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Perioperative aspiration: challenges and management options

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The preoperative evaluation and preparation of patients is important. It is prudent to confirm the patient has followed the fasting guidelines. Aspiration is linked to increased perioperative morbidity and mortality and an increased volume, acidity or particulate matter correlates with a higher risk of adverse outcomes.

The normal physiological protective mechanisms to prevent aspiration include the gastro-oesophageal junction, protective airway reflexes and upper oesophageal sphincter.

Robinson and Davidson suggest this strategy to reduce risk factors:

- Reduce gastric volume
- Avoid GA and sedation
- Reduce pH of gastric content
- Protect the airway
- Prevent regurgitation
- Careful extubation

Fasting guidelines are institution-specific. Aspiration and prevention of aspiration continue to create a challenging environment in the perioperative management of patients. Careful planning and management strategies must be in place before starting any case.

Keywords: perioperative, aspiration, challenges, management

Introduction

The preoperative evaluation and preparation of a patient is an important first step for the anaesthesiologist. It is an opportunity not only to conduct a thorough survey of medical records and do a physical exam, but to discuss any pertinent issues of the procedure with the patient. There should be a discussion about the requirements for fasting, and in the theatre reception, it is prudent to confirm the patient has followed the guidelines and is fasted.[1]

Our preoccupation with fasting stems from our fear of the elephant in the room – perioperative aspiration. Aspiration is linked to increased perioperative morbidity and mortality and an increased volume, acidity or particulate matter correlates with a higher risk of adverse outcomes.[2] The medications we use during this period cause absent or reduced airway reflexes and aspiration can occur during any type of anaesthetic.[2]

Incidence

Reviews suggest that the rate of aspiration during anaesthetic ranges from one in 300–7 100.[3,4] Significant morbidity occurs in one in 7 200 of these patients and one in 72 000–100 000 patients die subsequent to aspiration.[3,4] Most aspiration events occur during induction of anaesthesia, but aspiration can also occur during extubation and even intraoperatively.[5,6]

The UK: Fourth National Audit Project (NAP 4) revealed that in National Health Services Hospitals in the UK, aspiration was responsible for 50% of deaths and was the most common cause of death related to airway complications.[7]

In Australia, 30% of aspiration cases were admitted to a high dependency unit setting and of those, 4% demised according to the Australian Anaesthetic Incident Monitoring Study.[6]

In the US, the Anesthesia Closed Claims Project suggested that from 2000–2013, 57% of aspiration cases resulted in death and 14% in severe and permanent injury.[5]

Given the above statistics, it is of interest to the anaesthetist to know what to expect regarding the nature of gastric contents and gastric emptying. There are many methods used to evaluate gastric emptying or volume namely gastric aspirates, radiological studies, gastric ultrasound, and paracetamol absorption. Unfortunately, despite numerous studies using these techniques, gastric emptying time and gastric volume in fasted patients differ vastly from patient to patient.[8,9]

Normal physiology to prevent aspiration

The oesophagus extends from C6 vertebra to T11.[10] It is innervated by the autonomic nervous system.[10] Motility, blood flow and production of secretions is controlled by the enteric nervous system.[10] The parasympathetic system has an excitatory role on GI tract motility while the sympathetic system is inhibitory.[10] The upper third consists of striated muscle while the distal portion is smooth muscle.[10] The oesophagus has two distinct areas of high pressure: the upper (at the level of the cricoid cartilage) and lower oesophageal sphincters.[10]

The normal physiological protective mechanisms to prevent aspiration include the gastro-oesophageal junction, protective airway reflexes and upper oesophageal sphincter.[11]

Coughing, expiration and laryngospasm are upper airway reflexes (glottic closure reflexes)[10] that are triggered by sensory receptors in glottic and subglottic mucosa, which act to protect a patient from aspiration – these reflexes are diminished in the elderly and therefore they are more likely to aspirate perioperatively.[12]

The lungs are protected from toxic substances by a vagal reflex that causes constriction of bronchial smooth muscle and leads to bronchospasm.[10]

The upper oesophageal sphincter is formed by the cricopharyngeus, thyropharyngeus (circular oesophageal muscle) and inferior constrictor muscle of the pharynx.[10] Resting tone is 30–200 mmHg.[10] It prevents reflux of oesophageal contents into the pharynx in conscious patients.[11] The upper oesophageal sphincter opens and closes based on the pressure from the pharynx as it pushes food down.[10]

The gastro-oesophageal junction is an acute angle that exists between the last part of the oesophagus and the beginning of the stomach.[11] It helps to protect the oesophagus from acid reflux from the stomach in conjunction with the lower oesophageal sphincter.[11] The lower oesophageal sphincter is formed by circular oesophageal muscle and the diaphragm. Its resting tone is 10–45 mmHg.[10] Together they create a barrier pressure – a pressure higher than that of the stomach that prevents reflux.[11]

Most people aspirate small amounts of their oropharyngeal secretions[13] and at least 50% of healthy adults aspirate in their sleep.[14,15] One can conclude from this that most incidents of aspiration go unnoticed and resolve without intervention.[16]

What makes aspiration significant?

There are specific factors that are considered when looking at the severity of aspiration, namely gastric volume, the pH of the gastric content, and the particulate matter in the aspirate.[16]

A gastric volume of 2.5 ml/kg (25–40 ml) has been deemed to be significant. Studies using gastric ultrasound suggest a gastric volume of less than 1.5 ml/kg is considered an empty stomach but there is no conclusive evidence about the specific volume that decreases or increases the risk of aspiration.[2]

Highly acidic fluid is more damaging than pH-neutral fluid and a pH of 2.5 is frequently quoted as the acidity at which the most damage can occur if a patient has aspirated.[2]

Particulate aspirates can be particularly dangerous as they can cause both mechanical obstruction and inflammatory damage.[2]

On the background of this information, it is probably more accurate to say that the clinical presentation of aspiration depends not only on the above factors but also on the body's defence mechanisms.[16] Risk factors for aspiration are extensive:[11]

- Not fasting adequately
- Emergency surgery or recent trauma
- Mechanical problems such as intestinal obstruction, disruption of glottic closure or lower oesophageal sphincter due to tracheostomy, endotracheal intubation, head and neck cancer, bronchoscopy, nasogastric tube feeding
- Diseases such as diabetes or chronic kidney disease
- Raised intracranial pressure
- Pregnancy
- Morbid obesity
- Previous upper gastrointestinal (GI) surgery
- Positioning – head down or lithotomy
- Cholecystectomy and laparoscopic procedures
- Presence of supraglottic airway
- Positive pressure ventilation
- Light anaesthesia
- Increasing age
- Reduced consciousness
- Difficult airway
- Prolonged surgery > 2 hours
- Dysphagia due to neurological deficits (bulbar palsy) – cerebral palsy, Parkinson's disease
- Disorders of the upper GI tract such as oesophageal disease

The anaesthetic drugs we use can increase the risk of aspiration due to the attenuation of lower oesophageal sphincter tone.[17] This includes:

- Propofol
- Volatile anaesthetic agents
- β agonists
- Opioids
- Atropine
- Thiopental
- Tricyclics
- Glycopyrrolate[17]

Prevention of aspiration

Reduction of risk factors

Robinson and Davidson[11] suggest this strategy to reduce risk factors for aspiration:

- **Reduce gastric volume** with perioperative fasting, aspiration of stomach contents prior to starting case with nasogastric tube, and using prokinetics (metoclopramide 10 mg 6 hourly, erythromycin 100 mg 6 hourly or 250 mg 12 hourly).
- **Avoid general anaesthetic (GA) and sedation** where possible and use regional techniques.
- **Reduce pH of gastric content** with antacids (sodium citrate or magnesium trisilicate), H_2 histamine antagonists (cimetidine 400 mg 60–90 minutes before the procedure or ranitidine 150 mg 2 hours prior to induction, and ideally, 150 mg the night before) and proton pump inhibitors (omeprazole 20 mg, lansoprazole).

Note that these drugs have been proven to reduce gastric volume and acidity and their ability to prevent aspiration or emesis or reflux has not been evaluated.[1]

Anticholinergics are not recommended.[1]

- **Protect the airway** with tracheal intubation and second-generation supraglottic devices (ProSeal, Supreme, i-gel) which create a better seal and have a port for suctioning gastric content.
- **Prevent regurgitation** by using cricoid pressure and performing rapid sequence induction.
- **Careful extubation** when the patient is awake and airway reflexes have returned. Be careful of position – (whether head down, lateral or upright).

The prevention of aspiration with drugs is not routinely performed and is reserved for patient populations with increased risk of aspiration (as referenced above), i.e. patients with full stomach, symptomatic gastro-oesophageal reflux disease, hiatus hernia, presence of nasogastric tube, morbid obesity, diabetic gastroparesis and pregnancy.

Fasting guidelines

Clear fluids: this includes water, juices without pulp and coffee and tea without milk or cream as well as carbohydrate drinks. It takes about 12 minutes for the stomach to empty itself of 50% of water, and glucose-containing liquids take 90 minutes to clear.[18] Studies have shown that patients that are fasted overnight have similar gastric volumes to patients that have had clear fluids up to two hours before surgery.[19,20]

All other liquids are treated as solids – except for human milk. Milk is treated as solid because it curdles and becomes particulate matter in the stomach.[2] It contains protein and fat with increased gastric emptying time.[2] There have been no studies done on plant-based milk; they are considered the same as animal milk.[2] The need for extended fasting after drinking coffee and tea with milk is controversial but remains in place. One study has shown similar gastric emptying in patients who had 250 ml of tea with 50 ml of milk compared to patients who had 300 ml of tea. Both cleared the tea within 90 minutes.[21]

Solid food takes longer to empty from the stomach depending on the nutritional content. Gastric emptying begins after about one hour and 50% of gastric content passes into the duodenum in two hours.[22] Gastric emptying is slower if food has a higher caloric density, higher fat content and it has been shown that gastric emptying is slower in women and older patients.[2]

Chewing gum increases saliva production and stimulates gastric secretions but has no real effect on gastric volume or acidity – unless of course it is swallowed; then it becomes a solid object and is subject to solid fasting guidelines.[2]

Alcohol is not considered a clear fluid because of its ability to delay gastric emptying and decrease small intestine motility.[23]

Counselling of patients

Guidelines are institution-specific. The current guidelines from *UpToDate* highlighted below seem reasonable:[2,24]

Adults

- Clear fluids up to two hours before surgery.
- Light meal (solid foods and nonhuman milk): six hours.
- Chewing gum: stop chewing two hours prior to surgery but do not delay surgery unless swallowed.
- Fatty meal eight hours.
- Medications: can be taken up to two hours before surgery with clear liquids.

Children

- Clear fluids: up to one hour before surgery (increasing number of studies agree with this) but look at volume.
- Solids including sweets and chewing gum: six hours.
- Infants under six months:
  - Breast milk – three hours.
  - Formula – four hours.
- Infants over six months:
  - Breast milk – four hours.
  - Formula or cow's milk – six hours. The amount of food and the content of the food should be taken into consideration.

Special groups

The groups below deserve a special mention as they always pose difficulty in decision making – to delay or not to delay? Szabo and Rosenbaum suggest:[2]

- Obese – follow standard fasting guidelines.
- Pregnant – follow normal fasting guidelines – labouring patients have delayed gastric emptying so beware the emergency caesarean section after failed labour.
- Diabetes: patients with gastroparesis – ask about symptoms of autonomic dysfunction (30–60% incidence), they may require longer fasting times.
- Enteral tube feeds – the feeds are packed with carbohydrates, protein and fat and are considered a fatty meal:
  - Not intubated – stop NGT feeds eight hours before surgery.

- Intubated or tracheostomy (controversial) – is fasting necessary? There is a possibility of aspiration or microaspiration around the cuffed tube. A multidisciplinary discussion is necessary – take it case by case.
- Postpyloric feeding tube – can continue without a fast.
- Abdominal surgery in patient on enteral feeds – fast for eight hours.

Once patients have been identified whose risk for aspiration is higher than the average person, it is the job of the anaesthetist to mitigate this risk by performing awake tracheal intubation or rapid sequence induction with or without the use of cricoid pressure.

Rapid sequence induction was first described by Brian Sellick in 1961. The aim of the rapid sequence induction is to protect the airway of a patient and prevent aspiration by inducing and intubating in the shortest time possible. Cricoid pressure and the decision to use it or not remains controversial as the argument is that there are not enough studies to support its use and done incorrectly, it does more to harm or disrupt intubation than to protect the airway.[25] It seems the decision to use it depends on the region, or the institution and sometimes depends on the attending anaesthetist.[25] Most anaesthetists remain unhappy with this statement which emphasises the need for more robust studies to help determine if a place still exists for this manoeuvre in the anaesthetist's armamentarium.

Pulmonary aspiration

Curtis Mendelson described aspiration pneumonitis in 1946 after observing the phenomenon in pregnant women who had aspirated after anaesthesia.[12] He called it Mendelson syndrome.

Pulmonary aspiration can lead to pneumonitis or inflammation. It presents as either a chemical or bacterial aspiration pneumonia or mechanical obstruction.[16]

Chemical pneumonitis

Patients present with sudden onset dyspnoea, cough, hypoxaemia and tachycardia with a background history of aspiration.[16] They may have a low-grade fever and diffuse wheezes or crepitations on auscultation of the lungs.[16] Chest X-ray will show opacities on the dependent areas of the lung (this will depend on the position of the patient when they aspirated) and sometimes they present with diffuse opacities.[16]

Management

The patient's head should be turned to the side, and the mouth and the throat must be suctioned. If an endotracheal tube is in place, tracheal suctioning should also be performed.[16] Bronchoalveolar lavage can be done if there are people with the skills to do it, but it does not prevent chemical pneumonitis.[16]

Supportive care must be implemented: admission to a high dependency unit, support oxygenation, ventilation where necessary, etc.[16]

Antibiotics – when to start is controversial. They are frequently started at the time of aspiration[26] and approximately 10% of patients that have aspirated go on to have superinfections during the course of their illness.[27] Antibiotics can be commenced in patients who show continued or worsening respiratory distress after aspiration and they must be monitored for clinical improvement of symptoms.[16] Systemic glucocorticoids are not recommended.[28]

Bacterial pneumonitis

Bacteria that live in the upper airway or stomach, primarily streptococci and anaerobic organisms, colonise the lungs after aspiration.[16]

Presenting features depend on when during the course of disease the patient presents; onset is usually slow and characterised by fever, cough, purulent sputum (foul smelling) and dyspnoea.[16]

Management

- Antibiotics
- Community-acquired: augmentin 1 g 12 hourly.[16]
- Community-acquired requiring hospitalisation: ampicillin 1.5–3 g 6 hourly.[16]
- Severely ill patients: imipenem, meropenem, piperacillin-tazobactam.[16]

Mechanical obstruction

Aspiration of materials that cause obstruction or reflex closure of the airway.

Fluids: saline, barium, gastric contents, and water.

Solids: peanuts, vegetable particles, inorganic material, and teeth.

Clinical presentation depends on the level of the obstruction and includes cough, possibly wheezing with lung collapse or obstructive emphysema on chest X-ray.[16]

Management

Removal with flexible or rigid bronchoscopy.[16]

Conclusion

Aspiration and prevention of aspiration continue to create a challenging environment in the perioperative management of patients. Guidelines in place may help, but one must always be cognisant of the factors outside our control that can lead to aspiration. Careful planning and management strategies must be in place before starting any case.

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