CORRESPONDENCE

REUGITATION DURING LAPAROSCOPY

Sir,—May I be allowed to comment on the paper of Dr Duffy (1979) on regurgitation during laparoscopy before it is accepted into anaesthetic dogma that all such patients should receive metoclopramide and magnesium trisilicate.

In assessing the risk of Mendelson's syndrome in this operation, it might be wiser to look at the frequency of the syndrome itself rather than extrapolating from the detection of acid in the pharynx. In the series of 50 000 laparoscopies, studied prospectively under the aegis of the Royal College of Obstetricians and Gynaecologists (1978) the frequency of inhalation of gastric contents was zero. In approximately 5000 of these patients the trachea was not intubated. If drugs are to be given to protect against the remote possibility of aspiration, many thousands must be treated to prevent one such case. If metoclopramide and magnesium trisilicate were totally harmless then one might not cavil at the recommendation to give them. Where, however, is the evidence that they are harmless? It is unlikely that any drug can be given to many thousands of patients without some side effects.

In the case of magnesium trisilicate it is often forgotten that the compound is very alkaline and if inhaled will itself cause Mendelson's syndrome. If the stomach is empty (as is the case in most patients undergoing laparoscopy) then no neutralization of this alkali will occur and the patient is as much at risk as if the pH were 2.5 or less.

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REFERENCES


ANAESTHESIA-INDUCED RHABDOMYOLYSIS IN DUCHENNE MUSCULAR DYSTROPHY

Sir,—Patients with Duchenne muscular dystrophy (DMD) are generally not considered susceptible to anaesthesia-induced malignant hyperthermia and rhabdomyolysis (Richards, 1972). We report the occurrence of this complication in a 5-yr-old boy during adenotomy at a regional hospital. There was no family history of neuromuscular disease or anaesthetic-related complications. After premedication with atropine 0.15 mg and diazepam 1.5 mg, anaesthesia was induced with halothane and nitrous oxide in oxygen, followed by suxamethonium 25 mg. This prompted rigidity of masseter and limb muscles and was followed a few minutes later by cardiac arrest. The patient was successfully resuscitated. Metabolic acidosis (pH 7.13; base excess 20 mmol litre⁻¹) was corrected by i.v. sodium bicarbonate. Rectal temperature increased to 38.5 °C within 30 min. Urine passed subsequently was red and was shown to contain myoglobin. On admission to our hospital 3 h later, there was still marked rigidity predominantly affecting the lower limbs. This was relieved after i.v. dantrolene sodium 100 mg. The urine cleared within 24 h. Creatine phosphokinase (CPK), monitored daily, was 30 000 u. litre⁻¹ (normal < 80 u. litre⁻¹) on admission and decreased gradually over 1 week to be repeatedly normal 8–12 days later. The clinical findings were characteristic for DMD with waddling gait, positive Gower's manoeuvre and calf hypertrophy. The e.m.g. was consistent with a myopathic process. Quadriceps muscle biopsy, performed under local anaesthesia 3 months later, confirmed muscular dystrophy. CPK at this time was 9300 u. litre⁻¹.

We are aware of only four reports describing seven DMD patients with anaesthesia-induced rhabdomyolysis (Watters, Karpatic and Kaplan, 1977; Miller et al., 1978; Reske-Nielsen, 1978; Seay, Ziter and Thompson, 1978). Hyperthermia was mentioned in two of these (Watters, Karpatic and Kaplan, 1977; Reske-Nielsen, 1978), cardiac arrest in two others (Seay, Ziter and Thompson, 1978). Suxamethonium was administered in all cases, and halothane in all but one (Seay, Ziter and Thompson, 1978). Although the available literature does not allow us to estimate the frequency of these complications in DMD, we think that these patients are at risk and that, in particular, suxamethonium and halothane should be avoided.

The observation of repeatedly normal CPK values 8–12 days after the acute event was unexpected. We explain this as temporary CPK depletion of muscle cells as a result of excessive "leakage" of enzyme during the acute stage of rhabdomyolysis.

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REFERENCES


