1.6 mg and fentanyl 0.03 mg 45 min before surgery, and anaesthesia was induced with ketamine 50 mg. Endotracheal intubation was facilitated with suxamethonium 20 mg i.v. Anaesthesia was maintained with nitrous oxide in oxygen 2:1 and ventilation was controlled with pancuronium 1.5 mg. The neuromuscular blockade was antagonized by hyoscine 0.2 mg and neostigmine 1 mg i.v.

Arterial pressure was maintained at 110/80 mm Hg and the heart rate at 120 beat min⁻¹ until the injection of hyoscine which increased the rate to 160 beat min⁻¹. This effect lasted for about 5 min until the neostigmine produced a gradual reduction of heart beat to 68 beat min⁻¹, and 30 min later the heart rate remained stable at 100 beat min⁻¹.

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REFERENCE

ASPIRATION SYNDROMES IN PREGNANCY

Sir,—In the past decade it has become accepted practice to give oral alkali to patients in labour as prophylaxis against the acid aspiration syndrome.

We report two patients who received appropriate alkali therapy, in which the aspiration of alkaline stomach contents into the lungs during anaesthesia for Caesarean section resulted in differing courses.

The first patient, a 25-year-old primigravida, underwent an emergency Caesarean section under general anaesthesia after prolonged labour. Routine precautions at induction did not prevent the regurgitation and inhalation of stomach content, which was seen as a clear yellow fluid. Tracheal intubation was performed rapidly, and a suction catheter was inserted via the tube. A healthy child was delivered by routine lower segment Caesarean section. After antagonism of residual neuromuscular blockade and removal of the tracheal tube, there was ventilatory inadequacy and peripheral circulatory collapse. The patient was transferred to the intensive therapy unit, for treatment of the acid aspiration syndrome. Investigations included chest radiography (fig. 1) and analysis of the gastric aspirate. The patient died 8 h after the induction of anaesthesia.

The second patient, a 32-year-old primigravida, underwent an emergency Caesarean section under general anaesthesia because of obstructed labour. Before induction a nasogastric tube was passed and the stomach aspirated. Following induction, tracheal intubation was carried out without incident. During the operation, the cuff of the tracheal tube burst, laryngoscopy was performed immediately, and the tracheal tube changed without incident. Ten minutes later the cuff of the second tube burst, and at laryngoscopy a pool of whitish, minty-smelling fluid was observed in the pharynx; this was removed by suction, and a third tracheal tube inserted. The operation was completed uneventfully, and the patient recovered fully from anaesthesia, but 30 min later she developed tachypnoea, cyanosis and tachycardia. Chest radiography (fig. 2) was performed, and the patient was transferred to the intensive therapy unit for treatment of the acid aspiration syndrome. The patient recovered completely.

![Fig. 1. Chest x-ray of first patient.](image1)

![Fig. 2. Chest x-ray of second patient.](image2)
These two reports raise doubts about the efficacy of the magnesium trisilicate regime which has been so widely described in the U.K. and elsewhere (Crawford, 1972).

There is good evidence that an adequate regime, as recommended, had been carried out in these two patients. In the first patient, the documentation of individual doses of the drug was not complete, but a gastric sample obtained 90 min after the inhalation contained 14.3 mmol litre$^{-1}$ of magnesium (four times the normal concentration) and with a pH 7.6 unit. In the second patient, seven doses of 15 ml of magnesium trisilicate BPC was recorded as having been administered during the course of a 10 h labour. In addition, the anaesthetist observed the colour and odour of the fluid pharyngeal contents at the time of the second tracheal incubation.

The chest x-ray appearances in these patients suggested massive inhalation by both patients, but the clinical course and blood-gas measurements were different, as was the outcome. These case reports pose three questions.

First, is it rational to add a volume of fluid to the stomach of a woman in labour in the knowledge that intestinal and gastric stasis (Nimmo, Wilson and Prescott, 1975) is a normal consequence of labour? In the first patient there was x-ray evidence of gastric dilatation.

Second, are the effects of the inhalation of magnesium trisilicate less dangerous than the aspiration of normal stomach contents? To a certain extent, the volume of gastric fluid may be controlled by diet, by the administration of apomorphine, or the passage of a wide-bore nasogastric tube.

An important factor which has been overlooked is that the administration of an antacid increases the rate of production of intragastric hydrogen ion six- to 20-fold (Price and Sanderson, 1956).

Third, does the administration of magnesium trisilicate induce a false sense of security on the part of the anaesthetist in relation to his technique? Does a properly conducted magnesium trisilicate regimen invariably produce an alkaline aspirate, or at least an aspirate with a pH in excess of 3.5?

The authors would suggest that it does not. In a series of 27 patients (table I), in whom the recommended regime had undoubtedly been administered, four of the patients had a gastric pH within the range described as being liable to produce the acid aspiration syndrome.

<table>
<thead>
<tr>
<th>Magnesium trisilicate doses</th>
<th>Mean</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH of gastric aspirate (unit)</td>
<td>6.6</td>
<td>1.5-9.0</td>
</tr>
<tr>
<td>$\text{Mg}^{2+}$ concn in gastric aspirate (mmol litre$^{-1}$)</td>
<td>87.1</td>
<td>0.6-370.0</td>
</tr>
</tbody>
</table>

TABLE I. Number of administrations of magnesium trisilicate B.P. 15 ml and analysis of gastric aspirate in 27 patients undergoing operative delivery

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REFERENCES

