The functions of the stomach are:

- temporary storage of food input;
- mechanical breakdown of food into smaller particles;
- chemical digestion, with breakdown of proteins;
- regulation of output of chyme into the duodenum;
- secretion of intrinsic factor, vital for vitamin B₁₂ absorption.

Most anaesthetists are wary of this part of the gastrointestinal tract because of the potential for tracheobronchial aspiration of stomach contents. Nevertheless, we are required to administer anaesthesia to patients with full stomachs and an understanding of the relevant gastric physiology can help avoid and reduce the consequences of gastric aspiration.

For aspiration of stomach contents to occur there must first be gastro-oesophageal reflux, when gastric contents breach the lower oesophageal sphincter (LOS). The acid material must then flow back along the 30 cm oesophagus and be regurgitated (pass the upper oesophageal sphincter) to contaminate the pharynx. If the patient’s level of consciousness is reduced, the normally protective gag and cough reflexes will be obtunded and gastric contents may then reach the tracheobronchial tree (aspiration). Solid matter may obstruct the airways and the acidic liquid can cause bronchospasm, resulting in severe hypoxia and respiratory failure.

**Gastro-oesophageal reflux and the lower oesophageal sphincter**

The distal oesophagus passes through the crura of the diaphragm and joins the stomach at an acute angle, the gastro-oesophageal junction, which normally lies just below the diaphragmatic hiatus. The LOS is formed by the intrinsic circular smooth muscle of the lowest 2‒4 cm of the oesophagus, tonic contraction of which separates the gastric and oesophageal lumens. The LOS is a physiological sphincter which is normally closed with a resting pressure of 15‒25 mm Hg above gastric pressure (termed the barrier pressure). It relaxes on swallowing, a reflex mediated by the inhibitory neurotransmitters nitric oxide and vasoactive intestinal polypeptide. This allows saliva, ingested liquids, and food boluses, which are transported from the pharynx on the oesophageal peristaltic wave, to pass through into the stomach. The LOS then regains its tone.

LOS tone was thought to be the main defence mechanism against reflux of acidic gastric contents into the lower oesophagus. It was found that many patients with severe erosive oesophagitis had LOS pressures below 10 mm Hg. In these cases small increases in intra-gastric or intra-abdominal pressure resulted in free reflux of gastric acid into the oesophagus. Various factors are known to alter LOS tone and may affect the incidence of gastro-oesophageal reflux (Table 1). However, studies in normal individuals have since demonstrated that gastro-oesophageal reflux is a physiological event and healthy asymptomatic people have acid reflux sufficient to lower the pH in the distal oesophagus to below 4 for up to 4% of the time. It is now recognized that there are transient, unprovoked relaxations of the LOS which are of longer duration (10‒45 s) than those associated with swallowing (6‒8 s).¹ The frequency of these spontaneous relaxations correlates with the severity of oesophagitis, and explains how patients can have severe heartburn with a normal resting LOS tone. However, the severity of symptoms correlates poorly with the degree of acid reflux and the presence or extent of mucosal damage. Excessive gastro-oesophageal reflux also occurs in patients with disordered oesophageal motility and those with gastric motility disorder (GORD). In such cases, reflux is severe and present 24 h a day, producing symptoms such as heartburn and dysphagia.

**Measures to reduce the incidence and effects of aspiration of gastric contents**

(i) Minimize the amount of gastro-oesophageal reflux, by:
   (a) maintaining the tone of the LOS;
   (b) reducing the gastro-oesophageal pressure gradient;
   (c) using gravity.
(ii) Reduce gastric volume.
(iii) Increase gastric emptying.
(iv) Reduce acidity of gastric contents.

**Key points**

- Gastro-oesophageal reflux is a normal physiological event.
- The lower oesophageal sphincter (LOS) may relax spontaneously.
- The barrier pressure of the LOS is one of the defences against acid reflux.
- The head-up position discourages reflux and regurgitation.
- Antacids raise the pH of stomach contents but increase gastric volume.
Pathological
Succinylcholine
Thiopental
Cyclizine
Opioids

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proximal stomach (‘adaptive relaxation’).2 As the stomach filled the intra-gastric pressure would increase, the muscle layers relax as the stomach expands. It might be anticipated that as the stomach filled the intra-gastric pressure would increase, and so make reflux more likely.

Factors that increase LOS tone and so make reflux less likely
Factors that decrease LOS tone and so make reflux more likely

Physiological
Cholinergic stimulation
Swallowing
Oestrogen, progesterone

Pharmacological
Anti-cholinesterases, e.g. neostigmine
Anti-muscarinics, e.g. atropine, glycopyrrolate
Dopamine
Opioids
Thiopental
Alcohol

Gastro-oesophageal reflux and gravity
Both reflux and regurgitation are passive processes which are influenced by gravity and so are less likely to occur if the patient is semi-recumbent rather than supine. For mechanically ventilated patients the time spent supine is a critical factor associated with aspiration.3 Elevation of the head of the patient’s bed is included in the Institute for Healthcare Improvement’s ventilator care bundle for reducing the high mortality of ventilator-acquired pneumonia.4

Gastro-oesophageal pressure gradient
If intra-gastric or intra-abdominal pressure (which is transmitted to the stomach) is elevated there will be an increased pressure gradient across the LOS increasing the chance of gastro-oesophageal reflux occurring.

The stomach converts the episodic input of food from the oesophagus into a more continuous output of chyme into the duodenum. The fundus and body (the proximal stomach) act as a reservoir and their relatively thin, highly distensible smooth muscle layers relax as the stomach expands. It might be anticipated that as the stomach filled the intra-gastric pressure would increase, but there is little increase in intra-gastric pressure until gastric volume exceeds a litre. This accommodation results from a vagal reflex initiated by stretch receptors in the oesophagus (‘receptive relaxation’) and as a consequence of the presence of food in the proximal stomach (‘adaptive relaxation’).2

Intra-abdominal pressure is increased in various situations (including obesity, pregnancy, intestinal obstruction, and during laparoscopic surgery) and is associated with a higher incidence of gastro-oesophageal reflux. The patient with gastrointestinal obstruction develops distension of the gut proximal to the obstruction due to sequestration of fluid and gas. It may be possible to lessen this by inserting and intermittently aspirating a large-bore nasogastric tube which will help to decompress the gut and relieve the raised intra-abdominal pressure. The patient will be more comfortable if they are allowed to sit propped up with their hips and knees flexed as this reduces the tension in their abdominal wall muscles.

Gastric volume
The volume of the stomach is determined by:

- the amount of food and drink ingested;
- the volume of gastric juice produced;
- the rate at which the stomach empties.

Gastric volume is reduced by keeping a patient ‘nil by mouth’ not only because input is omitted but also because less gastric juice is secreted. Gastric juice is produced in response to eating. Normally the stomach produces about 2 litres a day but during fasting there is little or no secretion. It consists of hydrochloric acid, intrinsic factor, pepsinogens, and mucus (the secretions of the gastric gland cells, Table 2) and also contains water and electrolytes, particularly K+ and Cl−. It has a pH of 1–1.5.

Physical methods may be used to empty the stomach: a large-bore nasogastric tube may be inserted and aspirated occasionally but otherwise allowed to drain freely. Emetic agents are no longer used.

Gastric emptying
For the stomach to empty, the pressure generated by the antral pump must exceed the resistance of the pyloric sphincter. In general, emptying occurs at an exponential rate proportional to the volume of the stomach, that is, the fuller the stomach, the more rapidly it empties (Table 3). This is mediated by vagal excitatory reflexes (provoked by gastric distension). Gastrin is also released in response to antral
distension, and both these stimuli produce an increase in antral pump activity. The speed of emptying for liquids, or contents consisting of smaller particles, is faster than for solids. Thus, in the normal stomach, 95% of an ingested clear liquid reaches the duodenum within 1 h and 50% of a meal will pass the pylorus in 2 h.

The chemical composition of the chyme entering the duodenum also affects the rate of gastric emptying and various hormones are involved (Table 4). If the chyme is too acidic, secretin is released, which slows gastric emptying, reduces the production of gastric acid, and increases the secretion of alkaline pancreatic juice into the duodenum, and also reduces gastric emptying. If the chyme has too high a content of amino acids, gastrin is released. This increases contraction of the pyloric sphincter (as well as increasing gastric motility) and overall delays gastric emptying. Hypertonic chyme is detected by duodenal osmoreceptors and gastric emptying is slowed.

As the duodenum fills, stretch receptors are activated and inhibit the vagus which results in reduced gut tone and motility, temporarily reducing gastric emptying. As the duodenum empties this inhibition diminishes, the tone and motility of the gut increases, and gastric emptying is restored. The neural and hormonal mechanisms which originate from the duodenum and feedback to slow gastric emptying constitute the entero-gastric reflex. By regulating the rate of delivery of chyme into the duodenum, the absorption of nutrients in the small intestine is maximized.

Alcohol and opioids reduce the rate of gastric emptying, as do pain, fear, and anxiety (acting via the sympathetic nervous system which inhibits motility). It is now recognized that pregnancy per se does not alter gastric emptying. However, during labour there is delay because of pain, anxiety, and administration of opioids.

Prokinetic drugs may be used to stimulate enteral propulsion if there is no gastrointestinal obstruction, perforation, or haemorrhage. Metoclopramide is a centrally and peripherally acting dopaminergic D2 antagonist which increases the rate of gastric emptying and small intestinal transit, and also increases LOS constriction. It can be given i.v., i.m., or orally, in a dose of 10 mg 8 hourly, but is most effective i.v. Its use may be limited by adverse effects which include extra-pyramidal effects, tardive dyskinesia, and hyperprolactinaemia. Domperidone has similar pharmacological actions and is better tolerated but can only be given orally or rectally.

The macrolide antibiotic erythromycin has prokinetic properties and may produce abdominal cramps, nausea, and vomiting. It is an agonist at motilin receptors, stimulating strong antral contractions.

### Table 2: Secretions of gastric gland cells

<table>
<thead>
<tr>
<th>Released from</th>
<th>Release stimulated by</th>
<th>Release inhibited by</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pepsin</td>
<td>Gastrointestinal reflex</td>
<td>Acid pH</td>
<td>A proteolytic enzyme, stored as inactive pepsinogens, which facilitates protein digestion</td>
</tr>
<tr>
<td></td>
<td>ACh</td>
<td>Secretin (from the duodenum)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>H2 receptor antagonists PPIs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pathological</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 3: Factors affecting gastric emptying

<table>
<thead>
<tr>
<th>Factors that increase gastric emptying and so make reflux less likely</th>
<th>Factors that delay gastric emptying and so make reflux more likely</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiological</td>
<td>Pharmacological</td>
</tr>
<tr>
<td>Stomach distension</td>
<td>Anti-cholinesterases, e.g. neostigmine</td>
</tr>
<tr>
<td>Liquid content</td>
<td>Anti-muscarinics, e.g. atropine, glycopyrrolate</td>
</tr>
<tr>
<td>Smaller particles</td>
<td>Opioids</td>
</tr>
<tr>
<td>Parasympathetic stimulation</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anti-muscarinics, e.g. atropine, glycopyrrolate</td>
</tr>
<tr>
<td>Pathological</td>
<td>Alcohol</td>
</tr>
<tr>
<td>Erythromycin</td>
<td>Pyloric stenosis</td>
</tr>
<tr>
<td>Metoclopramide</td>
<td>Intestinal obstruction</td>
</tr>
<tr>
<td>Domperidone</td>
<td>Previous vagotomy</td>
</tr>
<tr>
<td>Intestinal obstruction</td>
<td></td>
</tr>
</tbody>
</table>

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Gastric contents

The morbidity of acid aspiration syndrome correlates with the volume, acidity, and the particulate nature of the material introduced into the lungs. Therefore, reducing the size of the particles and the acidity of the contents of the stomach will result in less severe pulmonary damage if aspiration occurs.

Parietal cell production of gastric acid is stimulated by various factors (Table 2). Inhibition of gastric acid secretion will also reduce the volume of the stomach’s contents.

Anti-muscarinic agents block acetylcholine (ACh) release from vagal postganglionic fibres and reduce gastric acidity. However, they are not used in practice because they also reduce LOS tone and delay gastric emptying, thereby increasing the risk of gastro-oesophageal reflux. H2 receptor antagonists reduce gastric acid secretion and both cimetidine and ranitidine may be given i.v. Ranitidine is preferred because it has a longer duration of action and fewer drug interactions.

Proton pump inhibitors (PPIs) irreversibly block the H⁺/K⁺ ATPase enzyme which catalyses the exchange of intracellular H⁺ for extracellular K⁺, the final step in the production of gastric acid by the parietal cells. With repeated doses PPIs are highly effective at producing achlorhydria. Omeprazole 40 mg may be given i.v. but as a single preoperative dose has no advantage over H2 receptor antagonists.

Oral antacids are given to raise the pH of the stomach’s contents by neutralizing gastric acid, but they increase gastric volume. Sodium citrate 0.3 M is used as it is non-particulate so causes less pulmonary damage if aspirated. 30 ml will neutralize the gastric contents within 10 min.

Using their knowledge of the relevant gastric physiology, the anaesthetist can reduce the incidence and consequences of gastric aspiration when required to administer general anaesthesia to a patient with a full stomach, gastro-oesophageal reflux, and/or obtundled airway reflexes.

Table 4 Gastric hormones

<table>
<thead>
<tr>
<th>Released from</th>
<th>Release stimulated by</th>
<th>Release inhibited by</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrin</td>
<td>Vagal stimulation</td>
<td>Gastric acidity</td>
<td>Stimulates gastric acid production</td>
</tr>
<tr>
<td></td>
<td>Distension of gastric antrum</td>
<td>Somatostatin</td>
<td>Increases secretion of pepsinogens</td>
</tr>
<tr>
<td></td>
<td>Distension of duodenum</td>
<td>Secretin (stimulated by duodenal acidity)</td>
<td>Increases secretion of mucus</td>
</tr>
<tr>
<td></td>
<td>Amino acids and peptides</td>
<td>Glucagon</td>
<td>Increases gastric motility</td>
</tr>
<tr>
<td></td>
<td>Alcohol</td>
<td></td>
<td>Constricts pyloric sphincter</td>
</tr>
<tr>
<td></td>
<td>Caffeine</td>
<td></td>
<td>Reduces gastric emptying</td>
</tr>
<tr>
<td>Secretin</td>
<td>Increased acidity in duodenal chyme</td>
<td></td>
<td>Reduces gastric acid secretion</td>
</tr>
<tr>
<td>CCK</td>
<td>Increased fat in duodenal chyme</td>
<td></td>
<td>Reduces gastric emptying</td>
</tr>
<tr>
<td>Somatostatin</td>
<td>Gastric acidity</td>
<td>Vagal stimulation</td>
<td>Inhibits gastrin secretion (and thereby gastric acid production)</td>
</tr>
<tr>
<td>Motilin</td>
<td></td>
<td></td>
<td>Reduces gastric motility</td>
</tr>
<tr>
<td></td>
<td>Entero-chromaffin-like cells in proximal small intestine</td>
<td></td>
<td>Associated with the inter-digestive migratory motor complex (MMC)</td>
</tr>
</tbody>
</table>

Illustrative clinical example

Suggested management of the patient with a potentially full stomach who requires a general anaesthetic

Before operation:
- Position the patient with head-up tilt and knees flexed
- Insert large-bore nasogastric tube and aspirate
- Leave nasogastric tube on free drainage
- Keep ‘nil by mouth’
- Give ranitidine 50 mg by slow i.v. injection
- Give metoclopramide 10 mg i.v. if no gastrointestinal obstruction, perforation, or haemorrhage

At induction of anaesthesia:
- Aspirate nasogastric tube
- Give sodium citrate 0.3 M (30 ml) orally
- Check patient positioned with head-up tilt
- Pre-oxygenate
- Apply cricoid pressure
- Induce anaesthesia with a rapid sequence induction
- Intubate with auffed oro-tracheal tube to secure the airway

Peroperatively:
- Maintain head-up position if possible
- Avoid increases in abdominal pressure
- Ensure adequate depth of anaesthesia

After operation:
- Extubate in the left lateral position
- Extubate when awake and gag and cough reflexes are present
References


Please see multiple choice questions 1–3