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NPO after Midnight for Children—A Reappraisal

ANESTHESIOLOGISTS have worried about pulmonary aspiration of gastric contents for many years. This concern has resulted in diverse modifications of practice designed to reduce the apparent "risk." Schreiner *et al.*, as well as other investigators, in this issue of ANESTHESIOLOGY, have begun to question the need for strict fasting guidelines; clear fluids, offered up to 2 h prior to scheduled induction, apparently do not increase the "risk" for pulmonary aspiration syndrome in children.[1-5] This change in practice offers the significant advantage of a more humane anesthetic experience for both children and their families. There may also be hidden benefits, *e.g.*, avoiding anesthetic induction in the severely hypovolemic infant, and bypassing the recent controversy over using glucose-containing solutions intraoperatively.[6,7]

The basis for our concern regarding pulmonary aspiration during general anesthesia may date to the first reported pediatric death (1848), although this may have been caused by the anesthesiologist pouring brandy into the patient's mouth to relieve "syncope."[8] Winternitz examined the relationship between acid and the clinical syndrome of pulmonary aspiration (1920).[9] Hall described pulmonary aspiration in pregnant patients (1940).[10] However, it is Mendelson (1946) who is generally credited with describing the pathophysiology of the acid aspiration syndrome, hence the eponym "Mendelson's Syndrome."[11] His paper sparked interest in the problem and early studies of anesthesia-associated mortality indicated that a very significant proportion of anesthetic deaths were directly related to pulmonary aspiration of gastric contents.[12-13] This observation led to many changes in anesthetic practice, each of which was intended to reduce the "risk." The routine, preinduction, mechanical aspiration of stomach contents and the pharmacologic evacuation *via* apomorphine-induced vomiting became common though neither of these techniques was particularly appealing to parturiants or most other patients.[12-14] Patients and anesthesiologists were equally negative in their acceptance of balloon-tipped catheters placed in the esophagus or gastroesophageal junction to mechanically obstruct vomitus or regurgitation.[15-16] Awake intubation or regional anesthesia were suggested as safer alternatives.

The cuffed endotracheal tube and the development of barbiturate and succinylcholine "rapid anesthetic induction" seemed to offer advantages over induction of anesthesia *via* mask; cricoid pressure (Sellick's maneuver) appeared to further reduce the "risk" of aspiration.[17-19] Concerns about barbiturate-induced hypotension or vomiting prior to onset of muscle relaxation led to varying recommendations regarding body position ("head up," "head down," "head up but legs flexed," "lateral position"); the use of muscle relaxants prior to intubation was also questioned.[12,15,17-22] The rapid induction technique was initially greeted with resistance and considered by some to be more dangerous than mask induction with 100% nitrous oxide![23]

The next focus of investigation was the pathophysiology of aspiration pneumonia. A $pH \leq 2.5$ was thought to be critical to the development of classic Mendelson's Syndrome in humans. Other factors were found to be contributory (pH, volume, particulate matter *vs.* liquid, composition of particulate matter), but a low pH and large volume of aspirate were most important.[24-29] The consequences of aspiration of gastric contents were divided into pneumonia due to aspiration of particulate matter (mechanical obstruction of airways), which tends to be

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less severe compared with classic acid aspiration (a chemical injury) that results in immediate, severe pulmonary damage. Appropriate preparation of surgical patients was stressed as was the period of fasting to assure an "empty stomach."[30]

Research then turned toward methods to counteract gastric acidity; preoperative antacids found their way into routine anesthetic practice.[31] Again much debate focused upon which antacid was best and focused on issues related to increasing gastric volume with the antacid, the pulmonary damage induced by some antacids, and the virtue of nonparticulate *versus* particulate antacids.[31-37] Much to the delight of drug companies, anesthesiologists next directed their research toward reduction of "anesthetic risk" through pharmacologic means; H_2 receptor antagonists were found to be effective in both reducing gastric residual volume and increasing *p*H.[38-40] Current research is aimed at discovering the agent that lasts the longest with the fewest side effects. Other medications have been shown to improve gastric emptying even in preoperative emergency surgical candidates. Fortunately, most investigators have not concluded that these medications be routinely administered.[41-43]

Clearly we have had great success in reducing the potential "risk" for pulmonary aspiration of acidic gastric contents, but what is the real present risk, particularly in routine healthy elective surgical patients? The basis for our concern must be re-examined in light of modern anesthetic practice. Several authors (myself included) have suggested that the critical combination to be avoided was a volume of 0.4 ml/kg and a *p*H < 2.5.[44] The unpublished data from rhesus monkeys by Roberts and Shirley has been quoted for many years as the necessary conditions for pulmonary aspiration of gastric fluid, *i.e.*, 20–25 ml for the average adult and 0.4 ml/kg for children.[37] In fact, a recent communication suggests that fluid with *p*H 1 and volume 0.4 ml/kg had minimal effect in rhesus monkeys.[45]

A critical examination of the original data reveals that decreasing *p*H and increasing volume are both important in rat, rabbit, guinea pig, and dog models, but the precise volume and *p*H have never been established for humans (obviously such a study is not ethically acceptable).[24-28,41-43,46] Furthermore, these papers assume that every milliliter of gastric fluid is regurgitated and then magically funnelled directly down the trachea as in the animal studies! Realistically, a much greater volume is likely to be required.[47-48]

There is no question that specific anesthetic conditions predispose to potential gastric aspiration. Pregnancy, a recent meal, physical injury, narcotics, mechanical or functional obstruction to digestion, gastroesophageal junction dysfunction, previous esophageal surgery, obesity, head injury, neurologic damage, in-coordination of swallowing and respiration, depressed level of consciousness (drugs, alcohol, general anesthesia), tracheostomies, and even lack of experience of the anesthesiologist have been associated with an increased incidence of pulmonary aspiration.[9-14,41-43] The competent anesthesiologist will recognize most of these circumstances and conduct his/her anesthetic accordingly (regional anesthesia, awake intubation, rapid sequence induction, and the complete retinue of acid aspiration prophylaxis).

However, there are two questions still to be answered: how common is clinically important pulmonary aspiration in the routine, healthy, elective, surgical patient with no recognized risk factors (adult or pediatric), and is it necessary for these "healthy"patients to abstain from ingesting both liquids and solids as long as current recommendation suggest?

Early studies of pediatric surgical patients reported anesthetic mortality from pulmonary aspiration at 39%; this high mortality rate reflected the lack of recognition of risk factors.[49] Certainly this is not true of present anesthesia practice. Olsson *et al.* retrospectively examined 185,358 anesthetic records in Sweden; the incidence of aspiration was threefold greater in children (0–9 yr) compared with adults (20–49 yr) (9/10,000 *vs.* 3/10,000).[50] Only 15 patients without known risk factors and who were having elective surgery were reported and in ten of these there was difficulty with airway management. More importantly, even when aspiration occurred, the majority of patients developed minimal symptomatology. There were no deaths reported in the elective surgical group and the overall mortality rate was 1:46,340. Tieret *et al.* prospectively examined the French experience with 40,240 pediatric patients (0–15 yr).[51] Twenty-seven major complications were reported with an eightfold greater incidence in children less than 1 y of age. Four children were reported to have aspirated (1:10,000); there was one death (1:40,000) unrelated to aspiration. The rate of clinically significant aspiration in routine healthy patients is apparently exceedingly low. A recent abstract has begun to examine the American experience: Borland *et al.* prospectively examined 10,000 pediatric anesthetic cases and documented ten cases of gastric fluid aspiration during the perioperative period.* Interestingly, the incidence was directly related to ASA physical status; five patients had known predisposing risk factors. The incidence in ASA physical status 1–2 patients was 5:10,000 cases and all recovered uneventfully; a more detailed analysis awaits publication of the complete data. These studies suggest that clinically important aspiration of gastric contents is

* Borland LM, Saitz EW, Woelfel SK: Evaluation of pediatric anesthesia care. Presented at the Section on Anesthesiology, American Academy of Pediatrics, March 1989 (abstract 23 and personal communications).

a rare event in healthy elective pediatric surgical patients; routine prophylaxis does not appear to be indicated.

What is the optimal period of fasting for children (or adults) that does not increase "risk"? Studies of gastric physiology have revealed that gastric emptying is dependent upon a number of factors: distension of the stomach, the type of gastric contents (solid *vs.* liquid), and the composition of the gastric contents (fat, protein, sugar).[52] The volume of liquid emptied from the stomach over time is proportional to the volume of fluid present; in adults, 250 ml of a 500 ml volume of isotonic saline will be emptied in 12 min.[53]

Several recent reports studying the duration of fasting and the timing of preoperative fluids have courageously re-evaluated what has been held as a sacred and necessary part of "safe anesthesia practice," *i.e.,* fasting from midnight.[1–5] This issue is particularly important for children in whom fasting is difficult and, when prolonged, may result in hypovolemia and/or hypoglycemia. The investigations of Schreiner *et al.* and other similar reasonably well-controlled studies compared standard fasting *versus* clear liquids (3 ml/kg to *ad lib* fluids) 2–3 h before scheduled induction.[1–5] In most of these studies there was no difference in gastric residual volume or *p*H between those patients who fasted for 8 h or more *versus* those who fasted 2–3 h! On the basis of their studies, several major pediatric institutions have now altered fasting guidelines with at least one institution offering clear fluids upon admission to the Day Surgical Unit. Although it may be premature to change our anesthetic practice based on the results of several small series of patients, these studies are an important starting point for future investigation. Large, well-controlled studies may result in significant modification of current fasting guidelines and may make anesthesia safer and less difficult for children (and adults).[6]

This brings us to other questions: If clinically important aspiration is a rare event in children (or adults), why all the papers directed at preventing it? Is this really a problem worth all the money and energy devoted to it? Are pharmaceutical companies in some kind of pharmacologic race to develop the "most effective" drug while playing on our fear of medical-legal issues? Is aspiration pneumonia in routine healthy patients a nonissue? Clearly, we have the means to pharmacologically reduce the risk of acidic gastric fluid aspiration; whether the drug effects last 2, 4, and 10 h probably makes little difference. I believe that we have had enough publications directed at preventing a problem that may not be clinically important, and suggest instead that we focus our attention upon fasting guidelines, and the type, timing, and volume of fluid that is "safe" for elective surgical patients to consume with and without premedication.

Another point of interest is that recent investigations as well as retrospective outcome reviews have indicated greater neurologic injury when hypoxemia occurs in the presence of hyperglycemia.[7] On the basis of these studies it has been suggested that intraoperative blood glucose values be maintained within the normal range. Logically, this leads us to question the routine use of glucose-containing solutions in the operating room where there is always the potential for hypoxemia. However, some pediatric anesthesiologists have also been concerned about the danger of undiagnosed intraoperative hypoglycemia, particularly in children who have fasted excessively. The practice of offering clear glucose-containing solutions 2–3 h prior to surgery may circumvent this ongoing controversy regarding the intraoperative use of glucose-containing iv solutions. Oral glucose-containing solutions should correct any tendency toward preoperative/intraoperative hypoglycemia for most patients. Such alteration in practice may provide an additional benefit: the incidence of severe hypotension during mask induction, due to relative hypovolemia in fasting infants and small children, may be reduced.

Studies such as Schreiner *et al.* should encourage further investigation of this sacred caveat called "preoperative fasting." I encourage others to conduct large prospective investigations to scientifically decide this issue, to lead us to pediatric anesthesia care that is both safe and more humane.

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