trisilicate is not now the alkali of choice, but there are alternatives. I believe that metoclopramide, by increasing lower oesophageal sphincter tone, hastening gastric emptying and preventing vomiting, is an appropriate drug to use.

These young women are usually otherwise healthy, often undergoing an investigative procedure. It is imperative that the anaesthetic technique is designed to keep the mortality rate at zero and the morbidity rate to the absolute minimum.

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REFERENCES

TYMPANIC MEMBRANE RUPTURE DURING NITROUS OXIDE ANAESTHESIA

Sir,—The use of nitrous oxide has been associated with the expansion of a pneumothorax (Eger and Saidman, 1965; Eger, 1974), an increase in intrathecal pressure (Fuller and Lewis, 1975), and increased intracranial tension following pneumoencephalography (Artru, 1978). Impairment of hearing following pressure change in the middle ear has been reported in susceptible patients (Patterson and Bartlett, 1976), but tympanic membrane rupture is a rare complication. Owens, Gustave and Selasoff (1978) reported two patients who had pre-existing ear disease.

A 28-yr-old woman weighing 75 kg was admitted for late termination of pregnancy and laparoscopic sterilization. She was healthy, but had had a mild cough 2 weeks earlier for which an antibiotic had been prescribed. There was no past history of earache or hearing deficiency.

Labour was induced by extramamniotic prostaglandin E2

and an infusion of syntocinon 40 i.u. in 500 ml of 5% dextrose was instituted. Eight hours later a 20-week-old fetus was delivered. The following morning the patient received papaveretum 15 mg and hyoscine 0.3 mg; anaesthesia was induced with thiopentone sodium 350 mg; tracheal intubation was facilitated by alcuronium 20 mg. Anaesthesia was maintained with nitrous oxide 6 litre min⁻¹, oxygen 4 litre min⁻¹ and halothane 0.5%, and intermittent positive pressure ventilation performed using a Penlon-Oxford ventilator delivering a tidal volume of 10 ml kg⁻¹ body weight at a frequency of 12 b.p.m. The patient was placed in the lithotomy position and the retained products of conception were evacuated; ergometrine 0.5 mg was given i.v. to promote uterine contraction. Uterine bleeding continued, so a further dose of ergometrine 0.5 mg was given, and an i.v. infusion of Hartmann’s solution was commenced. The patient was placed head-down and laparoscopic sterilization performed using a carbon dioxide pneumoperitoneum. During this procedure blood was noticed in the right external auditory meatus. This was removed, no further bleeding was noted, and surgery was completed uneventfully. Antagonism of neuromuscular blockade was achieved with neostigmine 2.5 mg and atropine 1.2 mg, and the patient made an uncomplicated recovery.

The following morning the ears were examined and a perforation of the right tympanic membrane was found. The only complaint made by the patient was of some “heaviness” on the affected side. She was referred to a Consultant Otolaryngologist who believed this to be an acute atraumatic rupture of the tympanic membrane, an opinion supported by the fact that it healed completely without treatment in 4 weeks.

I believe that nitrous oxide was the agent most likely to be responsible for this rupture.

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REFERENCES

CARDIOVASCULAR RESPONSES TO THE SITTING POSITION

Sir,—In a recent issue, Dalrymple, MacGowan and MacLeon (1979) noted considerable haemodynamic changes when patients were raised from the supine to the sitting position during neurosurgical procedures. They described marked decreases in cardiac index, stroke volume, \( P_{O_2} \) and \( Q_s/Q_L \) and a significant increase in \( (P_{A_o} - P_{A_{CO_2}}) \) and total peripheral resistance. They concluded that the maintenance of normotension in the sitting position did not necessarily signify an adequate circulatory status.

There is a considerable cardiovascular challenge involved in changing from supine to the sitting position in the relatively healthy patient. A “stable” arterial pressure may be recorded in the presence of an inadequate or marginal perfusion and care should be exercised in subjecting the elderly or poor-risk patient who does not have active compensatory mechanism in this position.

Albin and others (1974) studied the cardiovascular responses in eight patients aged 41–56 yr, all being ASA physical status I except for one patient who was ASA II. All patients underwent posterior fossa cranial nerve exploration and had no evidence of intracranial lesions nor increased I.C.P. Premedication, anaesthesia induction and maintenance and ventilation were standardized. Appropriate catheters were inserted and cardiac index (indocyanine green dye dilution cardiac output and body surface area measurements), oxygen transport (cardiac output and