Aspiration pneumonitis and the anaesthesiologist

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Summary

Aspiration pneumonitis remains an important cause of morbidity and mortality in any anaesthetic practice. The pathophysiology, pre-operative prophylaxis and treatment of this syndrome are briefly discussed.


In 1946 Mendelson1 reported a series of cases in which patients were thought to have aspirated stomach contents. His studies in animals were the first to report that aspirated liquid with a low acidic pH produced chemical burns of the lung. Numerous studies2-8 have demonstrated that aspiration is a potential problem in general anaesthetic practice. Conditions that predispose to aspiration are those which reduce the level of consciousness with compromise of glottic closure and the cough reflex, oesophageal dysfunction due to intrinsic or neurological disease, mechanical or anatomical interference with the lower oesophageal sphincter, delayed or absent gastric emptying (as in pregnancy, bowel obstruction, trauma, peritonitis, or after narcotics) and recent food ingestion. In addition many unpredictable factors predispose to peri-operative aspiration, such as sudden regurgitation of gastric contents, lower oesophageal sphincter incompetence, and uncertainty about food ingestion. The risk of aspiration is not confined to emergency operations. In only 60 - 80% of elective surgical patients1 is the gastric pH greater than 2,5 (that is, the critical pH below which acid aspiration produces a marked pulmonary inflammatory reaction3). A gastric residual volume of 0,4 ml/kg body weight is considered to predispose to a significant risk of aspiration.4 The risk of aspiration extends throughout the operative period, extubation being as hazardous as intubation.

The obstetric patient is particularly susceptible to aspiration. In reported series of maternal deaths due to anaesthesia, as many as 30% are caused by vomiting and aspiration.5 During pregnancy progesterone decreases gastro-intestinal motility and relaxes the lower oesophageal sphincter. The gravid uterus mechanically obstructs the duodenum and increases intragastric pressure during labour. There may be an increase in gastric acidity secondary to stimulation of the parietal cells by gastrin. During labour gastric emptying decreases with the accumulation of gastric juice and gas. These conditions decrease barrier pressure (lower oesophageal sphincter pressure minus intragastric pressure) and greatly increase the risk of regurgitation. Whatever the time interval between the last meal and either onset of labour or delivery, an empty stomach cannot be guaranteed.

Pathophysiology

Acid burn leads to epithelial degeneration of the bronchi, pulmonary oedema, and haemorrhage. Type I alveolar cells undergo necrosis and free, laminated inclusion bodies are seen in the pulmonary transudate. Within a few hours of aspiration, there is acute infiltration of polymorphonuclear cells and fibrin begins to be deposited in the alveolar space. Degeneration of alveolar type II cells and further necrosis of type I cells with detachment from the basement membrane are frequent. Within 24 - 36 hours marked polymorphonuclear infiltration results in alveolar consolidation and damage to the airways, which may give rise to mucosal sloughing and later organization of hyaline membranes. The lungs appear to be boggy, oedematous, and haemorrhagic. After 72 hours there is regeneration of bronchial epithelium, proliferation of fibroblasts, and a decrease in acute inflammation.9 Hypoxia occurs within seconds of the aspiration of acid; arterial oxygen tension (PaO2) is the most sensitive diagnostic test when aspiration of gastric contents is suspected. Reflex airway closure takes place and changes in surfactant lead to alveolar instability and atelectasis.4 There is a loss of alveolar capillary integrity, with the exudation of fluid and protein into the alveoli and bronchi. The interstitial and alveolar oedema further compromises the airway. The volume of fluid loss varies from patient to patient, and can even result in hypovolaemic hypotension. Finally alveolar haemorrhage and consolidation occur. The net result is an increase in airway resistance, a decrease in pulmonary compliance and functional residual capacity, and a large increase in intrapulmonary physiological shunt, resulting in arterial hypoxaemia.9 During the early stages compensatory hyperventilation often takes place causing a respiratory alkalosis and decreasing the partial arterial carbon dioxide pressure (PaCO2). Hypercapnia signals the inability to compensate, which is a late sign. However, metabolic acidosis may also occur secondary to inadequate oxygen delivery to the tissues.

Pre-operative prophylaxis

Present investigations are focusing on the use of clear antacids and parenteral medications that will increase gastric emptying, alter gastric acidity or decrease gastric secretion. Pre-operative use of antacids increased following successful gastric acid neutralization. However, sporadic reports have appeared describing pulmonary aspiration syndromes with severe pulmonary lesions and respiratory embarrassment, despite a gastric pH greater than 2,5.9 The pulmonary lesion appears to be secondary to the material itself rather than to its alkaline pH, because the aspiration of alkaline saline does not produce similar changes. Before abandoning the use of antacids, sodium citrate should be reconsidered for its positive attributes; it is non-particulate and produces minimal injury when instilled into the lungs. An amount of 30 ml of 0,3M sodium citrate has been shown to neutralize effectively a significant volume of acid.10 While the search for a highly effective, safe, aqueous antacid preparation continues, gastric acid neutralization with sodium citrate may provide a reasonable substitute for the questionable particulate
antacid suspensions. When patients aspirate stomach contents, the material frequently contains small, non-obstructive particles of foodstuff. The aspiration of such material has been shown to cause a prolonged inflammatory response clinically similar to that caused by acid.

A limited role is to be anticipated for the use of anticholinergics, including glycopyrrolate, except as synergists with histamine H₂-receptor antagonists. The only advantage of glycopyrrolate is its apparent safety in obstetric patients, as it is a quaternary ammonium compound with limited placental transfer. Premedication with glycopyrrolate will result in a gastric pH greater than 2.5 in almost all parturient candidates for elective caesarean delivery. Metoclopramide and domperidone (both dopamine receptor antagonists) act on the dopamine receptors in the chemo-ematic trigger zone to produce an anti-emetic effect. They also block peripheral dopamine receptors in the gastrointestinal tract, producing a rise in lower oesophageal sphincter pressure, dilatation of the pylorus, increased gastric peristalsis, and a more rapid rate of gastric emptying. Atropine and opioids, especially those with vagolytic activity, block the action and decrease the usefulness of metoclopramide. However, more work needs to be done on their safety in respect of the fetus, and on their efficacy as prophylactics against acid aspiration.

Cimetidine, a histamine H₂-receptor antagonist, has been shown to be effective in reducing gastric acidity before induction of general anaesthesia. Additional advantages are that it has no effect on gastric emptying but decreases gastric volume. For elective surgery in adults, cimetidine 300 mg given as an intravenous infusion over 15 minutes 1 hour before induction increases the gastric pH and decreases the gastric volume. Multiple oral doses seem more effective than a single dose, and an interval of 1 - 2 hours is necessary for it to be effective in lowering gastric pH, an interval probably reflecting the time needed for gastric emptying. As significant pulmonary changes may occur when cimetidine is aspirated, a sufficient time interval for gastric emptying should be allowed. Only a few reports of the use of cimetidine in emergency situations are available. The rapid intravenous administration of cimetidine before induction is ill-advised as it can produce dysrhythmias and hypotension. Although preliminary experience seems to indicate that the cimetidine levels achieved in the fetus do not produce either respiratory or cardiovascular depression, the data are not yet extensive enough to support its general use for caesarean section.

The problem of the value of prophylactic cimetidine for paediatric patients is similarly unresolved. As the risk of aspiration with extubation is equal to that with intubation, cimetidine therapy should be continued for at least every 4 - 6 hours, preferably by monitoring gastric pH. Cimetidine will be more effective if gastric volume is decreased by nasogastric suction. Prophylactic cimetidine is, however, no substitute for a correct anaesthetic technique. A number of histamine H₂-receptor antagonists, including tiotidine and ranitidine, are under development or undergoing clinical trials. Under study are the methyl-substituted E-prostaglandins, which inhibit gastric acid secretion and also protect the mucosa against numerous insults, including fever, acid, and alkali.

Treatment

Any patient at risk should be positioned so as to minimize the risk of aspiration in the event of regurgitation. Whenever possible the trachea should be intubated before the patient loses consciousness. If this is not feasible then a rapid-sequence induction with cricoid pressure (Sellick's manoeuvre) should be performed. The application of cricoid pressure is important because the protection of the patient from inhalation of vomit during induction depends solely on this one manoeuvre. Once aspiration is thought to have occurred, the airway should be cleared and any aspirate remaining in the oropharynx suctioned. If the patient is not awake and in command of his reflexes, endotracheal intubation should be performed, the cuff inflated and the trachea suctioned. Arterial blood should then be drawn for blood gas analysis.

Active continuous positive airways pressure (CPAP) with spontaneous breathing should be applied, progressing to intermittent or continuous mandatory ventilation if necessary. The CPAP should be titrated to minimize pulmonary shunt fraction without affecting cardiac output. Hypovolaemia from the shift of intravascular fluid into the lungs must be corrected. A pulmonary artery catheter should be inserted if maintenance of fluid balance and cardiac output become a problem. The prophylactic use of antibiotics can disturb the normal flora of the airway, thus facilitating the establishment of infection with more virulent and resistant organisms, and is therefore not indicated except where faecal material has been aspirated. Should pulmonary infection become clinically evident, the patient should be treated with the antibiotic specific for the organism(s) cultured. The use of corticosteroids has failed to improve pulmonary function or survival rates and is not recommended.

Conclusion

An effective prophylactic regimen before surgery, a good anaesthetic technique, and constant vigilance are important in preventing aspiration. While aspiration of acidic stomach contents produces the most severe pulmonary injury, neutral fluids and foodstuffs are certainly not innocuous. Treatment must be prompt and focused on aggressive pulmonary and cardiac support if recovery is to be rapid.

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REFERENCES

5. Awe WC, Fletcher WS, Jacob SE. The pathophysiology of aspiration pneumonitis. Surgin 1966; 60: 232-238.