Ways to prevent and treat pulmonary aspiration of gastric contents
Christian C. Apfel and Norbert Roewer

Purpose of review
Aspiration of gastric contents is a potentially life-threatening complication that has attracted much attention among anaesthesiologists. This review critically evaluates current knowledge and means by which aspiration can be prevented, and offers alternative management approaches.

Recent findings
Because of the low incidence of aspiration of gastric contents (approximately 1 aspiration/3000 general anaesthetic cases), gastric volume and pH have been used as surrogate end-points in many aspiration studies; however, the clinical relevance of these end-points is questionable. A few epidemiological studies have identified the impact of several risk factors. More importantly, they indicate that none of the generally accepted strategies can prevent aspiration in every patient. Because preventive strategies may not only be ineffective but may also exert additional risk (e.g. cricoid pressure, rapid sequence induction), the risk–benefit ratio for each technique must be reconsidered. We suggest a strategy in which these techniques can be used, but taking into consideration the individual’s risk for pulmonary aspiration.

Summary
None of the currently accepted methods can completely prevent the occurrence of pulmonary aspiration. Because they may themselves carry risk, the risk–benefit ratio should be considered, perhaps in a ranked order. New approaches are promising but randomized controlled trials are needed to validate their effectiveness.

Keywords
aspiration of gastric content, aspiration pneumonitis, cricoid pressure, Sellick manoeuvre

Introduction
Complications associated with anaesthesia were described early after the introduction of ether and chloroform in the middle of the 19th century. One such complication was ‘postnarcotic’ nausea and vomiting, the latter being described to occur more frequently if the patient had eaten recently [1]. Vomiting can lead to serious complications when protective reflexes are insufficient or absent, not only in the early postoperative period but also during the induction of anaesthesia. It is therefore not surprising that the first aspiration with lethal outcome was described at the same time [2]. Consequently, many studies have been reported, sometimes with disputable relevance, in order to gain an understanding of and to avoid this serious complication [3,4]. The present review seeks to clarify our knowledge and to elucidate its limitations in preventing and treating pulmonary aspiration of gastric contents.

Pathophysiology and risk factors
Numerous pathophysiological considerations and risk factors have been described, but their clinical impact must be judged with caution.

Physiology of the gastric contents
The gastric content (volume, pH, particles) results from the balance between gastric input (food, saliva and gastric secretion of about 1 ml/kg per h) and output (into the duodenum). In contrast to the impressively short clearance time for clear liquids of approximately 12 min, the time for non-clear liquids and especially solid particles to pass through the stomach is considerably longer [5] and is subject to variation among individuals. Thus, even after a well-defined period of fasting, there is no absolute certainty that the stomach is empty.

Reflux or vomiting of gastric contents
Usually, the lower oesophageal sphincter prevents the gastric contents from being regurgitated into the pharynx. According to our current understanding, regurgitation occurs if the barrier pressure [i.e. the difference between the pressure of the oesophageal sphincter (about 25 mmHg) and the intragastric pressure (about 5–10 mmHg)] becomes negative. This may occur when emptying of the stomach is disrupted (e.g. pylorus stenosis, obstructive ileus). So, when there is also an incompetence of the oesophageal sphincter, the increase in gastric volume
may lead to a negative barrier pressure so that regurgitation occurs.

In contrast to regurgitation, vomiting is caused by a reflex that causes contractions of the abdominal wall (and, to a lesser extent, of the stomach) and leads to very high intragastric pressures (over 100 mmHg is possible). Usually, vomiting is associated with an active opening of the oesophageal sphincter, whereas it remains closed in the case of retching so that no gastric content is expelled. However, if the intragastric volume is large enough, the resulting high pressure may easily be greater than the oesophageal pressure, so that gastric contents will be vomited anyway.

**Aspiration**

By definition, aspiration is the process of the inhalation or suction of any material. Because pulmonary aspiration of gastric contents is the most common complication in the fields of anaesthesia and intensive care medicine, aspiration of other materials such as meconium, foreign bodies and water is not considered here. In an awake person with normal anatomy, neither regurgitation nor vomiting leads to aspiration of gastric contents because of the presence of protective reflexes. However, abolition of the protective reflexes during general anaesthesia results in aspiration in 1 out of every 3000 patients [6].

Depending on the gastric contents, the type of aspirated material may induce a chemical inflammation, leading to pneumonia (e.g. acid gastric juice); it may induce bacterial inflammation, leading to pneumonia (e.g. bowel material due to ileus); or it may obstruct the airways, leading to atelectases (e.g. particles). However, in most cases the clinical picture is mixed and associated with different radiological signs.

It has been shown experimentally that aspiration of acidic material destroys the pulmonary structures that activate alveolar macrophages and liberate proinflammatory cytokines such as tumour necrosis factor and interleukin-8 [4]. Because this injury occurs in the first 20–40 s, the concept of bronchial lavage to ‘dilute’ the aspirate with saline is no longer in favour. Afterwards, neutrophilic granulocytes are activated so that thromboxane, oxygen radicals, leucotrienes and complement factors are liberated. Liberation of cytokines is thought to affect lung areas that are not primarily involved and to cause oedema in other organs such as the heart and kidneys. Experimental data suggest that the pulmonary injury may be biphasic, with a second peak occurring after 4–6 h [7]. According to epidemiological data, patients who do not develop symptoms within the first 2 h have an uncomplicated outcome [6]. However, when monitoring early signs and symptoms, it should be borne in mind that the full clinical picture may still develop. These patients should thus be monitored for at least 2 h.

**Risk for aspiration**

Aspiration has a multifactorial origin. Because its development is not clearly foreseeable, it is important to be aware of certain risk factors. Many patients have risk factors, and so pathophysiological reasoning leads clinicians to assume an increased risk. However, prophylactic strategies to prevent aspiration also carry potential risks of side effects (e.g. rapid sequence induction with an unexpected difficult intubation), so that a critical appraisal of the risk factors is needed. Thus, Table 1 lists only those risk factors that we consider to be well founded.

**Gastric volume and pH**

In 1974, Roberts and Shirley [8] stated that a critical pulmonary injury must be considered if gastric volume is greater than 0.4 ml/kg and pH is less than 2.5. Although this definition was based on an unpublished observation in an ape, it was taken as a surrogate parameter in numerous subsequent studies. Ten years later, James et al. [9] conducted a systematic investigation of the impact of volume and pH in the rat and found that considerably higher volumes or lower pH were necessary to cause lethal acid pneumonitis (Table 2).

In an editorial, Schreiner [10] pointed out that if this surrogate parameter proposed by Robert and Shirley were

---

**Table 1. Selection of risk factors in which increased risk for aspiration should be considered**

<table>
<thead>
<tr>
<th>Mechanisms</th>
<th>Underlying disease or circumstance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impaired protection reflexes</td>
<td>Brain injury, cerebrovascular insult, intoxication (e.g. alcohol), neurological impairment</td>
</tr>
<tr>
<td>Anatomical or mechanical reasons</td>
<td>Obstructive bowel diseases such as pyloric stenosis, ileus, hiatus hernia, reflux oesophagitis</td>
</tr>
<tr>
<td>Further causes</td>
<td>Pregnancy (2nd trimester or later), emergency cases, difficult airway management, multi-morbid patient</td>
</tr>
</tbody>
</table>

ASA, American Society of Anesthesiologists.

---

**Table 2. Mortality rates (%) in rats after aspiration of solutions of various pH and volume**

<table>
<thead>
<tr>
<th>Volume (ml/kg)</th>
<th>pH</th>
<th>1.0</th>
<th>1.4</th>
<th>1.8</th>
<th>2.5</th>
<th>3.5</th>
<th>5.8</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.2</td>
<td>20</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>0.3</td>
<td>90</td>
<td>0</td>
<td>9</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>0.4</td>
<td>90</td>
<td>40</td>
<td>9</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>1.0</td>
<td>100</td>
<td>90</td>
<td>20</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2.0</td>
<td>100</td>
<td>100</td>
<td>27</td>
<td>30</td>
<td>20</td>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>4.0</td>
<td>100</td>
<td>100</td>
<td>38</td>
<td>20</td>
<td>40</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>6.0</td>
<td>100</td>
<td>100</td>
<td>60</td>
<td>20</td>
<td>30</td>
<td>30</td>
<td>30</td>
</tr>
</tbody>
</table>

Reproduced with permission from James et al. [9].
to be applied, then about 30–60% of patients undergoing surgery would be classified as ‘at risk’ for pulmonary aspiration. Because this clearly contrasts with the average rate (about 1 in 3000), ‘It is time to put an end to using the criteria of Roberts and Shirley, a surrogate measure with no proven clinical basis, and to put an end to discussions of risk based on studies whose designs fail to meet basic criteria for adequate evidence’. [10].

Anatomical and mechanical factors
As mentioned in Table 1, a typical example of an increased risk for pulmonary aspiration is an intestinal obstructive disease such as pyloric stenosis or obstructive ileus. Also, a history of oesophageal reflux – although no strong data exist – appears to confer increased risk.

Pregnancy
During the physiological exception of pregnancy, numerous changes occur. First, elevated serum levels of gastrin and decreased levels of motilin may lower the pH while increasing the residual volume. Secondly, pregnancy is frequently associated with a reflux oesophagitis (it is assumed that an apparently insufficient lower oesophageal sphincter function is caused by a disturbed neuromuscular transmission). Thirdly, elevated intra-abdominal pressure may also lead to increased intragastric pressure; however, whether and to what extent elevated residual volume increases intragastric pressure remains questionable [11]. Nevertheless, data from large epidemiological studies confirm that the risk for aspiration is increased during pregnancy [6].

Reduced or absent protective reflexes
Aspiration-associated pneumonia is the leading cause of death in patients with neurological dysphagia, which accounts for about 450,000 patients in the USA, and is the cause of almost 20% of the deaths that occur in nursing homes [12]. This illustrates the importance of loss of protective reflexes (i.e. laryngeal incompetence) in increasing the risk for pulmonary aspiration. Applying this information to anaesthesia, intensive care and emergency medicine means that all conditions that affect protective reflexes, such as brain trauma, brain insult, seizures, drug intoxication and, especially, induction of and recovery from general anaesthesia, represent a threat for pulmonary aspiration of gastric contents. This may also be true for senility, provided that laryngeal protective reflexes are impaired, although no strong data are available to support this. Epidemiological studies, which describe a correlation between the age and the American Society of Anesthesiologists (ASA) physical classification, are in favour of this hypothesis.

Prophylaxis
It is generally accepted that the risk for pulmonary aspiration is considerably lower for regional than for general anaesthesia, because regional anaesthesia should not impair the protective reflexes. Apart from the risk for unexpected difficult airway management, aspiration is the main reason why maternal morbidity and mortality with caesarean section are considerably lower after regional than after general anaesthesia. However, this may only be true without the use of other concomitant medication given for sedation, which may itself impair the protective reflexes.

Intubation and laryngeal mask
Without doubt, intubation is considered the ‘gold standard’ for prevention of aspiration in anaesthetized patients, although aspiration may still occur. In contrast, a conventional laryngeal mask does not provide sufficient protection against aspiration. However, because the intubation itself, and not the laryngeal mask, may be associated with other complications such as dental or laryngeal damage, or may even provoke vomiting, the use of the latter should depend on the individual patient’s risk–benefit relationship. In this respect, a recently developed laryngeal mask named ProSeal® (The Laryngeal Mask Company Limited, St. Helier, Jersey, UK) appears promising. Apart from improvements that should enable better sealing of the respiratory tract, this device has an additional channel that leads to the oesophagus to allow for decompression and through which a gastric tube may be placed. In a study conducted in 10 cadavers [13], fluid instilled into the oesophagus did not reach the airway when the oesophageal lumen was left open. However, only large-scale, prospective, epidemiological studies will be able to demonstrate whether such an approach is equivalent to the current gold standard of tracheal intubation with a correctly blocked cuff.

Nil per os
Because of the variability between patients in the rate of gastric emptying, especially after intake of solid foods, there is no specific time that allows one to assume that the stomach is completely free of ingested food. Therefore, in patients undergoing elective surgery the recommended period of ‘nil per os’ is 6 h. However, numerous studies have demonstrated that clear fluid has a much faster emptying time (and breast milk passes faster than cow’s milk), and so the Task Force on Preoperative Fasting of the ASA has given the following recommendations (http://www.asahq.org/publicationsServices.htm): 2 h for clear fluids; 4 h for breast milk (in infants); and 6 h for a light meal, and for cow’s milk or baby food.

Drugs
The impact of antihistamines and antacids has been investigated in several studies. According to the ASA Task Force on Preoperative Fasting, these drugs are only justified when an increased risk for aspiration is present. Because of the long onset of antihistamines, they should
be started in the evening before anaesthesia (e.g. 300 mg ranitidine). The combination of antihistamines and metoclopramide immediately before the induction of anaesthesia is unlikely to lower gastric pH and residual volume. In contrast, antacids have an instantaneous and reliable onset of action, and can therefore also be administered just before induction of anaesthesia. Because particles may lead to severe pulmonary complications themselves when aspirated, only nonparticular antacids should be used, and we recommend sodium citrate (e.g. 30 ml of 0.3 mol sodium citrate), not only in obstetric anaesthesia but also in any patient in whom aspiration is a concern.

Nasogastric tube
In patients in whom gastric content is suspected, the use of a nasogastric tube is generally accepted. The primary idea is to release any potential pressure from it by draining air or fluids rather than empty the stomach, and it must be appreciated that a gastric tube does not ensure an empty stomach. Whether it can or should be left in place for induction is controversial, but there is some evidence that efficacy of cricoid pressure is not affected by the presence of a gastric tube [14].

Controlled cardia occlusion
For this reason the use of a nasogastric tube, which occludes the cardia with an inflatable balloon, appears to be the most logical approach [3,15]. Such a tube is inserted with a deflated balloon while the patient is asked to swallow. After suctioning the gastric contents, the balloon – which is located in the stomach – is blown up and the tube is gently pulled so that the balloon occludes the connection between the lower oesophageal sphincter and the cardia. Preliminary experimental and clinical studies suggest that such a device may safely prevent the reflux of gastric contents into the pharynx. This approach may therefore eliminate the need for rapid sequence induction (which has risks of its own) and may allow the use of a laryngeal mask [16].

Rapid sequence induction
During induction of general anaesthesia (i.e. when protective reflexes are lost until a cuffed tube is blocked in place), there is a potential risk for aspiration. In order to shorten this interval, a strategy termed ‘rapid sequence induction’ was developed. The main principle is that hypnotics (opioids) and muscle relaxants are given simultaneously (with cricoid pressure and without face mask ventilation to avoid gastric inflation) so that laryngoscopy and intubation can be performed within 60–90 s.

In clinical practice the following approach is widely accepted. A surgical suction device is switched on for direct access. The chest and upper part of the body are tilted 30–45° upward. Thorough denitrogenation is accomplished using 100% oxygen. A small dose of nondepolarizing muscle relaxant (about one-fifth of the dose needed for laryngoscopy) is administered and light cricoid pressure applied (10–15 N). Then, 500 mg thiopentone and 100 mg succinylcholine are administered intravenously, increasing the cricoid pressure to 35–40 N immediately after loss of conscience. Laryngoscopy is conducted after 60 s and the tube is inserted using an introducer. After the tube is blocked and in the correct place (confirmed by auscultation or capnography), the cricoid pressure is released, the airway will be secured and anaesthesia maintained.

For each of the above-mentioned manoeuvres, there is some controversy about their effectiveness. For example, the likelihood of reflux can be reduced by tilting the upper part of the body 30–45°, but this degree of tilt may promote aspiration of gastric contents if they are vomited. Thus, many anaesthesiologists prefer to perform induction of anaesthesia with a slight tilt downward, accepting a higher risk of reflux in return for prevention, or at least minimization, of significant aspiration into the lungs. Another issue is the use of succinylcholine or a nondepolarizing muscle relaxant. Succinylcholine has a slightly faster onset than vecuronium – a difference that may not be clinically relevant [17]. Succinylcholine might also be preferable if a ‘can’t intubate, can’t ventilate’ situation occurs, although clinically relevant desaturation may occur well before restitution of spontaneous ventilation [18]. However, succinylcholine may be contraindicated in traumatized or immobilized patients because it may cause cardiac arrest, trigger malignant hyperthermia, or lead to an allergic reaction, any of which can potentially carry significant costs for society [19]. Because a difficult airway occurs in about 1 out of every 10 000 anaesthesia patients and is about 10 times more common in obstetric anaesthesia, the indication for rapid sequence induction should be carefully balanced against other potential risks for pulmonary aspiration. It may thus be reasonable to use a safer approach such as fibreoptic intubation in an awake patient, or to use a balloon to occlude the cardia and proceed with a conventional induction in an unconscious patient rather than use rapid sequence induction. Also, if there is insufficient time for these options, it might still be safer to use a ‘modified’ rapid sequence induction. One possibility is to apply a gentle single breath with a face mask (with the adjustable pressure-limiting valve allowing no more than 25 cmH₂O) before administering the muscle relaxant to ensure that ventilation is possible if the patient cannot be intubated (even if succinylcholine is used) [18], but not to ventilate unless it is needed to avoid desaturation.

Cricoid pressure
In 1961, Sellick suggested that cricoid pressure should be applied in order to prevent regurgitation and subsequent aspiration of gastric contents [20]. Several studies
Evaluation of the usefulness of this procedure is complicated by the fact that cricoid pressure decreases the tone of the lower oesophageal sphincter and can provoke vomiting if it is applied in an awake patient. It should never exceed 20 N in awake patients. As summarised in a recent review [26], ‘There have been no studies proving that cricoid pressure is beneficial, yet there is evidence that it is often ineffective and that it may increase the risk of failed intubation and regurgitation’.

**Exeutubation**
Methods for preventing pulmonary aspiration of gastric contents usually concentrate on reducing gastric acidity and volume, or on induction of anaesthesia. However, there is still a considerable proportion of patients in whom aspiration occurs in conjunction with extubation and with postoperative nausea and vomiting [6,23]. This is not surprising because 20% of patients have impaired protection reflexes for up to 4 h after anaesthesia has ended. With the scarcity of studies on this problem, the current approach consists of using gastric decompression combined with extubation at a late stage when protective reflexes have returned as much as possible.

**Therapy**
Therapy for a witnessed aspiration of gastric contents is generally symptomatic and consists of tilting the operation table head down (Trendelenburg position) and suctioning the pharynx.

**Intubation**
The next urgent step is immediate intubation, followed by suctioning of the trachea and main bronchi as far as possible before reinstituting ventilation.

**Bronchoscopy**
If a fibreoptic bronchoscope is at hand then this should be used, provided the suction channel will not be obstructed with particulate material. Measuring the pH of the retrieved aspirate using Litmus paper is recommended. If aspiration of faeces is suspected, then a specimen should be retrieved for further microbiological testing or resistances.

**Further symptomatic treatment**
There are no studies to answer the question of how to manage a patient with pulmonary aspiration, and so the following recommendations are mainly based on common medical sense and expert opinion. The following methods should be dependent on the severity of the aspiration and its clinical course. In general, positive end-expiratory pressure ventilation is recommended, and arterial blood gases and chest radiography should be performed to estimate the degree of aspiration and its impairment of lung function. It should be kept in mind that the lung injury of acute acid aspiration may follow a biphasic pathogenesis, with a physiochemical injury initially and an inflammatory injury with a peak of about 2–3 h later [7]; patients should therefore be observed for at least 2 h after the event.

Usually, the amount of aspirate is difficult to estimate. However, if only a mild injury is suspected (e.g. if arterial oxygen tension is above 60 mmHg at an inspired oxygen concentration of 50%), then it may be justified to try to extubate the patient. If necessary, continuous positive airway-pressure assisted ventilation (up to 12–14 mmHg) may be justified. If the injury is severe then the patient needs to be kept intubated with an ideal positive end-expiratory pressure, and does therefore require intensive care treatment because this may lead to acute respiratory distress syndrome.

**Interventions of disputable benefits**
There have been attempts to dilute aspirated material with a bronchoalveolar lavage. This method is no longer used because particular material may be further pressed into the periphery. Likewise, an attempt to neutralize the acid aspirate with alkaline fluids is not indicated because it is now known that acid injury occurs within 60–90 s and the alkaline fluids may deteriorate the lung tissue as well.

Institution of empirical antibiotic prophylaxis is no longer favoured because it can lead to development of problematic micro-organisms with secondary resistances. According to Marik [12], most cases of aspiration that occur in conjunction with neurological disease in nursing homes are caused by acid-induced pneumonitis, and only 15% of these patients will develop a bacterial aspiration pneumonitis, and so routine antibiotic prophylaxis may not be justified. However, some authors still recommend treatment with an antibiotic that is effective against Gram-negative bacteria.
Because the inflammatory component peaks after 2–3 h, the use of steroids has been recommended since the late 1960s. However, although inflammation is diminished, no positive impact of this treatment on the course of the injury has been demonstrated; this approach is therefore no longer favoured [12].

**Conclusion**

Perioperative aspiration is a potentially life-threatening complication, with an incidence of about one in 3000 events. The majority of studies have used residual gastric volume and pH as surrogate measures – an approach with no proven clinical basis because such studies do not meet basic criteria for adequate evidence. We recommend adherence to the ASA guidelines on perioperative fasting (no clear fluids should be consumed within 2 h before an operation; 6 h for a light meal) to minimize the risk for aspiration, although interindividual variability in gastric emptying does not guarantee that the stomach is empty. In contrast to the standard approach of administering histamine antagonists with metoclopramide, sodium citrate appears to be most reliable in increasing pH shortly before induction of anaesthesia. Neither rapid sequence induction nor application of cricoid pressure can reliably prevent aspiration but may be associated with other complications. So, with the possible exception of ‘controlled cardia occlusion’, there is no technique that can absolutely prevent aspiration and resulting complications. Trendelenburg position and suctioning are the immediate interventions to prevent regurgitated gastric contents from aspiration if reflexes are impaired. Intubation with immediate tracheal suctioning are the next steps. Bronchoalveolar lavage, prophylactic antibiotics and dexamethasone have not been shown to be beneficial.

**References and recommended reading**

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest


