

## Editorial Views

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*Aspiration Syndromes in Pregnancy*

MORE THAN 30 YEARS AGO, Mendelson described the syndrome of pulmonary aspiration of gastric contents in obstetric patients undergoing general anesthesia.<sup>1</sup> Five of 66 patients aspirated solid material, resulting in massive acute respiratory obstruction; at least 40 of the remainder showed a different clinical picture, consisting of cyanosis, dyspnea, wheezing and hypoxia. The latter syndrome has been reproduced experimentally in animals by instilling into the lungs clear solutions of acid or filtered gastric contents having pH values of less than 2.5.<sup>1,2</sup> When the pH of the aspirate was greater than 2.5, the physiologic and morphologic responses were slight and transient; when pH was 1.5 or less, the responses were invariably severe, and resulted in a high mortality rate. These animal findings stimulated studies in obstetric patients, which revealed that almost 50 per cent of parturients have a gastric-fluid pH of less than 2.5.<sup>3</sup> Although the volume of gastric acid necessary to produce a significant pulmonary reaction in man when aspirated is unknown, data obtained from rhesus monkeys led Roberts and Shirley to propose that 0.4 ml/kg, about 25 ml in the average parturient, would be hazardous.<sup>4</sup> Using the criteria of pH less than 2.5 and gastric volume greater than 25 ml to define the "at-risk" population, they concluded that no obstetric patient would be without risk, regardless of the time of the last meal or the onset of labor.

Animal and human data implicating gastric acid as the primary causative factor in aspiration pneumonitis suggested that increasing the pH of gastric contents would provide an easy solution to this problem. Thus, many anesthetists adopted the practice of routinely administering 15 ml of antacid to all women in labor. However, in 1975, Taylor described pulmonary com-

plications following aspiration of gastric contents, of pH 3.5, in a patient who had received antacids.<sup>5</sup> In spite of the fact that the volume of gastric contents in this patient was large (estimated at 1,000 ml) and there were difficulties with tracheal intubation, this case was thought to reflect the inadequacy of current antacid regimens. It was subsequently suggested that a gastric-fluid pH greater than 3.5 should be achieved. To accomplish this, it was recommended that 30 ml of antacid be administered to the laboring patient every two to three hours. Many practitioners now adhere to such a regimen.

Having convinced ourselves that we were improving maternal safety by this practice, it is now difficult to have to consider that this therapy might have potentially dangerous effects. The experimental data presented by Gibbs and associates<sup>6</sup> in this issue, however, force us to do just this. They demonstrated that when a dilute solution of antacid is introduced into the lungs of dogs, the resultant decrease in arterial oxygen partial pressure and increase in pulmonary shunting are as severe as those caused by instillation of hydrochloric acid, pH 1.8. Morphologic changes at 48 hours were severe in both antacid- and acid-treated groups. At one month, however, the lungs of animals that had aspirated acid were normal, whereas those treated with antacids still demonstrated an extensive intra-alveolar cellular reaction. The focus for this florid response appeared to be the antacid particles, which were still visible in samples of lung tissue obtained 48 hours and one month after treatment. A group treated with alkalized saline solution, pH 5.8, showed only transient physiologic disturbances and histologic changes that were similar to those seen in the saline-treated control group.

Do these experimental data have clinical relevance? It would be tempting to dismiss them were it not for the clinical report of Bond and associates,<sup>7</sup> and the previous report of two cases,<sup>8</sup> describing severe physiologic disturbances and in one case, death, following aspiration of gastric contents containing antacid. In all three patients antacid therapy had been instituted during labor, and in two cases significant amounts of antacid were identified in the aspirate. Several possibilities exist to explain why complications developed. Morbidity might have resulted from aspiration of an excessive volume of gastric contents, regardless of pH, or from the presence of food particles in the lung. The latter have been shown to cause severe pneumonitis, either alone or in combination with gastric acid. Bond *et al.*, however, reported that food particles were not present in the aspirate, so that the possibility that aspiration of antacid was a contributing factor cannot be ignored. In any event, one must question the logic of repeatedly administering 30 ml of any fluid to a patient in whom gastric emptying is significantly slowed. Routine antacid therapy may indeed result in the accumulation of large gastric residues of antacid emulsion, posing as great an aspiration hazard to the parturient as gastric acid. Additionally, antacids may themselves stimulate acid secretion and some, particularly aluminum hydroxide, further delay gastric emptying.<sup>9</sup> Some of the problems associated with antacids may be avoidable. As an alternative to particulate-containing solutions, the use of 0.3 molar sodium citrate as a single-dose antacid has been proposed.<sup>10</sup> Preliminary data suggest that its instillation into the lungs of animals produces a much less severe pulmonary insult than that caused by antacid suspensions.

It obviously is essential to ascertain whether morbidity and mortality from Mendelson's syndrome has decreased following the widespread use of antacids. However, nationwide statistics reporting maternal mortality from anesthesia, and specifically from aspiration, are difficult to obtain. Estimates are that anesthesia may be responsible for 5 to 15 per cent of all maternal deaths,<sup>11</sup> and that of these, perhaps half result from aspiration of gastric contents. An additional unknown is the proportion of obstetric patients receiving antacid treatment prior to anesthesia. In the United Kingdom, a triennial report, "The Confidential Enquiry into Maternal Mortality," examines the etiology of every maternal death occurring in that country. The number of deaths from aspiration during 1973–1975, the period after antacids were widely used, was virtually identical to that occurring during 1970–1972, the years just prior to their introduction.<sup>12</sup> In the more recent period, however, all patients who died had received antacids,

whereas only three of 14 in the earlier period had been so treated. A report from New Zealand quotes the incidence of severe pulmonary acid aspiration in all operative cases (including obstetrics) from 1967 to 1969 as one in 11,000.<sup>13</sup> During the period 1970–1972, when the use of magnesium trisilicate was becoming widespread for both obstetrical and surgical patients, the incidence of severe aspiration was reported to be one in 40,000. Mortality from this complication, however, was unchanged; approximately one in 115,000 patients died from aspiration during both periods.

It is estimated that the cesarean section rate in the United States is increasing by approximately 1 per cent per year.<sup>14</sup> About half of these operations are performed with general anesthesia.<sup>15</sup> Thus, pulmonary aspiration of gastric contents is likely to be a continuing problem. Since antacids are not the panacea they originally appeared to be, what else can be done to decrease the incidence of aspiration pneumonitis? There has been an unfortunate tendency for obstetric anesthetists to focus only on gastric-fluid pH, ignoring volume or the presence of particulate matter. Failure to empty the stomach preoperatively may be an important predisposing factor to aspiration. Although passage of a wide-bore gastric tube is unpleasant, it is probably advisable to do this when the patient has eaten after the onset of labor. Technical difficulties in tracheal intubation probably lead to many cases of aspiration, and may be avoided by careful preoperative assessment and the availability of appropriate equipment and experienced personnel. Awake tracheal intubation should also be considered, particularly when the patient has a difficult airway. Alternatively, more widespread use of regional anesthesia would almost certainly contribute to decreased maternal mortality from aspiration.

Various pharmacologic approaches to decreasing the acidity and volume of gastric contents have been pursued in recent years. Cimetidine, a histamine H<sub>2</sub> receptor antagonist, inhibits gastric acid secretion,<sup>16</sup> although volume is unaffected by this agent. Metoclopramide, a drug that will soon be available in this country, increases gastric emptying by promoting peristalsis and relaxing the pyloric sphincter.<sup>17</sup> In addition, metoclopramide is a potent antiemetic and increases tone in the lower esophageal sphincter, thereby tending to discourage gastroesophageal reflux.<sup>18</sup> Further research on these drugs is needed, particularly with respect to their effects on the newborn, before they are introduced into clinical practice. The use of anticholinergic agents to inhibit gastric-fluid acidity has also been suggested, in spite of the relaxant effect of these drugs on the gastroesophageal sphincter. Stoelting<sup>19</sup> was unable to demonstrate an increase

in gastric-fluid pH from either atropine, 0.4 mg, or glycopyrrolate, 0.2 mg, although Baraka *et al.*<sup>20</sup> reported that glycopyrrolate, 0.4 mg, decreased from 66 to 34 per cent the number of parturients with gastric-fluid pH values of less than 2.5.

How should the practicing anesthetist respond to this confusing mass of information? The reader who is looking for hard data regarding the safety and effectiveness of antacid prophylaxis will be disappointed. Although there is no question that antacids increase the pH of gastric contents, evidence that their routine use decreases maternal mortality is totally lacking. Until the value of antacids is established by means of a large-scale, prospective randomized study, each practitioner must decide individually whether their use is indicated. Meanwhile, for those who continue to employ them, more precise comparisons of antacids are needed to determine which are the safest. Aluminum hydroxide, present in most proprietary antacids, seems to be a particularly noxious ingredient.<sup>3,9</sup> If sodium citrate is not of value, then surely the development of an effective but non-irritant antacid is not beyond the scope of the pharmaceutical industry.

Finally, a yardstick is needed by which to measure our successes and failures. Improved reporting and analysis of maternal deaths from anesthesia are essential. All too often, these deaths are included only under other categories, such as deaths from cesarean sections or pulmonary complications, making it impossible to extract the relevant data. Obstetric anesthetists must become involved in such data collection. Only when better statistics are available will we be able to assess whether we are really making any progress.

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