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Responses to Atropine, Glycopyrrolate, and Riopan of Gastric Fluid *p*H and Volume in Adult Patients

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Anticholinergic drugs produce dose-related reductions in the acidity and volume of gastric secretions. These drugs could reduce the severity of acid-aspiration pneumonitis should vomiting or regurgitation occur during the operative period. Indeed, atropine (15–20 μg/kg) or glycopyrrolate (7.5–10 μg/kg) given intramuscularly an hour before anesthetic induction in children increased the incidence of gastric fluid samples with *p*H values greater than 2.5.[1] Furthermore, glycopyrrolate also reduced gastric fluid volume. However, similar doses of atropine or glycopyrrolate administered to adult patients would exceed the usual preanesthetic medication dose and might introduce undesirable side effects. This report describes gastric fluid *p*H and volume following more traditional preanesthetic medication doses of atropine (0.4 mg) or glycopyrrolate (0.2 mg) administered to adult patients scheduled for elective operations.

METHODS

Two hundred and fifty nonobese adult patients scheduled for elective operations were studied. The study protocol was approved by the Indiana University School of Medicine Human Study Committee. No patient had a disease or was receiving a drug known to influence gastric secretion or motility. All had fasted for at least seven hours before induction of anesthesia. Depending on the preanesthetic medication administered im 60–90 minutes before anesthetic induction, the patients were divided into four groups:

I, morphine 10–15 mg (75 patients)
II, morphine–atropine, 0.4 mg (75 patients)
III, morphine–glycopyrrolate, 0.2 mg (75 patients)
IV, morphine–atropine–Riopan† (10 ml followed with 10 ml of water 30 minutes before anesthetic induction) (25 patients)

Anesthetic induction was accomplished with gallamine (0.3 mg/kg), followed a minute later by thiamylal (4 mg/kg) and succinylcholine (2 mg/kg). iv. A cuffed endotracheal tube was placed and ventilation controlled with 60 per cent nitrous oxide and 40 per cent oxygen. Within 5 minutes after tracheal intubation an 18-French gastric tube (sump-type) was placed in the stomach. Gastric contents were aspirated for *p*H and volume determination. Gastric fluid *p*H was measured within 15 minutes after obtaining the sample, using a Corning *p*H meter and Beckman electrode.

Data were analyzed using the Chi-square test or analysis of variance. $P < .05$ was considered significant.

RESULTS

Patient ages and weights were similar in the four groups (table 1). Compared with patients receiving

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† Magaldrate (magnesium and aluminum hydroxide as a single molecule), 80 mg/ml.

TABLE 1. Patient Characteristics for Each Study Group (Mean ± SE)

	Age (Years)	Weight (kg)
Morphine	44 ± 4	68 ± 5
Morphine–atropine	43 ± 6	73 ± 4
Morphine–glycopyrrolate	45 ± 3	68 ± 3
Morphine–atropine–Riopan	39 ± 5	78 ± 4

morphine only, the percentages of patients with gastric fluid *p*H values below 2.5, volumes of more than 20 ml, and the combination of *p*H below 2.5 and volume of more than 20 ml were not altered (Chi-square test, $P > .05$) by addition of atropine or glycopyrrolate to the preanesthetic medication (table 2). Riopan was effective in increasing gastric fluid *p*H to above 2.5 in every patient, but was associated with a greater incidence of gastric fluid volumes more than 20 ml (table 2). Gastric fluid *p*H values were above 6.5 for 20 of 25 patients receiving Riopan. The lowest gastric fluid *p*H after Riopan was 4.2, in a patient with a measured aspirate volume of only 10 ml. Gastric fluid volumes in patients receiving Riopan ranged from 5 to 65 ml.

Mean gastric fluid *p*H and volume were not different among Groups I–III (analysis of variance, $P > .05$) when patients were separated within each group based on *p*H's above and below 2.5 or volumes of more and less than 20 ml (table 3). Likewise, there was no difference among Groups I–III (analysis of variance, $P > .05$) comparing only those patients within each group with the combination of gastric fluid *p*H below 2.5 and volume of more than 20 ml (table 4).

DISCUSSION

These data demonstrate that atropine, 0.4 mg, or glycopyrrolate, 0.2 mg, does not increase gastric fluid *p*H to above 2.5 or reduce gastric fluid volume to less than 20 ml. Sixty-three per cent of patients receiving morphine as preanesthetic medication had gastric fluid *p*H's below 2.5. This incidence was not decreased ($P > .05$) by the inclusion of atropine or glycopyrrolate in the preanesthetic medication. Likewise, a similar number of patients (23 to 27 per cent) had gastric fluid volumes of more than 20 ml with or without administration of an anticholinergic drug.

Acid-aspiration pneumonitis is most likely when the *p*H of the aspirated fluid is below 2.5 and the volume exceeds about 0.4 ml/kg.[2–4] It was not possible to determine whether the stomach was completely emptied by the gastric tubes placed in our patients. Therefore, the measured gastric fluid volumes may have underestimated the true volumes. Thus, 20 ml (about 0.3 ml/kg) was selected as a conservative estimate, above which gastric fluid volume combined with a low *p*H was a potential hazard. Even the *p*H value of 2.5 is controversial, as emphasized by the reported occurrence of acid pneumonitis following inhalation of fluid with a *p*H of 3.5.[5] Nevertheless, using the guidelines of a gastric fluid *p*H below 2.5 and volume of more than 20 ml, the present data suggest that 16–17 per cent of fasted patients are vulnerable to acid pneumonitis should aspiration of gastric fluid contents occur.

These data from adult patients do not agree with those in a previous study of pediatric patients demonstrating the efficacy of atropine and glycopyrrolate in elevating gastric fluid *p*H to above 2.5 and thus reducing the hazard of acid-aspiration pneumonitis.[1] The most likely explanation is the smaller dose of anticholinergic drug administered to our adult patients. For example, extrapolating the μg/kg dose administered to pediatric patients[1] to a 70-kg adult patient would have resulted in using 1.0–1.4 mg atropine or 0.5–0.7 mg glycopyrrolate in the preanesthetic medication. This range exceeds the usual adult anticholinergic dose for preanesthetic medication and might result in undesirable side effects.

The efficacy of antacid therapy depends upon the time allowed for neutralization and gastric fluid volume. Thirty minutes seem adequate for neutralization of acid, as gastric fluid *p*H was above 4.0 for all patients and more than 6.5 for most patients receiving Riopan. It seems unlikely that the fasted adult patient without gastrointestinal disease would have a gastric fluid volume that would preclude effective neutralization with an antacid. Our data demonstrates that gastric fluid in volumes as great as 65 ml was effectively neutralized by Riopan.

In conclusion, atropine or glycopyrrolate in usual adult doses for preanesthetic medication did not increase gastric fluid *p*H to above 2.5 or reduce volume to below 20 ml at the time of anesthetic induction. In contrast, an antacid, Riopan, given shortly

TABLE 2. Percentages of Patients with Gastric Fluid *p*H above 2.5 and/or Volume below 20 ml

	*p*H Below 2.5	Volume More Than 20 ml	*p*H Below 2.5; Volume More Than 20 ml
Morphine (n = 75)	63	27	16
Morphine–atropine (n = 75)	58	27	17
Morphine–glycopyrrolate (n = 75)	52	23	16
Morphine–atropine–Riopan (n = 25)	0*	60*	0*

* $P < .05$ compared with morphine.
n = number of patients.

TABLE 3. Gastric Fluid *p*H and Volume (Mean ± SE) for Those Patients within the Same Study Group According to *p*H below or above 2.5 or Volume above or below 20 ml

	*p*H		Volume	
	Below 2.5	Above 2.5	More Than 20 ml	Less Than 20 ml
Morphine	1.6 ± .08 (n = 47)	4.9 ± .3 (n = 28)	42 ± 6 (n = 20)	10 ± 2 (n = 55)
Morphine–atropine	1.8 ± .08 (n = 43)	4.6 ± .4 (n = 32)	61 ± 16 (n = 20)	8 ± 3 (n = 55)
Morphine–glycopyrrolate	1.9 ± .09 (n = 37)	5.2 ± .2 (n = 38)	55 ± 10 (n = 17)	8 ± 3 (n = 58)

n = number of patients.

before anesthetic induction reliably elevated the gastric fluid *p*H to far above 2.5. This desirable effect must be weighted against the possible hazards of increased gastric fluid volume in patients pretreated with an antacid. Routine antacid therapy may be reasonable to consider in view of the fact that 16–17 per cent of fasted patients awaiting elective operations were found to be at risk should aspiration have occurred (*i.e.*, gastric fluid *p*H below 2.5 and volume of more than 20 ml). Certainly, antacids would seem indicated when a cuffed endotracheal tube is not placed or difficult and prolonged anesthetic induction or tracheal intubation is anticipated.

TABLE 4. Gastric Fluid *p*H and Volume (Mean ± SE) for Those Patients within the Same Study Group Manifesting *p*H below 2.5 and Volume above 20 ml

	*p*H	Volume (ml)	Number of Patients
Morphine	1.6 ± .06	47 ± 9	12
Morphine–atropine	1.8 ± .1	70 ± 15	13
Morphine–glycopyrrolate	1.7 ± .08	60 ± 14	12

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Wolff–Parkinson–White Syndrome during Anesthesia

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A major problem in anesthetic practice is the diagnosis and treatment of cardiac arrhythmias, especially when they arise for the first time during general anesthesia. Since it was first described in 1930, the Wolff–Parkinson–White syndrome remains one of the most interesting and, at the same time, one of the most difficult of the cardiac arrhythmias to treat. We report a case in which this syndrome arose entirely unexpectedly during general anesthesia, and offer some recent considerations of the subject.

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REPORT OF A CASE

A healthy 22-year-old man was scheduled for arthroscopy of the right knee with general anesthesia. There was a history of mild asthmatic bronchitis, but the patient had no complaint of any kind except signs and symptoms related to his knee. Clinical examination, chest x-ray and routine laboratory investigations revealed no abnormality. An ECG was not obtained because the patient was less than 30 years of age and there was no specific indication for it.

Premedication: Atropine, 0.5 mg, and promethazine, 50 mg were given.

Induction: When the patient was connected to Lead I of the ECG, before induction of anesthesia, as is our practice, some supraventricular extrasystoles were seen, probably due to an accessory pathway (fig. 1*A*). The patient denied feeling palpitations or any abnormality of his heart beat. Blood pressure was normal. Induction was commenced with Clemastine, 2 mg, followed by