TABLE 1. Serum Cholinesterase Levels Pre- and Post-liver Transplant

| | Pre-transplant | 5 Months Post-transplant | 9 Months Post-transplant | Normal |
|---|---|---|---|---|
| Serum cholinesterase (u) | 40 | 113 | 175 | 90–160 u |
| Dibucaine number (%) | 56 | 89 | 84 | >80 |
| Fluoride number (%) | 50 | 81 | 73 | >61 |

TABLE 2. Liver Function Tests Pre- and Post-liver Transplant

| | Pre-liver Transplant | 5 Months Post-liver Transplant | 9 Months Post-liver Transplant |
|---|---|---|---|
| SGOT (units/l) | 80 | 395 | 170 |
| SGPT (units/l) | 44 | 159 | 233 |
| Alkaline phosphatase (units/l) | 335 | 123 | 148 |
| LDH (units/l) | 262 | 251 | 248 |
| Bilirubin (mg/dl) | 3.7/1.9 | 3.3/2.1 | 2.3/1.4 |
| Albumin (g/dl) | 2.2 | 4.3 | 4.9 |
| PT (pt/control) (s) | 17.2/12.2 | 12.4/12.6 | 11.9/12.3 |
| PTT (pt/control) (s) | 37.8/33.6 | 29.1/31.1 | 34.5/32.3 |

SGOT = serum glutamic oxalacetic transaminase; SGPT = serum glutamic pyruvic transaminase; LDH = lactic dehydrogenase; PT = prothrombin time; PTT = partial thromboplastin time.

donor phenotype (from Pi ZZ to Pi MM).[6] Follow-up studies on these patients show no evidence for reversal to the original phenotype or diminishing alpha l-antitrypsin in the serum levels.[6] Our patient had a similar pattern. Laboratory results showed that the recipient alpha l-antitrypsin phenotype changed and became the same as the donor's when studied 10 months post-transplantation. This patient, in addition, has a new PChE genotype. PChE changed from $E^uE^a$ to $E^uE^u$ after liver grafting.

In summary, two genetic disorders (alpha l-antitrypsin and serum cholinesterase deficiency) were successfully treated by orthotopic liver transplantation. The role of the liver in the pathophysiology of both diseases is demonstrated.

## REFERENCES

1. Gurtner Th, Kreutzberg G, Doenicke A: Comparative studies on cholinesterase activity in serum and liver cells. Acta Anaesthesiol Scand 7:69–82, 1963
2. Kalow W, Genest K: A method for the detection of atypical forms of human serum cholinesterase, determination of dibucaine numbers. Can J Biochem 35:339, 1957
3. Viby-Mogensen J: Correlation of succinylcholine duration of action with plasma cholinesterase activity in subjects with the genotypically normal enzyme. ANESTHESIOLOGY 53:517–520, 1980
4. National Institute of Health Consensus Development Conference Statement: Liver transplantation. Hepatology 4:107S–110S, 1984
5. Alagille D: Alpha 1-antitrypsine deficiency. Hepatology 4:118–145, 1984
6. Hood J, Koep L, Peters R, Schroter G, Weil R, Redeker A, Starzl T: Liver transplantation for Advanced liver disease with alpha-1 antitrypsin deficiency. N Engl J Med 302:272–275, 1986

Anesthesiology
67:274–277, 1987

# Sodium Bicarbonate Buffers Gastric Acid during Surgery in Obstetric and Gynecologic Patients

EVELINE A.M. FAURE, M.D.,* HENRY S. LIM, M.D.,† BARRY S. BLOCK, M.D.,‡
PHEBE L. TAN, M.D.,§ MICHAEL F. ROIZEN, M.D.¶

Buffering gastric acid with nonparticulate oral antacid prior to induction of general anesthesia may reduce pulmonary damage if aspiration ensues. Previous studies in rabbits have shown that severe pulmonary dysfunction resulted from aspiration of acid fluid with a *p*H lower than 2.5, while aspiration of acid fluid with a *p*H of 2.5 or above caused little or no damage.[1]

Aspiration of emulsion type oral antacids, and suspensions of aluminum and magnesium hydroxide caused diffuse histologic changes in the lung tissue of dogs.[2] In contrast, the aspiration of hydrochloric acid (HCl, *p*H 4.5) buffered by a nonparticulate antacid such as Bicitra® caused only a transient decrease in $Pa_{O_2}$ in rabbits.[3]

The ideal preoperative oral antacid should be nonparticulate, fast-acting, and highly effective at low volumes. Sodium bicarbonate ($NaHCO_3$), a clear liquid antacid with a *p*H of 7.5–8.0, meets these criteria. This study was designed to determine the volume ratio of

* Assistant Professor of Anesthesia and Critical Care.
† Professor of Anesthesia and Criticial Care.
‡ Assistant Professor of Obstetrics and Gynecology.
§ Assistant Professor of Anesthesia and Critical Care.
¶ Professor and Chairman of Anesthesia and Critical Care.

Received from the Department of Anesthesia and Critical Care, and the Department of Obstetrics and Gynecology, Division of Maternal-Fatal Medicine, the University of Chicago, Chicago, Illinois. Accepted for publication March 25, 1987.

Address reprint requests to Dr. Faure: Department of Anesthesia and Critical Care, the University of Chicago, 5841 S. Maryland Avenue, Box 443, Chicago, Illinois 60637.

Key words: Antacid: sodium bicarbonate. Gastrointestinal tract: buffer; *p*H.

TABLE 1. Gastric Fluid Volume and *p*H before Instillation of $NaHCO_3$ in Patients Undergoing Elective Gynecologic or Obstetric Surgery

| | # Patients | Mean* Gastric Volume (ml) | Range of Gastric Volume (ml) | Median (Mean†) pH | Range of *p*H |
|---|---|---|---|---|---|
| Group I (gynecologic) | 54 | 29.4 ± 21.7 | 5–115 | 2.34 (1.99) | (1.15–7.90) |
| Group II (obstetric) | 15 | 46.0 ± 19.9‡ | 14–80 | 2.45 (2.04)§ | (1.32–7.45) |
| Total | 69 | 32.5 ± 21.9 | 5–115 | 2.41 (2.00) | (1.15–7.90) |

* Values are expressed as mean ± 1 SD.
† Calculated from [H+], see text.
‡ P < 0.005, group I *vs.* group II.
§ P = N.S., group I *vs.* group II.

$NaHCO_3$ to gastric acid needed to raise the *p*H of the gastric fluid close to 7.0 in surgical patients, and to measure the duration of such buffer protection in patients undergoing elective surgery.

## METHODS

The study included 70 (ASA P.S. I–II) female patients undergoing elective surgery under general anesthesia. Group I consisted of 55 gynecological patients scheduled for elective hysterectomy; group II consisted of 15 obstetrical patients scheduled for elective cesarean section. The study was approved by the Clinical Investigation Committee of the University of Chicago, and informed consent was obtained from each patient. All patients fasted overnight preoperatively. One hour prior to surgery, patients in group I were sedated with meperidine 1 mg/kg and atropine 0.4 mg/kg im. Patients in group II received atropine 0.4 mg/kg im. Patients in both groups received intravenous 5% dextrose in lactated Ringer's solution during surgery. Anesthesia was induced with intravenous thiopental 4–5 mg/kg and maintained with nitrous oxide/oxygen and enflurane, or narcotics in group I, and nitrous oxide/oxygen alone in group II until the delivery of the baby. The tracheas of all patients were intubated following intravenous administration of pancuronium or succinylcholine. Immediately after intubation, a no. 18 French Salem sump nasogastric tube containing an intraluminal epidural catheter was inserted into the stomach. Gastric fluid was aspirated with a bulb syringe several times while repositioning the nasogastric tube, until no gastric fluid could be obtained. The volume and *p*H of gastric fluid were measured at room temperature using a *p*H meter (Radiometer A927, Copenhagen). The total amount of gastric fluid collected was then returned to the stomach through the nasogastric tube. A volume of $NaHCO_3$, 8.4%, equal to 5% of the volume of gastric fluid was then injected into the stomach through an intraluminal epidural catheter to avoid contamination of the subsequent samples by $NaHCO_3$. The patient was placed in Trendelenburg position, and the surgeon was asked to palpate the stomach to verify proper position of the gastric tube and aid in mixing the contents. Samples of gastric fluid were obtained through the main channel of the nasogastric tube, and the *p*H was measured at 10, 30, 60, 90, and 120 min.

## STATISTICAL ANALYSIS

The mean gastric volumes prior to instillation of $NaHCO_3$ of group I and group II were compared by using the Student's *t*-test.[4] Because of the logarithmic property of *p*H, the median *p*H values from group I and group II were compared by using the non-parametric "median test." The level of significance chosen was $P < 0.05$.

## RESULTS

The mean total volume of gastric fluid in 69 patients prior to the injection of $NaHCO_3$ was 32.5 ± 21.9 (1 SD) (range 5–115 ml), with a mean *p*H of 2.00. The *p*H was converted to hydrogen ion [H+] concentration; the means [H+] calculated were then converted to *p*H. We excluded one additional patient undergoing gynecological surgery who had a gastric volume of 500 ml, because we suspected that she suffered from a hypersecretory disorder. The largest gastric volume in the remaining 69 patients was 115 ml, which required 5.75 ml of sodium bicarbonate for neutralization. We observed a difference in gastric volumes and *p*H values between group I and II (table 1). The mean gastric volume was higher in the obstetrical group. The mean volume of $NaHCO_3$ injected was 1.60 ± 1.09 ml (range .4–5.75). Ten minutes after the injection of $NaHCO_3$,

TABLE 2. Median and Mean* *p*H Values of Gastric Fluid at 10, 30, 60, 90, and 120 Min (Mean Volume 1.60 ± 1.09 ml, Range 0.4–5.75 ml) in Patients Undergoing Elective Gynecologic or Obstetric Surgery

| Time (Min) | Median (Mean*) | (Range) | # Patients |
|---|---|---|---|
| 10 | 7.16 (6.83) | (5.62–8.20) | 69 |
| 30 | 7.24 (6.85) | (5.60–8.30) | 69 |
| 60 | 7.10 (6.74) | (5.64–8.30) | 65 |
| 90 | 7.12 (6.12) | (4.74–8.28) | 33 |
| 120 | 6.60 (6.27) | (5.60–7.86) | 19 |

* Calculated from [H+], see text.

TABLE 3. Gastric Volume and *p*H in Patients Undergoing Elective Gynecologic or Obstetric Surgery with *p*H < 2.5 and *p*H >2.5 Prior to Instillation of $NaHCO_3$

| | # Patients | *p*H Median (Mean*) | Range of *p*H | Mean† Gastric Volume (ml) | Range of Gastric Volume (ml) |
|---|---|---|---|---|---|
| *p*H < 2.5 | | | | | |
| Group Ia (gynecologic) | 32 | 1.99 (1.77) | (1.15–2.45) | 34.1 ± 22.4 | 8–115 |
| Group IIa (obstetric) | 9 | 1.92 (1.82) | (1.32–2.46) | 47.8 ± 15.2 | 30–80 |
| Total | 41 | | | | |
| *p*H > 2.5 | | | | | |
| Group Ib (gynecologic) | 22 | 4.12 (3.43) | (2.86–7.90) | 22.9 ± 18.7 | 5–80 |
| Group IIb (obstetric) | 6 | 4.16 (3.46) | (2.82–7.45) | 38.2 ± 26.0 | 14–80 |
| Total | 28 | | | | |

* Calculated from [H+], see text.

† Values are mean ± 1 SD.

the *p*H had risen to a mean of 6.83, and remained in the range of 6.12–6.85 for 120 min in all patients (table 2).

We then compared the data from patients with a *p*H lower than 2.5 from both groups (group Ia and IIa). Fifty-eight percent of the gynecological patients and 60% of the obstetrical patients had a *p*H below 2.5 (mean *p*H 1.77 and 1.82, respectively). The mean gastric volumes in these patients were 34.1 ± 22.4 and 47.8 ± 15.2. No difference was observed between the groups in regard to *p*H or volume of gastric fluid. Table 3 displays the mean, median, and range for *p*H and gastric volume in patients with *p*H below 2.5 and *p*H greater than 2.5.

Ten minutes after the injection of the calculated dose of $NaHCO_3$, the *p*H had risen to a median value of 6.94 in the low *p*H group and 7.61 in the high *p*H group, and the median *p*H remained between 6.55 and 7.65 throughout the 120-min study period. Figure 1 displays the median values and ranges of *p*H in these subgroups.

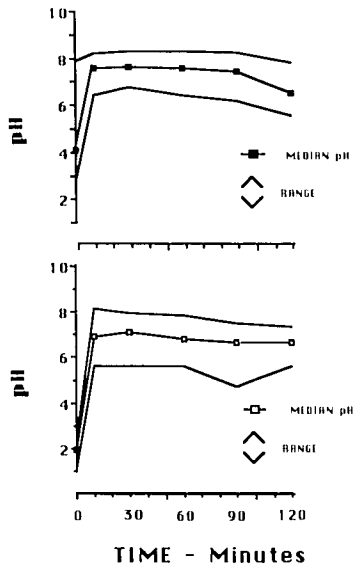


FIG. 1. Median *p*H and range in patients with *p*H < 2.5 (lower panel) and *p*H > 2.5 (upper panel) after instillation of $NaHCO_3$ over 120-min study period.

## DISCUSSION

Sodium bicarbonate, 8.4%, in a volume equal to 5% of gastric fluid volume, was effective in buffering gastric acid to near neutral values. This effect lasted for 120 min. While we attempted to collect all gastric fluid by nasogastric sampling, it is likely that this technique would not consistently yield all gastric fluid. Nevertheless, the volume of gastric fluid that we did obtain appears representative of total gastric fluid, and the dose of bicarbonate given caused neutralization of the measured gastric acid. The sustained *p*H elevation may be explained by inhibition of gastric motility during general anesthesia.

Neutralization of gastric acid has been achieved with several antacids and histamine$_2$ receptor antagonists. Conklin and Ziadlow-Rad[5] reported that 1 ml of Policitra-LC® (*p*H 5.2) or Bicitra® (*p*H 4.5), respectively, buffers 18 ml and 8 ml of 0.1 HCl to a *p*H of 2.5. Gibbs and Banner[6] reported that 30 ml of Bicitra® effectively increased the *p*H of stomach contents above 2.5 in 88% of their patients. Viegas *et al.*[7] measured an increase in gastric *p*H from 2.1 to 6.2 in surgical patients, 15–20 min following an oral dose of 15 ml of sodium citrate (*p*H 8.5). Chen *et al.*[8] evaluated Alka Seltzer® (two tablets dissolved in 30 ml of water) orally in surgical patients, and found gastric *p*H to be above 4 in all patients.

The volume of antacid needed to achieve effective neutralization of gastric acid in the above studies is larger than the volume of $NaHCO_3$ utilized in our study, where a mean of 1.60 ml was found to elevate the *p*H to near neutral levels in most patients. In addition, particulate antacids, such as Maalox and Mylanta®, have been shown to produce lung damage because of tissue irritation from the particulate matter.[3] In contrast, clear liquid antacids, such as Alka Seltzer® dissolved in water, alone and in combination with HCl, did not produce a significant increase in pulmonary shunting when instilled into the lungs of dogs.[8]

Histamine$_2$ receptor blocking drugs, such as cimeti-

dine, and agents that facilitate gastric motility, such as metoclopromide, are effective in reducing both gastric volume and acidity when given orally or intravenously 40–60 min prior to surgery, but are not effective in emergency surgery.[9,10] Histamine$_2$ receptor antagonists interfere with drug metabolism by inhibiting cytochrome P450, decrease liver blood flow, and may produce hypotension and cardiac dysrhythmias.[11–13] Because of the low volume needed to buffer gastric fluid, the liklihood of $NaHCO_3$ causing systemic effects is remote. The percentage of patients with a gastric *p*H lower than 2.5 prior to instillation of $NaHCO_3$ (59%) in our study compares favorably with the percentages found by Stoelting[14] (63%) and Hester[15] (53%) in patients undergoing elective surgery. Also, we found a significant difference in gastric volumes between gynecological and obstetrical patients. Both groups had a mean volume larger than 25 ml, which is considered critical.[1] In patients with a *p*H lower than 2.5, the mean *p*H was 2.00, which compares well with the mean *p*H of 1.6–1.8 found by Stoelting.[14]

In summary, our data suggest that a relatively low volume of $NaHCO_3$ is efficient in raising gastric *p*H to above 2.5 within 10 min, and that this protective effect lasts for at least 120 min.

## References

1. Teabeaut TR II: Aspiration of gastric contents: An experimental study. Am J Pathol 28:51–67, 1962
2. Gibbs CP, Schwartz DJ, Wynne JW, Hood CI, Kuck EJ: Antacid pulmonary aspiration in the dog. ANESTHESIOLOGY 51:380–385, 1979
3. Eyler SW, Cullen BF, Murphy ME, Welch WD: Antacid aspiration in rabbits: A comparison of Mylanta and Bicitra. Anesth Analg 61:288–292, 1982
4. Dixon WJ, Massey FJ: Introduction to Statistical Analysis. New York, Mc Graw-Hill, 1969, p 315
5. Conklin KA, Ziadlou-Rad F: Buffering capacity of citrate antacids. ANESTHESIOLOGY 58:391–392, 1983
6. Gibbs CP, Banner C: Effectiveness of Bicitra as a preoperative antacid. ANESTHESIOLOGY 61, 97–99, 1984
7. Viegas OJ, Ravindran RS, Shumacker CA: Gastric fluid *p*H in patients receiving sodium citrate. Anesth Analg 60:521–523, 1981
8. Chen CT, Toung TJK, Haupt HM, Hutchins GM, Cameron JL: Evaluation of the efficacy of Alka Seltzer effervescent in gastric acid neutralization. Anesth Analg 63:325–329, 1984
9. Hodgekinson R, Glassenberg R, Joyce TH, Coombs DW, Ostheimer GY, Gibbs CP: Comparison of Cimetidine (Tagamet) with antacid for safety and effectiveness in reducing gastric acidity before elective cesarean section. ANESTHESIOLOGY 59:86–90, 1983
10. Manchikanti L, Marrero TC, Roush JR: Preanesthetic Cimetidine and Metoclopramidie for acid aspiration prophylaxis in elective surgery. ANESTHESIOLOGY 61:48–54, 1984
11. Feely J, Wilkinson GR, McAllister CB, Wood JJ: Increased toxicity and reduced clearance of lidocaine by Cimetidine. Ann Intern Med 96:592–594, 1982
12. Freston JW: Cimetidine II. Adverse reactions and patterns of use. Ann Intern Med 97:728–734, 1982
13. Schulze-Delrieu K: Metoclopramide. N Engl J Med 305:28–33, 1981
14. Stoelting RK: Responses to atropine glycopyrrolate, and riopan of gastric fluid *p*H and volume in adult patients. ANESTHESIOLOGY 48:367–369, 1978
15. Hester JB, Heath ML: Pulmonary acid aspiration syndrome: Should prophylaxsis be routine? Br J Anaesth 49:595–599, 1977