Volume and *p*H of Gastric Juice in Obese Patients

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Since Mendelson's[1] description of the symptom complex of the aspiration low-*p*H stomach contents into the tracheobronchial tree with resultant destruction of bronchial mucosa and chemical pneumonitis, there have been numerous reports[2,3] identifying obstetrical patients as a particularly high-risk group. A close parallel can be drawn between the obstetric patient who has an intra-abdominal tumor (pregnancy) and the obese patient who has an extraperitoneal tumor (abdominal fat). The purpose of this paper is to identify the nature of the *p*H and volume of gastric juice in preoperative obese patients to determine whether they represent a high-risk group.

METHOD

One hundred and six healthy, non-smoking male and female adult patients scheduled for elective operation were studied. Each patient denied using alcohol in excess. None of the patients was receiving any medication prior to operation. Prior to this study all patients had received nothing by mouth for eight to 11 hours. After premedication with a combination of Innovar (1.5–2.0 ml) and diphenhydramine (50–100 mg), im, or no premedication, infusion of 5 per cent dextrose in Ringer's lactate solution was begun.

Blood-pressure cuff and electrocardiogram monitors were placed when the patient arrived in the operating room.

After preoxygenation and treatment with gallamine (10–20 mg, iv), anesthesia was induced with thiopental (250–700 mg, iv). Succinylcholine (100–120 mg, iv) was used to facilitate endotracheal intubation. Halothane–nitrous oxide anesthesia was then started. A no. 18 French nasogastric tube (sump-type) was placed after intubation and the gastric contents aspirated and retained. Acid concentration of this sample was determined in duplicate using a *p*H meter. The operation was allowed to proceed. Immediately upon opening the peritoneum and before abdominal exploration, the tip of the nasogastric tube was positioned in the dependent portion of the stomach. Any remaining gastric juice was then added to the first aspirate and the total volume recorded.

Four groups of patients were studied:

I-A, obese, no premedication, n = 6

I-B, obese, premedication with Innovar and diphenhydramine, n = 50

II-A, non-obese, no premedication, n = 18

II-B, non-obese, premedication with Innovar and diphenhydramine, n = 32

Obesity was defined as 100 pounds in excess of ideal weight as predicted for height.[4]

Means of the gastric *p*H's and volumes for groups I-A and I-B and II-A and II-B were compared by an analysis of variance to ascertain any effect of premedication both within and among groups. No difference could be found. Subsequently, the gastric sample data were pooled into group I (obese) and Group II (non-obese). The means for these two groups were compared using Students' t test for unpaired data. $P < 0.05$ was considered significant.

Similarities in variability for group populations of *p*H and volume were evaluated using an f test for ratio of group variances. The variabilities for Group I and Group II were dissimilar; therefore, a Wilcoxon two-sample test was performed to evaluate the probability that the two groups represented samples of the same population.

RESULTS

Comparative patient data are presented in table 1. Considering sex distribution and

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TABLE 1. Comparative Patient Data (Means ± SD)

	N	Sex Distribution Male/Female	Patient Age (Years)	Patient Weight (kg)	Patient Height (cm)	Type of Operations
Group I (obese)	56	7/49	36.5 ± 8.8	141.6 ± 25.9	167.3 ± 8.8	Jejunoileal small-bowel bypass
Group II (non-obese)	50	7/43	42.0 ± 13.4	69.2 ± 13.4	166.1 ± 10.8	Cholecystectomy Colonic resection Total abdominal hysterectomy Exploratory laparotomy
Significance of difference		N.S.*	$P < 0.05$	$P < 0.001$	N.S.*	

*Not significant by analysis of variance.

height, there was no significant difference between groups. However, mean weights in the two groups were significantly different; Group I patients, on the average, weighed twice as much as Group II patients. Although one can demonstrate a statistically significant difference between mean ages of the patients in the two groups, this difference is not relevant to this study.

When *p*H's and volumes of gastric juice were compared (table 2), there was a significant difference between Group I and Group II. Of particular interest is the percentage of patients within each group with the combination of a low *p*H (<2.5) and a large volume (>25 ml) of gastric juice. Seventy-five per cent of the obese group, compared with none of the non-obese group (Group II) had this combination (table 2).

Although the types of operations in the two groups differed, we were unable to correlate the presence or absence of bowel prep as a variable influencing the gastric juice findings. There was also little variability in the amounts of gastric juice obtained after the peritoneum was entered. Moreover, there was no relationship between this volume and obesity. However, since the *p*H's of the additional gastric fluid samples showed so much intra- and inter-patient variability for both groups, these samples were not considered to reflect the fasting state.

An attempt to ascertain whether the color of gastric juice could be related to *p*H was unsuccessful (table 2). Color was considered clear, amber, yellow, or green. There was no correlation between *p*H and color of the initial aspirate. Further, there was no difference between the numbers of samples presumed to be bile-stained in the two groups (green versus non-green).

Figure 1 shows the frequency of occurrence of a particular *p*H within each group. Clearly seen is that the two groups studied represent two different frequency distribution curves, *i.e.*, that of Group I is skewed to the left, while that of Group II describes the normal bell-shaped distribution.

Therefore, it seems appropriate to compare the group medians for *p*H rather than the

TABLE 2. Characteristics of Gastric Juice

	Volume* (ml)	*p*H*	Color (Green/Non-green)	Combined (*p*H < 2.5 + Vol > 25 ml)
Group I (obese)	42.3 ± 3.3	1.7 ± 0.11	5/51	42/56 (75 per cent)
Group II (non-obese)	14.7 ± 1.7	3.7 ± 0.18	4/46	0/50 (0 per cent)
Significance of difference	$P < .001$	$P < .001$	N.S.	$P < .001$

* Mean ± SE by Wilcoxon two-sample test.

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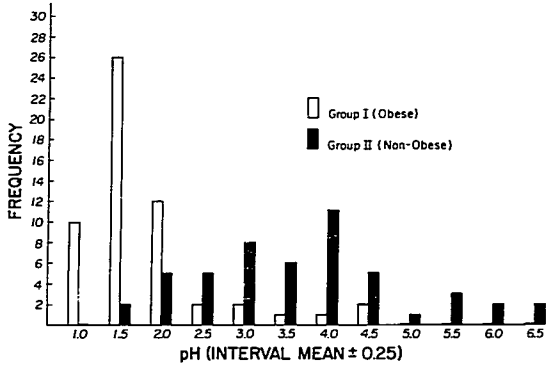

FIG. 1. Frequency distributions of gastric *p*H's in Group I (obese) and Group II (non-obese). Note that the distribution curves differ. While that of Group I is skewed to the left with both median and mode at *p*H = 1.3 units, that of Group II follows the normal bell-shaped distribution, with median at 3.7 and mode at 4.0 *p*H units.

means; this can be done with the use of nonparametric statistical tests (Wilcoxon two-sample). The *p*H medians for the obese (Group I) and non-obese (Group II) patients were 1.3 and 3.7, respectively; the modes were 1.3 and 4.0, respectively.

Similarly, nonparametric statistical analysis of the two groups for volumes of gastric juice shows the dissimilarity between groups. Corresponding volume medians for the obese and non-obese groups are 37.7 and 11.0 ml, respectively. Figure 1 demonstrates this difference with respect to *p*H.

DISCUSSION

Teabeaut[5] was primarily responsible for pointing out that the predominant insult from the aspiration of liquid gastric contents was related to the acidity and volume. Negligible roles were assigned to gastric enzymes and bacteria. It was shown that an aspirate with a *p*H above 2.5 produced little more than the minimal response evoked by distilled water. Furthermore, both Teabeaut[5] and Taylor *et al.*[6] found that as the *p*H of the aspirate decreased to 1.5, pulmonary parenchymal damage increased to a maximum. Therefore, the patient most at risk would be one who had a gastric content volume of 25 ml or more with a *p*H below 2.5.

It has been recognized that gastric emptying time in obstetric patients may be delayed for 24 to 48 hours after food intake.[3] Taylor[7] demonstrated that 55 per cent of intrapartum patients have more than 40 ml of liquid gastric juice, and in 42 per cent of patients the *p*H's were less than 2.5. Our data suggest that obese patients are at even greater risk. Eighty-eight per cent of our obese patients had gastric *p*H's below 2.5, while 86 per cent had more than 25 ml of liquid gastric juice. In addition, 75 per cent of our obese patients had the combination of increased volume and *p*H < 2.5.

These findings in obese subjects could reflect 1) an increase in gastric acid secretion; 2) delayed gastric emptying time; or both. Considering the first hypothesis, there is no evidence in our study or in others to suggest that obese patients have a faster than normal rate of gastric acid secretion. The other hypothesis could, however, explain the observed combination of low *p*H and increased volume. For example, an extraperitoneal mass (abdominal fat) in the recumbent, preoperative obese patient could prolong gastric emptying time. Increasing gastric juice volume would lead to antral distention, promoting gastrin release; subsequent lowering of *p*H by increased parietal cell secretion would be the result.

Our data suggest that particular care should be exercised in the induction of anesthesia in obese patients. Prevention of aspiration would be much superior to treatment. It is our practice to consider the obese patient as having a potential full stomach, even for elective

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operations. One should consider either tracheal intubation under topical anesthesia with the patient awake or a rapid intravenous induction–intubation sequence. This latter method can be accomplished as follows: preoxygenation; pretreatment with nondepolarizing muscle relaxants to prevent fasciculations; rapid induction with intravenously administered agents; modest head-up position; a Sellick maneuver[8]; the trachea quickly intubated with a cuffed endotracheal tube.

Since it is unlikely that the problem of aspiration of gastric contents will ever be completely solved, prevention is superior to *post hoc* treatment.

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Anesthetic Management of a Parturient with Myotonia Atrophica

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Of the three myotonic syndromes, myotonia congenita, paramyotonia congenita, and myotonia atrophica, the last is most feared. It is a hereditary degenerative disease of muscle, especially skeletal muscle, characterized by abnormal delay in relaxation following contraction.[1,2] As the disease progresses, involvement of skeletal, cardiac, and smooth muscle may lead to impairment of many systems. Abnormal pulmonary function, swallowing disturbances resulting in aspiration, EKG abnormalities, and a variety of arrhythmias have been reported.[3–6] In women, the disease is associated with ovarian insufficiency, amenorrhea, infertility, and a high incidence of early abortion in those patients who become pregnant.[7,8] Involvement of the adrenal glands may produce clinical adrenocortical insufficiency in either sex. Evidence for smooth muscle involvement has also been reported.[10] Obstetric patients with myotonia congenita who reach term often have prolonged labor, although too few cases have been described to establish the relationship between myotonia atrophica and abnormal labor patterns.[11] Hydramnios has been reported to occur with myotonia atrophica, perhaps secondary to the inability of the fetus to swallow.[12] Although overt myotonia atrophica in newborn infants has not been reported, neonates born of mothers affected by the disease may initially have difficulty in swallowing and sucking. In view of the disturbed muscle function, and the possibility of multisystemic and smooth muscle involvement, the anesthetic management of patients who have myotonia atrophica, especially obstetric patients, may be difficult.

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Received from Yale University School of Medicine, New Haven, Connecticut 06504. Accepted for publication June 29, 1975.
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