHYRIA

Sir,—Acute intermittent porphyria (AIP) is the most common of the acute hepatic porphyrias occurring in the U.K. The condition arises from decreased activity of the enzyme, porphobilinogen deaminase, leading to the characteristic increase in the concentration of precursors before the block in the metabolic pathway. In common with other acute porphyrias, it is possible to precipitate an acute attack by administration of some drugs, and of particular concern to the anaesthetist is the choice of an appropriate i.v. induction agent. Thiopentone, methohexitone [1], Althesin [2], etomidate [3] and ketamine [4] have been reported to be porphyrinogenic. Most reports of the use of propofol involved patients with variegate porphyria (VP). We could find only one case report of the use of propofol and AIP [5]. Therefore, we would like to report the case of a patient with AIP in whom propofol was used.

A 26-yr-old female with a documented history of AIP since 1974 who underwent general anaesthesia using propofol as the induction agent [5]. Mitterschiffthaler and colleagues [5] reported the case of a 43-yr-old female who underwent general anaesthesia using propofol as the induction agent, without subsequent biochemical or clinical adverse effects. These authors, while reporting the apparent safety of propofol in AIP, were guarded in their recommendation until further successful administrations had been documented.

The assessment of the porphyrinogenicity of a drug depends usually on the ability to detect increased concentrations of porphyrin precursors following its administration. It is obvious, however, that the relationship between increased concentrations of porphyrin precursors and manifest clinical symptoms is not a simple one [6]. Despite this, and in accordance with current criteria, we feel this case provides additional evidence to support the use of propofol as an induction agent in AIP, and more generally its use in the context of acute porphyrias.

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REFERENCES