INFLUENCE OF PREOPERATIVE GASTRIC ASPIRATION ON THE VOLUME AND pH OF GASTRIC CONTENTS IN OBSTETRIC PATIENTS UNDERGOING CAESAREAN SECTION

J. G. BROCK-UTNE, C. ROUT, J. MOODLEY AND N. MAYAT

Pulmonary aspiration of gastric contents continues to be of major concern in obstetric anaesthesia [1]. The combination of an intragastric pH of 2.5 or less with a gastric contents volume greater than 25 ml are generally regarded as placing the patient at risk of chemical aspiration pneumonitis [2], although this condition has been reported in a patient following the aspiration of stomach contents of pH 3.5 [3], and intragastric pH values of greater than 2.5 may cause pulmonary damage in rats [4].

Attempts to increase the pH of gastric contents have met with varying degrees of success [5,6]. The use of antacids either alone [7] or in combination with H$_2$-receptor antagonists [8] increases pH adequately in the majority of patients, but this is less effective in patients presenting in labour for emergency Caesarean section [9].

Several methods have been used to decrease intragastric volumes before emergency Caesarean section, including preoperative orogastric intubation [5,10], mechanical stimulation of vomiting [11], i.v. apomorphine [12] and metoclopramide [13,14]. For many years, at King Edward VIII Hospital it has been routine practice to aspirate the stomach via a wide bore (28-French gauge) stomach tube before administration of 30 ml of sodium citrate 0.3 mol litre$^{-1}$ in patients presenting for emergency Caesarean section. Exceptions to this practice are patients with hyper-

**SUMMARY**

Aspiration of gastric contents, the most common anaesthetic cause of maternal mortality, is decreased by emptying of the stomach and the use of antacids and H$_2$-receptor antagonists. One hundred and eighty-three mothers presenting for emergency Caesarean section were allocated to three groups. In group 1, the stomach was emptied before operation via an orogastric tube and thereafter 30 ml of sodium citrate 0.3 mol litre$^{-1}$ was ingested 5–15 min before induction of general anaesthesia (our usual practice). Group 2 received only 30 ml of sodium citrate 0.3 mol litre$^{-1}$. Group 3 received ranitidine 50 mg i.v. before operation, 5–15 min before induction of anaesthesia, in addition to sodium citrate. Our results show that preoperative gastric emptying with an orogastric tube followed by sodium citrate is preferred if anaesthesia should be induced 15–20 min later. However, the use of ranitidine and sodium citrate is preferred at subsequent times. Although our data show that preoperative gastric emptying decreased the mean intragastric volumes before Caesarean section, the number of patients at risk of acid aspiration was not reduced. In view of these findings and the unpleasantness of orogastric intubation, we suggest that routine preoperative gastric aspiration via an orogastric tube is not justified, although the manoeuvre should still be used following a recent meal.
amounts of lipid solvent administered, but these differences were not significant. However, changes in post traumatic metabolism may also have caused disturbances of the enzyme systems responsible for serum lipid clearance and metabolism [11].

Interestingly, HDL-cholesterol concentrations also tended to be greater in patients receiving propofol. We cannot explain these findings, but the possible protective properties of HDL-cholesterol [12] may render this a desirable side effect of propofol infusion.

In summary, infusion of propofol produced adequate sedation in adult patients who required mechanical ventilation in the ICU. When used for long term sedation by continuous infusion, the lipid solvent for propofol may replace separate infusion of parenteral lipids for nutrition. The contraindications to long term infusion of propofol include inborn and acquired anomalies of lipid metabolism [13] and known allergy to this agent. Daily monitoring of serum concentrations of lipids is necessary, especially if high doses are administered for long periods. However, further studies are required to confirm the safety and efficacy of propofol in a greater number and variety of ICU patients.

REFERENCES
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tension, cardiac disease, fetal distress or acute haemorrhage.

The unpleasant nature of orogastric intubation has led many of our anaesthetists and surgical colleagues to question its value. However, before instituting a major change in labour ward policy, we decided to investigate the effect of preoperative gastric emptying on the intragastric pH and volume of stomach contents at induction of anaesthesia. We have compared three treatment groups: preoperative gastric emptying followed by oral sodium citrate, preoperative oral sodium citrate alone, and preoperative oral sodium citrate in combination with a single i.v. injection of ranitidine.

PATIENTS AND METHODS

We studied 183 healthy mothers (aged 18–38 yr) referred for emergency Caesarean section at 36–42 weeks gestation. Patients with evidence of cardiopulmonary disease, hypertension, diabetes or gross obesity (> 110 kg) were excluded. Informed consent was obtained and the study was approved by the Ethics and Standards Committee of this Medical Faculty.

Gastric emptying was performed using an orogastric tube before operation in 63 patients and the tube was removed before induction of anaesthesia. Thereafter 30 ml of sodium citrate 0.3 mol litre$^{-1}$ was administered by mouth and the time noted (group 1). Another 60 patients (group 2) received sodium citrate only, while group 3 (60 patients) received ranitidine 50 mg by slow (1 min) i.v. injection 5 min before induction of anaesthesia, in addition to oral sodium citrate.

A standard general anaesthetic technique was used with the usual precautions against aortocaval occlusion. All patients received metoclopramide 10 mg i.v. followed by glycopyrrolate 0.2–0.4 mg i.v. or atropine 0.3–0.6 mg i.v. [15]. After preoxygenation, anaesthesia was induced with thiopentone 3–4 mg kg$^{-1}$. Cricoid pressure was applied with induction and relieved following intubation of the trachea with a cuffed tracheal tube; this was facilitated by suxamethonium 1–1.5 mg kg$^{-1}$ i.v. Anaesthesia was maintained with 0.5 % halothane or 1 % enflurane and 50 % nitrous oxide in oxygen, and the lungs ventilated mechanically to achieve normocapnia. Further neuromuscular blockade was obtained using alcuronium 12.5 mg and increments of 2.5 mg when necessary.

A 16-French gauge nasogastric tube was inserted after tracheal intubation and its position verified by the simultaneous injection of air and auscultation over the epigastrium. Each group of patients was classified further into six subgroups, A–F. Aspiration of gastric contents for pH and volume measurement was performed at 15–20 min in group A, at 1 h in group B, 2 h in group C, 2.5 h in group D, 3 h in group E and at 3.5 h in group F. Patients were allocated randomly to groups 1 and 2, while group 3 was studied after the completion of groups 1 and 2 because of initial unavailability of ranitidine for i.v. use. Although the technique of gastric aspiration cannot guarantee complete gastric emptying, and volumes recorded reflect only the maximum gastric aspirate obtained at the time, every effort was made to increase yield by manipulation of the tube.

At delivery, syntocinon 5 units was given i.v. and 15 units was added to the i.v. infusion. At termination of surgery, the residual effects of alcuronium were antagonized by injection of glycopyrrolate 0.8 mg with neostigmine 2.5–5 mg i.v. Gastric contents were sampled at the times noted above, measured volume was recorded and the pH measured with Merck indicator paper; the results were standardized against a Beckman instrument pH electrode. A good correlation between the results obtained with the Merck paper and the various pH electrodes has been reported previously [5,16].

Volume and pH data were compared using the Mann–Whitney U test. The numbers of patients considered to be at risk of pulmonary aspiration in each group were compared using the Fisher Exact Test. For the purposes of this study our definition of an “at risk” patient was one with an intragastric volume > 25 ml and pH ≤ 3.5. $P < 0.05$ was considered to be statistically significant.

RESULTS

In all groups the colour of the gastric content ranged from clear to deep green. The latter suggested the presence of bile, but this was not always associated with an alkaline pH. In three patients, no gastric contents was aspirated. All three had had a stomach tube passed before operation and gastric aspiration performed 15–20 min after ingestion of sodium citrate. All patients were in active labour for an indeterminate period of time before admission. The majority received a combination of pethidine 100 mg and
Promazine 50 mg i.m. for labour pains. The slow i.v. injection of ranitidine did not cause any adverse effects.

Maternal age, weight, gestation and parity were similar in the three groups (table I).

Mean pH was significantly greater in group 1 than in groups 2 and 3 (P < 0.02) at 15–20 min and was significantly greater in group 3 at 2, 2.5 and 3 h than in other two groups and significantly greater in group 3 than in group 1 at 3.5 h (P < 0.02) (table II).

Measured volumes were significantly lower (P < 0.05) in group 1 than in groups 2 and 3 at 15–20 min and was significantly greater in group 3 at 2, 2.5 and 3 h than in other two groups and significantly greater in group 3 than in group 1 at 3.5 h (P < 0.02) (table III).

There was no significant difference in the number of patients at risk in groups 1 and 2 (P = 0.35), groups 1 and 3 (P = 0.13) or groups 2 and 3 (P = 0.06) (tables IV–VI).
DISCUSSION

The results of our study appear to confirm previous reports [5,17-19]. Sodium citrate alone produced an intragastric pH < 3.5 for at least 15-20 min. However, one patient had an intragastric pH of 3.5 and the mean intragastric pH in this group was significantly lower than in those patients who also had their stomachs emptied before operation. This result is not surprising when the higher sample volumes are taken into account, indicating that decreasing intragastric volume before administration of an antacid is of value in increasing pH. This effect may not persist for longer than 15-20 min, despite significantly smaller volumes (presumably because of continued gastric acid secretion) and the period of emergence from anaesthesia may not be affected.

However, in the patients who received i.v. ranitidine, pH values were significantly greater than in both the other groups after 2 h, but intragastric volumes were not significantly smaller. This suggests that ranitidine decreased hydrogen ion secretion, but not the total volume of secretion, in these patients. There is, however, some evidence to suggest that ranitidine does decrease gastric volume in elective Caesarean section [20]. It is possible that a larger dose of ranitidine, a longer period following administration, or both, might exert a beneficial effect on volume. Although there was no significant difference in mean pH values between the groups at 1 h, all the values in the patients given ranitidine exceeded 4, whereas each of the other groups at this time included a patient considered to be at risk from the combination of low pH and large gastric volume.

Mean measured volumes at 15-20 min, 1, 2 and 2.5 h after administration of sodium citrate were significantly smaller in patients who underwent gastric emptying before operation, although only at 15-20 min were there no patients with a measured volume greater than 25 ml. However, the evidence for a critical volume necessary to produce acid pneumonitis is somewhat tenuous. Unpublished work by Roberts and Adamsons, alluded to in Roberts and Shirley’s paper of 1974 [2], suggested that, in Rhesus monkeys, aspiration of gastric contents in excess of 0.4 ml kg⁻¹ (approximately 25 ml in human parturients) would be hazardous. Animal studies undertaken before this work involved the tracheal instillation of fluid up to 4 ml kg⁻¹ of varying pH in order to produce the acid aspiration syndrome [11,21]. Recently, it has been shown [22] that human aspirate (pH 1, volume 0.4 ml kg⁻¹) instilled into monkeys did not produce the clinical signs of aspiration pneumonitis.

It is possible that we are being unrealistic in pursuing an ideal of less than 25 ml of stomach contents in patients for emergency Caesarean section. A wide bore orogastric tube would seem to be the best way to achieve this, but the technique cannot guarantee an intragastric volume of less than 25 ml (particularly after 1 h).

Although the use of a stomach tube to decrease intragastric volume before general anaesthesia is often recommended in the literature [9,10], we know of only one study that has examined the effects of this technique before emergency obstetric anaesthesia [23]. Of alternative techniques, induced vomiting, whether mechanical or pharmacological, is as unpleasant as the passage of an orogastric tube and i.v. metoclopramide may not be effective following the administration of opioid analgesics [24]. In a comparison between pre-operative gastric aspiration by stomach tube and i.v. apomorphine, Holdsworth, Furness and Roulston [23] concluded that both methods were equally effective in decreasing volume, and that neither guaranteed an empty stomach.

Ranitidine i.v. produced higher mean pH values from 1 h onward but, not surprisingly, did not cover induction of anaesthesia. Sodium citrate alone is inadequate for emergency Caesarean section as the beneficial effect lasts only for 15-20 min and further doses of sodium citrate must be given to cover the emergence period. Oral ranitidine has been given to obstetric patients 2 h before surgery together with sodium citrate, producing a gastric pH > 3 [25]. Ranitidine has the added advantage that it increases lower oesophageal sphincter tone [26] and does not possess many of the side effects of cimetidine [25,27-31]. We observed no adverse clinical effects following slow i.v. injection of ranitidine in our 60 patients. The reason for giving ranitidine only 5 min and not the recommended 60 min before induction was that we wished to assess the extent and duration of any effects of ranitidine at fixed time intervals in comparison with the other two groups.

Our results show, not surprisingly, that pre-operative gastric emptying significantly decreased mean intragastric volumes before Caesarean section, but that this did not significantly decrease
Preoperative gastric emptying

the number of patients at risk of acid aspiration. At 15–20 min none of the groups had patients at risk, implying that sodium citrate alone might be effective for a short period, although far larger numbers would be needed in each group in order to exclude a type II error. Although it is possible to reduce intragastric volume by gastric aspiration before operation, the unpleasantness of the procedure, the uncertain role of volume in the pathogenesis of the acid aspiration syndrome, and the inability of the technique to reduce the number of patients at risk of acid aspiration, does not justify its routine use. However, we shall continue to use orogastric aspiration following a recent meal. The use of i.v. ranitidine in addition to sodium citrate may be useful if given at least 1 h before operation, and this provides additional prophylaxis for the recovery period following general anaesthesia.

REFERENCES