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The Effectiveness of Sodium Citrate as an Antacid

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Vomiting or regurgitation of stomach contents and subsequent aspiration during administration of general anesthesia continue to be a leading cause of maternal mortality. Reports from England and Wales indicate that aspiration is the major preventable cause of maternal death during cesarean section.[1] Because aspiration of acidic gastric contents is more harmful than aspiration of nonacidic contents,[2–4] prophylactic administration of antacids has been recommended to decrease gastric acidity.[5] Although antacids are effective, we have shown in dogs that particulate antacids, if aspirated, can produce harmful physiologic and histologic changes.[4] However, sodium citrate, a nonparticulate antacid, is essentially harmless when aspirated, producing only transient hypoxia and minimal tissue changes.[6] Although sodium citrate has been recommended previously for decreasing gastric acidity before general anesthesia,[7,8] the effectiveness of this antacid has been questioned.[9] Also, most reports concerning its use have appeared in England rather than America.[7–9] Thus, we thought it appropriate to reevaluate the effectiveness of sodium citrate for patients undergoing cesarean section. Also, because this agent's potential for elevating the *p*H of large volumes of acidic liquid might not be apparent clinically, we determined this potential *in vitro* and compared the results with results obtained from similar experiments utilizing two particulate antacids.

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METHODS

At intervals ranging from 10 to 47 min before the induction of anesthesia, 30 ml chilled (more palatable) sodium citrate was given to 26 patients scheduled for elective cesarean section. None were in labor and all had fasted overnight. After the induction of anesthesia and endotracheal intubation, a Salem sump tube was placed in the stomach and its contents aspirated through one lumen immediately and through the other just before extubation. The first samples were obtained 12–50 min after the ingestion of the antacid and the second samples 58–195 min after ingestion. The volume and the *p*H of each sample were measured and recorded. The *p*H was measured with an Orion® digital ionalyzer *p*H meter and electrode. Mean *p*H was calculated by adding all values and dividing by the number of samples.[10] The 0.3 M sodium citrate mixture was prepared by the hospital pharmacy according to the following formula: 88.2 g sodium citrate; 100 ml simple syrup; 1.0 ml mint flavor; and 1,000 ml H_2O q.s. The protocol was approved by the Human Experimentation Committee of the University of Florida and informed consent was obtained from each patient.

The *in vitro* portion of the experiment was accomplished by adding hydrochloric acid incrementally to 30 ml each of 0.3 M of sodium citrate, Kolantyl Gel,® and Mylanta.® The latter two antacids are commercially available particulate antacids. The experiment was performed by using hydrochloric acid with three different *p*H values: 0.8, 1.0, and 1.5.

RESULTS

Sodium citrate, 30 ml, elevated the *p*H of the gastric contents to above 2.5 for all 26 patients at the time of the first sample and for 25 of the 26 patients at the time of the second sample. The lowest *p*H in the first sample was 3.2 and the lowest in the second sample was 1.8. The means of the two samples were 5.7 and 5.2, respectively. The volumes, including the total of the first and second samples, ranged from 6 ml to 110 ml. All aspirates were liquid, *i.e.*, they contained no partially

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TABLE 1. Mean Values of Gastric Samples after Intubation (1st Sample) and before Extubation (2nd Sample) Following Preinduction Administration of 30 ml 0.3 M Sodium Citrate

	1st Sample			2nd Sample			
	Time (min)	*p*H	Volume (ml)	Time (min)	*p*H	Volume (ml)	Total Volume (ml)
Mean	26.7	5.7	26.3	80.7	5.3	27.8	54.1
SD	10.3	0.9	22.6	35.3	1.4	22.0	32.0
Range	12–50	3.2–7.1	7–80	58–195	1.8–7.0	6–90	17–110

digested food. The data are presented and summarized in table 1.

Results of the *in vitro* study revealed that sodium citrate can elevate *p*H of larger amounts of hydrochloric acid to above 2.5 than either of the two particulate antacids. Numerical values are presented in table 2.

DISCUSSION

Patients undergoing general anesthesia are most susceptible to hazards of aspiration during induction just before intubation and during emergence just after extubation. Our data indicate that 30 ml of 0.3 M sodium citrate administered before the induction of general anesthesia will maintain the *p*H of liquid gastric contents above 2.5 in nearly all patients throughout the operation. These data support those of Lahiri and colleagues[7] as well as of Holdsworth *et al.*,[8] but are at odds with the work of Hester and Heath.[9]

These latter authors reported that sodium citrate did not elevate the *p*H of gastric contents to above 2.5 consistently. That we achieved better results than Hester and Heath may be explained by the fact that we administered 30 ml of antacid rather than 15 ml. We chose 30 ml because it is the amount of particulate antacid that we have always administered to our patients, as have others.[11] Also, since we administered our antacid before entering the delivery room and since patients moved from the stretcher to the operating table, the movement may have agitated the stomach enough to mix its contents and the antacid thoroughly. That such might be the case is supported by a recent study of Holdsworth and colleagues who found that mixing sodium citrate with stomach contents was enhanced considerably by rotating the patient from supine to left lateral position and, even more, by rotating the patient 360°.[8]

The largest volume of stomach contents encountered in this study was 110 ml. However, because sampling through a nasogastric tube does not ensure retrieval of all material, it is likely that larger volumes were there. Even if larger amounts were present, our *in vitro* data indicate that 30 ml of 0.3 M sodium citrate can elevate the *p*H of large quantities of highly acidic contents to above 2.5. Thus, sodium citrate should be an effective antacid when administered prophylactically before cesarean section to patients who have not eaten solid foods recently.

Whether sodium citrate, or any antacid, effectively elevates *p*H of partially digested food is not answered. However, we have preliminary data indicating that the combination of sodium citrate plus partially digested food is a much more benign aspirate in the dog than the combination of a particulate antacid plus partially digested food.[12]

Since it is well-established that a significant number of patients undergoing elective cesarean section have gastric contents with a *p*H less than 2.5, we did not believe control samples of gastric contents for *p*H measurements were necessary. Sixty-six per cent of patients undergoing elective cesarean section in both the studies of Baraka and colleagues and of Roberts and Shirley were found to have stomach contents with a *p*H less than 2.5.[5,13] Furthermore, we did not wish to deny our patients the benefit of antacid prophylaxis to reestablish control values.

We have shown that 30 ml of 0.3 M sodium citrate effectively elevates the *p*H of liquid stomach contents in patients undergoing cesarean section. Although the administration of this antacid can reduce the severity of aspiration should it occur, the prevention of aspiration lies in the more liberal use of regional anesthesia as well as in the strict adherence to established methods of inducing anesthesia in patients whose stomachs are not empty. Furthermore, because the aspiration of partially digested food is particularly damaging to the lungs,[14] it is imperative for anesthesiologists and obstetricians to warn patients before they enter the hospital about the hazards of eating before delivery and to advise them not to do so.

TABLE 2. Volume (ml) of HCl (*p*H 0.8, 1.0, and 1.5) Neutralized by 30 ml 0.3 M Sodium Citrate, Kolantyl Gel®, and Mylanta®

	Hydrochloric Acid		
*p*H*	0.8	1.0	1.5
Sodium Citrate (8.5)	140	255	750
Kolantyl Gel® (8.1)	100	160	360
Mylanta® (8.0)	75	100	300

* Initial values for each antacid are in parentheses.

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Causalgia of Vascular Etiology Following an Abdominal Injury

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Causalgia is a pain syndrome characterized by chronic, severe, constant, burning pain which frequently is associated with vasomotor disturbances, delayed return of function, and trophic changes.[1] Major causalgia is often seen following penetration of the body by a high-velocity projectile, near or through peripheral nerves in the upper or lower extremity.

An unusual case of severe causalgia affecting the lower extremity secondary to a gunshot wound to the abdomen is described. The uniqueness of this case was the early development of symptoms of causaligia following vascular abdominal trauma with complete absence of peripheral nerve injury.

Report of a Case

A 21-year-old, healthy man was admitted for a gunshot wound to the abdomen. A 38●-caliber bullet entered the right upper quadrant and traveled in a downward direction from right to left and front to back. The exit of the wound was located 1 cm above the left iliac crest at the level of the posterior axillary line.

The patient arrived at the Emergency Room in critical condition and underwent an emergency exploratory laparotomy which included resection of the third and fourth portion of the duodenum, repair of the superior mesenteric vein, and repair of an extensive injury to the aorta below the renal arteries. After surgery, the patient was transferred to the Surgical Intensive Care Unit in stable condition. The postoperative course was uneventful until the fifth day, when he began to complain of burning pain in the left buttock and left leg. Within five days, the pain became unbearable and could not be relieved completely with narcotic analgesics. Four weeks after the accident, the patient was referred to the Pain Clinic for evaluation of the persistent pain. Prior to our consultation, a pseudoaneurysm of the abdominal aorta was ruled out.

When the patient was first seen, the pain was severe, and he would not allow anyone to touch the affected part. The pain was constant and characterized as pulsatile and burning in nature, radiating from the left buttock toward the posterior aspect of the left thigh and calf through the plantar aspect of the foot. The pain was exacerbated with heat and when lying in the right lateral decubitus, but some relief was obtained in the left lateral decubitus position.

On physical examination he was uncooperative, irritable, and extremely anxious. The skin of the left leg was cold and moist with no atrophic changes. The neurlogical examinations before and after the treatment did not show any sensory or motor deficits. The rest of the examination was within normal limits. A diagnosis of causalgia was made, and a diagnostic left lumbar sympathetic nerve block was carried out the same day. A 22-gauge needle was inserted 5 cm lateral to the L2 spine, and 15 ml of bupivacaine 0.25 per cent was injected. The pain disappeared almost completely within a few minutes after the block, and the extremity became warm and dry. There was an average increase of 5° C in the skin temperature in the left toes. The effect of the block lasted for over 10 hours. This procedure was repeated twice with the same results. As symptoms recurred after each block dissipated, and as the intervals between each block did not increase, the patient was advised to have a left lumbar sympathectomy to obtain

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