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Antacid Therapy in Obstetrics

To the Editor: —The recently increasing number of publications on methods of elevating intragastric pH preoperatively is producing considerable confusion within the anesthetic community and diverting attention from the anesthesiologist's primary goal in this respect, that of preventing any soiling of the tracheobronchial tree, whether by acid or antacid.

One investigator¹ finds oral administration of cimetidine 90 min before anesthesia valuable prophylaxis: another² finds the group treated by this procedure "not significantly different from the control group."

Conflicting reports of possible harm from antacid aspiration³ cause further uncertainty among obstetrical anesthesiologists, for whom the use of cimetidine for prophylaxis is bedevilled by timing problems and also the lack of proof of its innocuousness to the fetus or newborn. While we strongly support recommendations that safer antacid preparations be developed,³ we are deeply concerned that some may elect to cease attempts to neutralize the gastric juice in parturients.

A recent case brought to our attention has led us to reconsider the significance of one of our experiments with Rhesus monkeys, performed in 1974.⁴ In this case, which cannot be reported fully because of continuing litigation, a young woman anesthetized without endotracheal intubation aspirated during surgery. The volume aspirated was not massive, yet produced sudden cardiac arrest. The heart was restarted without difficulty, but unfortunately there were neurologic sequelae. The *p*H of the aspirate is unknown.

In the Rhesus monkey study, a tracheostomy tube was inserted into the left lung and 0.4 ml/kg of gastric juice neutralized with THAM to pH 7.45 was injected down the tube. Tachypnea and a slight increase in blood pressure occurred. After half an hour, during which the animal's condition had stabilized and the arterial blood P₀₂ returned to base levels (50–60 torr), the experiment was repeated, but this time 0.4 ml/kg of acidified gastric juice (pH 1.26) was injected into

the opposite lung. Changes in blood pressure, respiratory rate, and pulse rate this time were dramatic, and within 3 min, led to cardiorespiratory arrest accompanied by blood-stained frothy material in the tracheal tube. The arrest was easily reversed by closed-chest compression and artificial ventilation. The similarity between the findings in this study and those in the patient described above is striking.

We are left with the question: Can the aspiration of highly acidic material cause sudden pulmonary edema with spasm and hypoxia sufficient to cause prompt cardiac arrest? If so, dare we not neutralize gastric contents, even at the risk of some pulmonary damage, while the search for a more ideal solution continues? The final answer will probably be difficult to document though easy to define—has this new awareness of the risks of aspiration over the past five years resulted in a decreased overall mortality?

R. BRYAN ROBERTS, M.D. Professor and Chairman Department of Anesthesiology Wright State University School of Medicine Dayton, Ohio 45431

MICHAEL A. SHIRLEY, M.D. Pittsfield, Massachusetts

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Direct (Transthoracic) Endobronchial Intubation

To the Editor: — Traditionally, one-lung ventilation is accomplished by use of a long endotracheal tube, a bronchial blocker, or, most commonly, a double-lumen endobronchial tube.

Recently, a transthoracic endobronchial intubation

was performed during a right thoracotomy and pneumonectomy for epidermoid carcinoma. This technique was chosen after attempts to pass either an endobronchial or a long endotracheal tube into the left mainstem bronchus were unsuccessful. Instead, the