ISOFLURANE IN DENTAL ANAESTHESIA FOR CHILDREN

Sir,—Two papers in the April 1986 issue of the Journal confirm the findings of earlier studies that isoflurane may cause more airway irritation than halothane. Although the results of the two reports are by no means dissimilar, the authors arrive at quite different conclusions as to the usefulness of the newer agent. McAteer and his colleagues believe that isoflurane provides a satisfactory alternative to halothane for use in the paediatric outpatient dental clinic, although offering no clear-cut advantage. Cattermole and his associates, on the other hand, take the view that it cannot be recommended because of the effects of its pungency.

The airway irritation caused by isoflurane may be minimized by the i.v. induction of anaesthesia. However, volatile agents are clearly convenient for use in infants and small children in whom venous access may not always be readily available before the induction of anaesthesia. It is in these circumstances, when isoflurane is used as the main agent in the unpremedicated child, that increased secretions and coughing may be troublesome. This is unfortunate because isoflurane would seem to have important advantages to offer: by its use the drawbacks of halothane such as the onset of ventricular arrhythmias, adrenaline incompatibility and the hazard, albeit remote, of hepatotoxicity, are largely circumvented. Other desirable features of isoflurane, although not readily apparent during clinical use, such as its low oil/gas partition coefficient with reduced implications of toxicity, metabolic stability and minimal impairment of myocardial contractility, should be kept in mind.

Isoflurane may well be the last inhalation agent to be marketed during the next few years. It would seem that its use in paediatric practice has yet to be fully determined and may well depend upon the extent to which anaesthetists are willing to tolerate a rather less smooth induction than with halothane in order to gain the benefits it has to offer.

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REFERENCES

CARDIAC ARREST FOLLOWING I.V. VERAPAMIL COMBINED WITH HALOTHANE ANAESTHESIA

Sir,—Halothane and verapamil depress cardiac contractility and interfere with electrical conduction (Reves et al., 1982). The potential problems of anaesthesia in patients receiving calcium-channel blockers have been emphasized (Prys-Roberts, 1984) and in a recent report in the Journal Ramsay and colleagues (1986) demonstrated a decrease in cardiac performance in dogs after verapamil administered i.v. during halothane anaesthesia.

We report a case of cardiac arrest in association with halothane anaesthesia preceded by verapamil i.v. A 56-year-old male treated with digoxin because of atrial fibrillation, was admitted to the hospital with haematometasis and melaena caused by gastric haemorrhage. Clinically he was shocked: arterial pressure 80/50 mm Hg; heart rate 150 beat min\(^{-1}\) in atrial fibrillation. Serum potassium concentration was within normal limits. Since it was impossible to correct the bleeding with blood transfusion, a laparotomy was performed. Anaesthesia was induced with ketamine 50 mg i.v. followed by suxamethonium 150 mg to facilitate tracheal intubation. Anaesthesia was maintained with nitrous oxide in oxygen 2:1 via a circle system, supplemented with fentanyl 0.2 mg i.v.

After haemostasis, the arterial pressure increased to 140/80 mm Hg. Because of persisting atrial fibrillation (heart rate of 160 beat min\(^{-1}\)), verapamil 5 mg was administered i.v., and resulted in a decrease in heart rate to 110 beat min\(^{-1}\). Ten minutes later 0.5% halothane was added to the gas mixture; profound hypotension was evident after 5 min with signs of ischaemia (S–T depression) and first degree heart block (prolonged P–R interval), and was followed by cardiac arrest. Open chest cardiac compression was commenced in combination with the administration of noradrenaline, atropine and calcium i.v. Cardiac output returned within 1 min and the patient was hyperventilated for 24 h, after which the recovery was uneventful. The patient was discharged from the hospital 10 days later without neurological sequelae. ECG and creatine kinase isoenzyme revealed no evidence of myocardial infarction.

The most likely reason for the event was the additive depressant effects of halothane and verapamil on global cardiac function as assessed in laboratory studies (Kapur and Flacke, 1981; Kapur et al., 1984), justifying caution in the simultaneous use of these two drugs.

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REFERENCES