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REVIEW ARTICLE

Pulmonary aspiration of gastric contents in anaesthesia

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Pulmonary aspiration of gastric contents is one of the most feared complications of anaesthesia. Prevention of aspiration by identification of patients at risk, preoperative fasting, drug treatment and various anaesthetic manoeuvres are cornerstones of safe anaesthetic practice. Not surprisingly, the number of articles and case reports regarding the pulmonary consequences of gastric aspiration is vast and recommendations are often conflicting. We consider briefly the pathophysiology of this condition and examine the evidence regarding incidence, morbidity and mortality, and possible treatment options.

In particular, we will consider if our fear of aspiration is exaggerated and what evidence supports the routine use of pharmacological agents to decrease gastric acidity and volume.

Pathophysiology

The pulmonary consequences of gastric aspiration fall into three groups: (i) particle-related, (ii) acid-related and (iii) bacterial.

Particle-related complications

Acute airway obstruction leading to arterial hypoxaemia may cause immediate death. Prompt removal of inhaled particles, oxygenation of the patient and prevention of further aspiration by tracheal intubation are essential for survival.[4 103] Such events in the perioperative period are usually readily apparent.

Acid-related complications

The concept of a critical pH and volume of aspirate was introduced in 1974 by Roberts and Shirley[91] from data obtained in rhesus monkeys. The results were extrapolated to humans to identify patients at risk of pulmonary aspiration and allow subsequent studies to make categorical statements about safety. The critical pH of 2.5 and critical volume of 0.4 ml/kg body weight (or approximately 25 ml) have since been challenged. Schwartz and colleagues[93] reported in a study on dogs that aspiration of gastric contents of pH 5.9 at 2 ml kg^{-1} caused severe hypoxia and increased pulmonary shunting. If food particles were present, hypercapnia resulted, and acidosis and pneumonitis developed. No deaths occurred within 48 h. Similarly, aspiration of bile with a pH of 7.19 caused severe chemical pneumonitis and non-cardiac pulmonary oedema in the pig.[86]

Instillation of hydrochloric acid into the trachea of monkeys produces mild radiological and clinical changes after volumes of 0.4 and 0.6 ml kg^{-1}, but not death. The LD_{50} was 1.0 ml kg^{-1}. Extrapolation of these data would give a critical volume for severe aspiration in adult humans of approximately 50 ml.[87]

The harmful effects of acid aspiration may occur in two phases: (i) immediate direct tissue injury and (ii) subsequent inflammatory response.[28 48 52] A chemical burn occurs within 5 s from the central airways to the alveoli and within 15 s all of the acid has been effectively neutralized. Desquamation of the superficial cell layer with complete loss of ciliated and non-ciliated cells occurs within 6 h.[118] Regeneration is seen after 3 days with complete recovery after 7 days. Alveolar type II cells are markedly sensitive to hydrochloric acid and degenerate within 4 h after acid aspiration. A rapid increase in lysophosphatidyl choline within 4 h after acid exposure may lead to an increase in alveolar permeability and increased lung water.[3 29] Increased lung water reduces lung compliance, increases ventilation–perfusion mismatching and increases the alveolar–arterial oxygen tension difference.

The second phase is characterized by acid-mediated induction and release of pro-inflammatory cytokines such as tumour necrosis factor alpha (TNFα) and interleukin-8 (IL-8). These may in turn trigger expression of the adhesion molecules L-selectin and beta-2 integrins on neutrophils, and intercellular adhesion molecules (ICAM) on lung

endothelium, further promoting a neutrophilic inflammatory response.[30 69 77]

Localized aspiration leads to a generalized inflammatory response with possible cardiopulmonary failure.[68] Thromboxane-dependent neutrophil sequestration in acid aspiration-induced lung injury may occur but evidence regarding the benefit of selective thromboxane inhibition is conflicting.[28 117]

In summary, gastric aspiration may combine a particulate injury that causes focal inflammatory changes and foreign body reaction, with a diffuse acidic damage. Particle and acid aspiration synergize to increase alveolar capillary leak.[19 53]

Bacterial-related complications

Gastric contents are not sterile and community-acquired lung infections after aspiration are usually caused by anaerobes. Mixed aerobes–anaerobes are found in hospital-acquired aspiration pneumonia.[46] *Pseudomonas aeruginosa*, *Klebsiella* and *Escherichia coli* account for most Gram-negative nosocomial pneumonias whereas *Staphylococcus aureus* is the main Gram-positive pathogen. This pattern has not changed over recent years[22] and is similar in both children and adults.[11] Gram-negative and ventilator-acquired pneumonias, 34% of which are caused by aspiration of gastric contents and oropharyngeal secretions, are thought to be significant determinants of death in postoperative pneumonia.[71]

Patients who develop nosocomial pneumonia often have altered oropharyngeal flora, mainly Gram-negative bacilli. Transfer of gut flora into the lungs by aspiration may occur since in 87% of intensive care patients, one or more organisms were cultured simultaneously from the upper airway and stomach[76].

Detection of gastric aspiration

Asymptomatic aspiration of gastric contents can occur during sleep in 45% of normal individuals and in up to 70% of patients with depressed consciousness.[43] Diffuse rales, wheezing, tachypnoea, tachycardia and low grade fever are clinical signs suggestive of pulmonary aspiration. Invasive investigations may confirm aspiration, such as fibreoptic bronchoscopy, bronchoalveolar lavage, percutaneous needle aspiration and open lung biopsy. Fibreoptic bronchoscopy is recommended as a first-line approach in suspected foreign body aspiration[15] and erythema at the major bronchial carina is observed after aspiration.[14] However, bronchoalveolar lavage and protected brush specimens are more useful in the diagnosis of nosocomial pneumonia than pulmonary aspiration of gastric contents. Percutaneous needle aspiration and open lung biopsy may yield a definitive diagnosis but are associated with a high complication rate and are unhelpful immediately after aspiration.[67] Less invasive methods include chest x-ray and scintigraphic studies. Diffuse infiltrates or consolidation of dependent pulmonary segments may be seen. Radiographically visible infiltrates are almost always apparent within several hours and improve over the next 48–72 h. An increase in infiltrate suggests superinfection or retained secretions.[4 31 111] However, the radiographic signs are not pathognomonic. Scintigraphic studies can be useful in detecting silent aspiration,[107] and foreign body aspiration can be detected in children by ventilation–perfusion imaging.[62] Measuring the glucose content of tracheal aspirates as a marker of aspiration in tube feeding patients is unhelpful.[49] Continuous oesophageal pH monitoring has been used in children[73] but has not been assessed in the adult general surgical patient.

In summary, if aspiration is not witnessed, or tracheal suction does not yield gastric contents or enteral feed, there is no specific diagnostic test to confirm pulmonary aspiration of gastric contents.

Incidence, morbidity and mortality

General surgical patients

It is often stated that the incidence and associated morbidity and mortality of pulmonary aspiration of gastric contents is high in the general surgical population.[21 75] Large studies on the incidence of aspiration and its associated mortality during general anaesthesia have only become available in the past two decades (Table 1). Only two studies were concerned primarily with pulmonary aspiration.[82 112]

A Swedish study of 185 385 general anaesthetics in 1986 found an aspiration rate of 1 in 2131. There were four fatalities (1 in 45 454 patients undergoing general anaesthesia): one was in an intensive care ASA IV patient undergoing gastrectomy, another was in a patient with kyphoscoliosis and respiratory insufficiency, and in the other two there was failed intubation during resuscitation. Forty-seven per cent of patients with reported aspiration developed aspiration pneumonitis and 17% required mechanical ventilation.[82] A 6-yr retrospective study from the Mayo Clinic of 215 488 general anaesthetics for elective and emergency surgery between 1985 and 1991 found an incidence of pulmonary aspiration of 1 in 3216. Three fatalities occurred in ASA III–V patients, resulting in a mortality rate of 1 in 71 829. The patients' pre-existing diseases and types of surgery were not reported. Eighteen of 29 patients who were considered at risk of pulmonary aspiration had been given preoperative medication to reduce gastric volume and increase gastric pH. The use of such treatment in the 24 patients who required intensive care support is not reported.[112]

Five further large studies concentrated on the general morbidity and mortality associated with anaesthesia. A French prospective survey of complications associated with anaesthesia in 198 103 anaesthetics performed between 1978 and 1982 showed 14 cases of pulmonary aspiration of gastric contents (1 in 14 150), two of which resulted in coma. No other morbidity or fatality was reported.[104] The only large recent British study reported six patients who

Table 1 Incidence (cases/anaesthetic), morbidity (cases/anaesthetic) and mortality (cases /anaesthetic) of pulmonary aspiration in the general surgical population (NR=not reported)

Country	Study size	Aspiration	Incidence	Morbidity	Mortality	Reference
Sweden	185 385	87	1/2131	1/3944	1/45 454	Ollson[82]
USA	215 488	67	1/3216	1/16 576	1/71 829	Warner[112]
France	198 103	14	1/14 150	1/99 052	Nil	Tiret[104]
UK	84 835	6	1/14 139	1/42 418	1/84 839	Leigh[59]
South Africa	240 483	1	NR	NR	1/240 483	Harrison[35]
Finland	338 934	5	NR	NR	1/67 786	Hovi-Viander[39]
Canada	112 721	101	1/1116	NR	NR	Cohen[16]

were admitted to an intensive care unit between 1984 and 1988 with suspected aspiration—a reported incidence of 1 in 14 139. Two of these patients developed acute respiratory distress syndrome (ARDS), of which one died (an 81-yr-old).[59]

A 10-yr review of deaths attributable to anaesthesia in South Africa found two deaths after vomiting, regurgitation and inhalation. One followed a Caesarean section and one patient had intestinal obstruction. A total of 240 483 cases were studied, giving a mortality rate of 1 in 240 483 in the general surgical population.[35] A Finnish study surveying all anaesthetics administered in 100 hospitals in 1975, reported five deaths from aspiration in 338 934 general anaesthetics resulting in a mortality rate of 1 in 67 786. Type of surgery, underlying disease process and degree of urgency were not reported.[39] A greater incidence of aspiration of 1 in 1116 patients was reported in a Canadian study. No morbidity or mortality values after pulmonary aspiration were given.[16] Another 10-yr review of 108 aspirations reported a 30% mortality rate in surgical and non-surgical patients but the incidence of aspiration was not known as the number of operations or admissions was not given, nor was the cause of death reported.[58]

A very large mortality of 62% in 47 patients after aspiration pneumonia was reported by Cameron, Mitchell and Zuidema.[13] Fifteen patients had received a general anaesthetic. These authors found that mortality increased from 41% if one lobe was involved, to more than 90% if more than one lobe was involved. A similar result has not been reported since.

The relationship between increasing use of the laryngeal mask airway and pulmonary aspiration was reported in a letter by Brimacombe and Berry.[9] A postal survey of 758 intensive care beds in Australia between 1990 and 1991 identified eight aspirations with one patient developing aspiration pneumonitis. However, there were no fatalities.

There are few studies of silent regurgitation during anaesthesia, which may reflect the difficulties in its detection. One study reported an overall rate of 7.8% of which 8.6% aspirated, despite the use of a cuffed tracheal tube. The rate was as high as 17.8% in upper abdominal operations. No morbidity or mortality was reported.[5]

In summary, the overall risk of detected aspiration during general anaesthesia appears to be small, with minimal morbidity and almost negligible mortality in the general surgical patient. Except for the study from the Mayo clinic,[112] none of the studies assessed the use and value of antacid medication.

Paediatric patients and neonates

The incidence of regurgitation during anaesthesia in children is unknown but it has been stated that aspiration pneumonitis is rarely associated with paediatric anaesthesia.[85] A French prospective survey between 1978 and 1982 of 40 240 paediatric general anaesthetics reported only four aspirations, two during maintenance and two in the recovery period. No morbidity or mortality was reported.[105]

However, silent pulmonary aspiration may be more frequent than in adults and was detected in a study using a barium mixture in 44 of 56 children of which 22 had no neurological or anatomical abnormalities.[79] Perioperative regurgitation, as determined by continuous oesophageal pH monitoring, occurred in three of 120 patients (2.5%) with no respiratory consequences.[73] Perioperative aspiration is rare, occurring in up to 1 in 1162 children, but is about three times more common than in adults.[82]

It has been suggested that a diagnosis of aspiration pneumonia in children should be made only if there are swallowing difficulties, known gastro-oesophageal reflux or a witnessed aspiration.[100]

Data on pulmonary aspiration and its associated morbidity and mortality are scarce in children and studies of the effects of prevention of aspiration are not available.

Obstetric patients

Mendelson[72] first described 66 cases of aspiration between 1932 and 1945, with an incidence of 1 in 667 parturients and two deaths, both caused by acute upper airway obstruction (mortality 1 in 22 008). This led to research into the causes and consequences of pulmonary aspiration.

Maternal deaths from pulmonary aspiration are recorded in the triennial *Confidential Enquiry Into Maternal Deaths in the UK*. There has been a steady decline from 18 deaths in 1964–66 to 11 deaths in 1976–78 to one death in 1991–93.[18 23 40] As the total number of anaesthetics administered is not known, the mortality rate for aspiration as a result of anaesthesia cannot be determined. Harrison noted one death in an obstetric patient from vomiting and aspiration during a 10-yr survey involving 240 483 patients, but did not report the total number of obstetric general anaesthetics.[35]

Table 2 Possible risk factors for regurgitation and pulmonary aspiration

Increased gastric content	Increased tendency to regurgitate	Laryngeal incompetence
Delayed gastric emptying	Decreased lower oesophageal sphincter tone	General anaesthesia
Gastric hypersecretion	Gastro-oesophageal reflux	Emergency surgery
		Inexperienced anaesthetist
		Night-time surgery
Overfeeding	Oesophageal strictures/carcinomas	Head injury
Lack of fasting	Zenker's diverticulum	Cerebral infarct/ haemorrhage
	Achalasia	Neuromuscular disorders
		Multiple sclerosis
	Extremes of age	Parkinson's disease
		Guillain–Barre
	?Diabetic autonomic neuropathy	Muscular dystrophies
		Cerebral palsy
		Cranial neuropathies
		Trauma, burns

The incidence of non-fatal aspiration in obstetric patients is said to be 1 in 6000 with light general anaesthetic for vaginal deliveries resulting in only mild aspiration pneumonitis, but 1 in 430 for Caesarean section.[54] An Italian study in 1991 reported an aspiration risk of 1 in 1547 for Caesarean section under general anaesthesia. Only one of eight patients had to undergo ventilation for 24 h.[24] Another study of an almost identical population gave a similar result, with a risk of pulmonary aspiration of 1 in 1431.[56] A high incidence of aspiration of 1 in 900 during Caesarean section and 1 in 9200 parturients, with no fatalities, were reported recently, which compares favourably with Mendelson's original values.[97] However, these values are at least twice those of the general surgical population.

Physiological changes occurring in pregnancy and immediately post-partum are important factors that increase the risk of pulmonary aspiration and also alter morbidity and mortality.[2 38 64 88] It is not known if the change in anaesthetic practice with rapid induction of anaesthesia and cricoid pressure, or if therapy to reduce gastric volume and increase gastric pH, have contributed to the reduced mortality since its first description in the obstetric patient. In otherwise healthy women in the childbearing age group, short-term morbidity is high but overall prognosis is excellent and long-term effects on pulmonary function are minimal.[97]

Prevention

Identification of predisposing factors for pulmonary aspiration is paramount in its prevention. Risk factors include: (i) increased gastric pressure, (ii) increased tendency to regurgitate and (iii) laryngeal incompetence. Table 2 summarizes conditions which may predispose to aspiration.[34 44 65 82 85 112]

The value of gastric volume and pH in the prediction of gastro-oesophageal reflux is questionable.[33] There appears to be no correlation between body mass index, smoking, duration of fasting, alcohol consumption and either gastric fluid volume or pH. However, it is known that prolonged fasting results in increased gastric pH.[42 65]

Fasting

Many studies have attempted to identify patients at risk with various durations of fasting in various settings, and these have been reviewed by Kallar and Everett.[47] Clear fluids given up to 2 h before elective surgery does not adversely affect gastric contents in healthy patients, with no difference between hospital inpatients and outpatients. There are no large-scale follow-up studies to show that liquid intake has *no* effect on the incidence and outcome of pulmonary aspiration. The authors recommended no relaxation of fasting guidelines for solids (6 h) but these recommendations are based largely on critical values of gastric volume and pH, which have not been proved in humans.

Recommendations for fasting in paediatric anaesthesia are 6 h for solids and 2 h for clear fluids. Children requiring emergency surgery should avoid all food and drink for at least 6 h if possible, with non-urgent cases being delayed overnight.[85] However, fasting guidelines in children may be difficult to implement.[113] Gastric volume and pH are apparently not influenced by repeated general anaesthetics, anxiety, premedication or overnight stay.[20]

There is no consensus regarding fasting during labour. The need to identify women with a low and high risk of emergency Caesarean section has recently been suggested.[96]

Pharmacotherapy

Surveys on the rate of use of antacid prophylaxis are rare. Recently, Kluger and Willemsen[51] reported a national survey of prophylaxis against aspiration in New Zealand. At least one treatment was used in 91% of elective non-obstetric and 85% of acute non-obstetric surgery compared with 98% in both elective and emergency obstetrics. Other existing surveys on the rate of antacid prophylaxis limit themselves to obstetric anaesthesia. British surveys found some form of chemoprophylaxis in 99% and in almost 100% of all emergency Caesarean sections.[102 108] In Australian practice, a value of 89% for both elective and emergency Caesarean section was reported.[12] Mechanical emptying of the stomach

in 44% of emergency Caesarean section patients reduced antacid prophylaxis to 60% in Norway.[98]

There are no data to show improved outcome after the use of antacids, H_2 receptor blockers, proton pump inhibitors or prokinetics.[109] Most studies suggest improved safety from reduced gastric volume and/or increases in gastric pH.[7 10 17 25 36 45 50 55 60 63 66 69 70 74 78 80 81 83 90 95 99 101 106 114–116 120] As there is no evidence to indicate the value of antacids or other means of increasing gastric pH, or of prokinetic therapy, a formal cost:benefit calculation is not possible. However, using the published values of mortality of approximately 1 in 100 000 (it should be remembered that the use of prophylactic measures may not influence this mortality) the use of any treatment would have to be extremely inexpensive and be associated with no side effects. Although rare, prophylactic measures can cause unwanted effects. Histamine receptor antagonists are associated with sinus bradycardia and atrioventricular block, hepatotoxicity and neuropsychiatric complications.[1 110] The effects of histamine receptor antagonists on immune function have received attention recently.[89] Proton pump inhibitors are mainly metabolized in the liver by the cytochrome P450 system and can delay elimination of diazepam, phenytoin and warfarin. Hepatoxicity, candidiasis, leucopenia and dry mouth have also been reported.[1]

There are no studies of the effects of bile aspiration and positive pressure ventilation. The logic of using drugs to alter gastric volume and pH in order to prevent gastric acid aspiration is based on data obtained in rhesus monkeys extrapolated to humans and there is no direct evidence to support the present use of antacid prophylaxis. Such treatment may indeed be harmful. Perhaps the anaesthetists' worry about this event has led to unnecessary use of drugs to prevent pulmonary aspiration of gastric contents in the non-obstetric population.

Anaesthetic techniques

For patients at risk of pulmonary aspiration, rapid sequence induction with application of cricoid pressure, as described by Sellick,[94] is now the norm and is used commonly in combination with antacid prophylaxis.[51] However, aspiration may still occur, as shown in the report of *Confidential Enquiry Into Perioperative Deaths*. Moreover, the ability to maintain adequate cricoid pressure for the necessary length of time and the accuracy of delivery of cricoid pressure are doubtful. There are no prospective clinical studies of the efficacy of this manoeuvre.

The laryngeal mask airway has changed anaesthetic practice, but it does not protect against pulmonary aspiration of gastric contents.[32 84] It is recommended that the laryngeal mask airway is not used in morbidly obese patients, and measures should be taken to ensure that the stomach is empty.[8]

Treatment

If aspiration has occurred in the anaesthetized patient, general measures such as 30° head-down tilt, suctioning of all material from the oropharynx, tracheal intubation and tracheal suctioning before mechanical ventilation or spontaneous breathing using 100% oxygen are advised.

High-dose steroids have been given systemically or by nebulizer in an attempt to diminish the inflammatory response but are of no proven benefit.[6 26 27 41 57 119] In the critically ill, however, the use of steroids has an adverse effect on mortality.[6 57]

There are no controlled studies examining empirical antibiotic therapy in the treatment of pulmonary aspiration. In clinical practice, however, antibiotics are often used in the treatment of aspiration pneumonia before isolation of pathogens. When antibiotics have to be given empirically, the general medical condition of the patient must be considered. In hospital, aspiration may be treated with a beta-lactamase resistant penicillin, a cephalosporin or clindamycin in conjunction with an aminoglycoside.[61 92 103] A low hospital mortality in severe aspiration pneumonia can occur without the immediate use of antibiotics.[37]

Patients who remain asymptomatic for 2 h are most unlikely to have respiratory sequelae.[112]

Conclusion

The incidence of pulmonary aspiration in general surgical patients is small, and only slightly greater in obstetric and paediatric patients. The resulting morbidity per anaesthetic is low and mortality very small. Changes in anaesthetic practice and training have probably contributed to this. In the general surgical patient, the true incidence of aspiration during anaesthesia remains unclear and the exact association with increased postoperative morbidity and mortality is not evident for both silent and witnessed aspiration. The published reported incidence of observed aspiration during anaesthesia is very small and has a very low mortality. Apart from a concern about aspiration of particulate matter causing hypoxia and death in the general surgical patient, and more so in the obstetric patient, are anaesthetists right in their fear of aspiration? The frequent use of pH increasing drugs or gastric volume reducers for premedication is of no proven benefit, may be harmful and is at present unwarranted.

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