Enflurane for controlled hypotension

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Summary

Enflurane was substituted for halothane in an established technique of controlled hypotension, involving beta-blockade and sympathetic ganglion blockade, for 2 groups of patients. One group was undergoing major plastic surgery and/or major maxillo-facial surgery and these patients breathed spontaneously. The other group was undergoing major neurosurgery and received intermittent positive pressure ventilation (IPPV). All patients were carefully monitored during anaesthesia and for 24 hours postoperatively. Good operation conditions were produced and no ill effects of controlled hypotension were detected in any of the patients.

KEY WORDS: enflurane, hypotension.

Introduction

Controlled hypotension during anaesthesia for certain major surgical procedures has a number of distinct advantages. There is a reduction in overall blood loss, often avoiding the need for large transfusions of blood, with all the attendant problems, and a better, almost bloodless, field is provided, which allows shorter anaesthetic and surgery time, resulting in less physiological upset in the patient.

It is, however, a potentially dangerous technique, if not carried out by an experienced anaesthetist, using a controllable easily reversible technique. Over a period of 10 years, a technique using halothane, beta-blockade in the form of practolol and sympathetic ganglion blockade using trimetaphan, was found by the author to be a relatively safe and controllable form of controlled hypotension. This study was set up to investigate the possibility of substituting enflurane for halothane, thus producing an even safer technique.

Method

The patients were in 2 main groups. Group 1 were patients undergoing major plastic surgery and/or

maxillo-facial surgery. These patients breathed spontaneously. Group 2 were patients undergoing major neurosurgery, such as clipping of cerebral aneurysms and these patients received intermittent positive pressure ventilation (IPPV). Ages ranged from 15 years to 71 years.

The patients were pre-medicated with lorazepam and anaesthesia was induced with althesin or etomidate (if the patient had a history of allergy). Both drugs are metabolised rapidly and have an only transitory effect on blood pressure. Endotracheal intubation was performed with the aid of suxamethonium chloride. Anaesthesia was maintained with oxygen, nitrous oxide and enflurane (in concentrations up to 5%), plus pancuronium bromide if IPPV was used. Controlled hypotension was achieved by producing a beta-blockade, followed by a sympathetic ganglion blockade.

Compensatory tachycardia occurs during controlled hypotension and is caused by stimulation of the baroreceptors in the aortic and carotid sinuses by falling blood pressure and adrenaline release from surgical stimulation during light anaesthesia. This tachycardia is detrimental because it produces a high myocardial oxygen demand and a short diastolic period and hence reduced coronary perfusion. Beta-blockade controls this tachycardia. Beta-blockade was judged to be present when the heart rate became stable irrespective of changes in blood pressure variation in anaesthesia or surgical intervention. Practolol was used undiluted and trimetaphan was used in the concentration of 10 mg/ml and administered in incremental doses of 5 mg to 10 mg.

At the end of the period of controlled hypotension the concentration of enflurane was reduced and the beta-blockade was reversed using intravenous atropine.

During anaesthesia, routine monitoring of ECG, heart rate, respiration (impedance pneumography), arterial blood pressure, urine output and serial blood gas estimations were carried out. All the patients were catheterised and all intravenous fluids were passed through a blood-warming coil. Impedance

pneumography provided very sensitive monitoring of respiration. Hypoxia of the medullary respiratory centre would have produced changes in respiration in the patients breathing spontaneously. Arterial pressure was measured from a cannula inserted into the radial or dorsalis pedis artery. A 20G Longdwell teflon cannula was used to prevent permanent damage to the artery. An Acker 840 transducer with a disposable dome was used in conjunction with a Simonsen and Weel monitor. A 3-way tap next to the cannula allowed frequent blood gas sampling.

If the period of controlled hypotension exceeded 1 hr 45 min, an infusion of 250ml of 20% mannitol was given to ensure adequate renal function. A ripple pad underneath the patient varied the pressure on the patient's calves and was especially useful in long cases. Monitoring of heart rate, ECG, blood pressure, urine output, and neurological state was continued for 24 hr after the operation.

Results and discussion

This technique provided ideal conditions for both surgeons and anaesthetists. Posture was not required to augment the hypotension which was easily reversible.

Group 1 contained 26 patients (11 males and 15 females) who received 28 controlled hypotensive anaesthetics. Group 2 contained 17 patients (6 males and 11 females) and 18 controlled hypotensive anaesthetics.

Duration of the hypotension varied from 15 min to 6 hr, in 40 cases it was under 180 min. The longer durations of hypotension were required for Group 1 patients. Only one of the neurosurgical patients required more than 1 hr of controlled hypotension. The longest periods of hypotension were required for patients undergoing excision of tumours of the head and neck, with major reconstructive surgery or block dissection of the neck. Two of these patients were

pregnant. One was a case of recurrent malignant melanoma of the face, which had apparently been aggravated by pregnancy and it was therefore decided to terminate the pregnancy at the end of the operation. The other case was a recurrent mixed parotid tumour and that pregnancy proceeded normally.

Most patients were normotensive pre-operatively and had an average mean blood pressure of 60–55 mmHg during the period of controlled hypotension. The few patients who had a slight to moderate hypertension before operation only had their mean blood pressure lowered to approximately 80 mmHg.

No ill effects of controlled hypotension were detected in any of the patients. In fact, this particular technique was almost certainly advantageous in some of the neurosurgical patients because enflurane produces a smaller rise in intracranial pressure than halothane (Cunitz, Danhauser and Grub, 1976). Cerebral perfusion pressure equals mean blood pressure minus mean intracranial pressure (ICP). It is important, therefore, if the blood pressure is lowered, that the ICP does not rise too much if reasonable and safe operating conditions are to be maintained.

Conclusion

It was found that enflurane could be substituted for halothane in an established, safe technique of controlled hypotension. The lower incidence of cardiac arrhythmias found with enflurane anaesthesia compared to halothane anaesthesia, combined with the influence on ICP mentioned above would suggest that enflurane might increase the margin of safety in a potentially hazardous technique.

Reference

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