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The Anatomy and Physiology of the Stomach

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Aims

To detail the anatomy and physiology of the stomach.

Introduction

The stomach is the most dilated part of the digestive tube, having a capacity of 1000–1500 ml in the adult. It is situated between the end of the oesophagus and the duodenum – the beginning of the small intestine. It lies in the epigastric, umbilical, and left hypochondrial regions of the abdomen, and occupies a recess bounded by the upper abdominal viscera, the anterior abdominal wall and the diaphragm. It has two openings and is described as having two borders, although in reality the external surface is continuous. The relationship of the stomach to the surrounding viscera is altered by the amount of the stomach contents, the stage that the digestive process has reached, the degree of development of the gastric musculature, and the condition of the adjacent intestines. However, borders are assigned by the attachment of the peritoneum via the greater and lesser omentum, thus dividing the stomach into an anterior and posterior surface.

The principal function of the stomach is to mix the food with acid, mucus and pepsin and then release the resulting chyme, at a controlled rate into the duodenum for the process of absorption. Gastric motility is controlled by both neural and hormonal signals. Nervous control originates from the enteric nervous system as well as the parasympathetic (predominantly vagus nerve) and sympathetic systems. A number of hormones have been shown to influence gastric motility – for example, both gastrin and cholecystokinin act to relax the proximal stomach and enhance contractions in the distal stomach. Other functions of the stomach include the secretion of intrinsic factor necessary for the absorption of vitamin B_{12} (Figure 2.1).

Anatomy

Embryology

Towards the end of the fourth week of embryonic development, the stomach begins to differentiate from the primitive foregut – a midline tube, separated from the developing pericardium by the septum transversum and dorsally to the aorta. Initially a fusiform dilation forms, beyond which the midgut opens into the yolk sac. The foregut, owing to the presence of the pleuroperitoneal canals on either side, is connected to the dorsal wall by a mesentery that is continuous with the dorsal mesentery of the mid- and hindguts. Thus a primitive mesentery extends from the septum transversum to the developing cloaca. The liver and ventral pancreas (uncinate process) develop from the

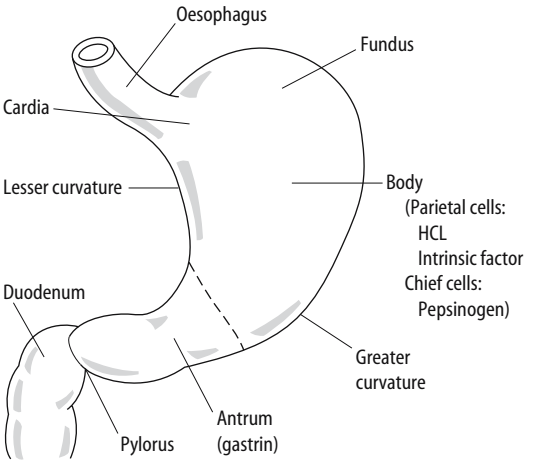

Figure 2.1. The regions and functions of the stomach. (With permission from Review of Medical Physiology, WF Ganong, 13th edition, Lange Medical Press, 1987.)

ventral aspect of the foregut and grow into the septum transversum, thus forming a ventral mesentery – the ventral mesogastrium. As the embryonic period continues the growth of the two "borders" becomes notably altered and the curvature of the stomach becomes apparent (Figure 2.2). The distal end rotates ventrally and with the increased growth of the dorsal border the concavity of the lesser curvature becomes apparent. With further increasing growth of the entire gut and the return of the gut to the abdominal cavity the stomach becomes rotated along its cranial-caudal plane so that the "stomach sac" rotates and the original right surface becomes dorsal and the left ventral. The position of the dorsal and ventral mesogastrium is affected by the rotation (Figure 2.3).

As the dorsal mesogastrium becomes increased in length, it folds upon itself forming the lesser omentum. This lies transverse rather than

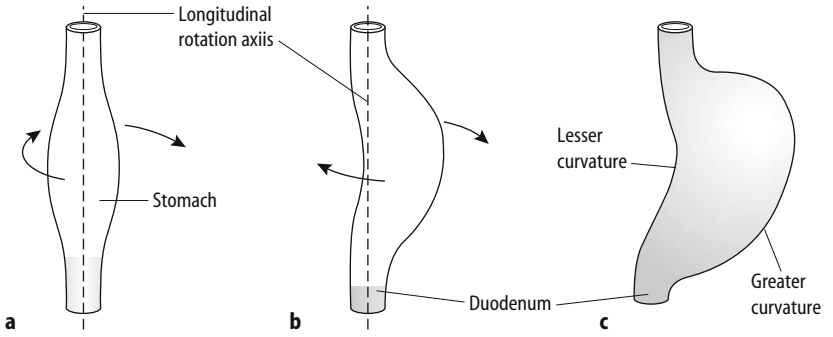

Figure 2.2. a–c The rotation of the stomach along its longitudinal axis.

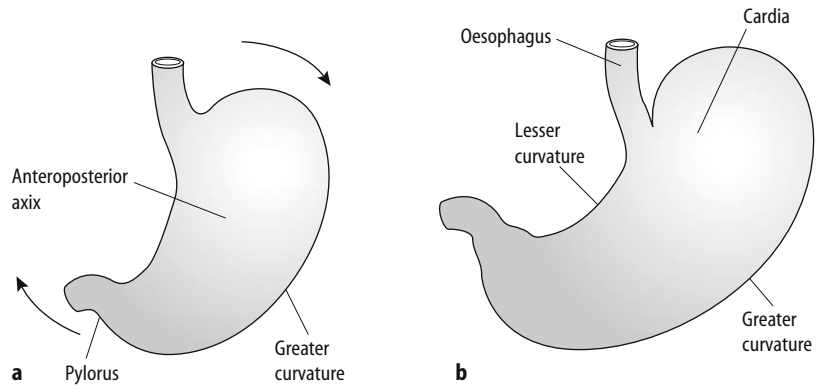

Figure 2.3. The rotation of the stomach along its anteroposterior axis. (With permission from Langman's Medical Embryology, 5th edition, Williams & Wilkins, Baltimore, 1985.)

anteroposterior and leads to the formation of the lesser sac. This lies between the stomach and posterior abdominal wall, bounded laterally on the left by the dorsal mesogastrium, anteriorly by the stomach and laterally on the right by the developing liver. The foramen of Winslow is the only opening into the space and formed by the free border of the lesser omentum, between the stomach and liver (Figure 2.4).

With the rotation of the stomach, the duodenum is carried to the right. Initially the duodenum is fixed by a thick mesentery to the posterior abdominal wall. However, with this rotation the duodenum comes to lie on the posterior abdominal wall and the primitive mesentery disappears. This results in the duodenum coming to lie retroperitoneally. Similarly the bilary ducts and pancreas come to lie within the concavity of the duodenum, the bile duct having passed behind its proximal part.

Within the folds of the dorsal mesogastrium the spleen develops and this remains intimately attached to the stomach.

Congenital Abnormalities

Pyloric Atresia

Almost all cases of gastric atresia occur in the pyloric region and may present as a membrane occluding the lumen, as a gap in continuity, or as a fibrous cord intervening between patent portions at the gastroduodenal junction. There is a reported association with epidermolysis bullosa. Clinically the condition presents as upper abdominal distension and bile-free vomiting in the newborn. Maternal hydramnios occurs in approximately 50% of cases.

Duplications

True or complete duplication of the stomach is exceedingly rare. More common (but also rare) incomplete duplications may be defined as spherical or tubular enteric formations which lie in contiguity with the normal alimentary tract and which share with it a common blood supply, and usually a common muscle coat. These cyst-like structures, or duplication cysts, usually do not communicate with the normal lumen. They may have a mucosal lining and may be pedunculated. A duplication cyst of the stomach is a communicating or non-communicating cyst lined by gastric, intestinal or pancreatic epithelium, and usually located along the greater curvature. Occasionally it may be situated in the wall of the pyloric region; in such cases encroachment on the lumen may produce gastric outlet obstruction, or an appearance resembling infantile hypertrophic pyloric stenosis. In non-communicating duplication cysts, accumulation of acid and pepsin may produce a local inflammatory reaction, perforation, abscess formation and peritonitis.

Congenital Double Pylorus, Pyloric Membrane, Web or Diaphragm

Congenital double pylorus is an extremely rare condition. A pyloric membrane is defined as a thin, circumferential mucosal septum in the pyloric region, projecting intraluminally perpendicular to the long axis of the "antrum". It is composed of two layers of gastric mucosa, with a central core of submucosa and muscularis mucosae. It is generally regarded as a congenital anomaly and is usually associated with symptoms and signs of gastric outlet obstruction.

Ectopic Pancreatic Tissue

Aberrant pancreatic nodules have been reported in the upper gastrointestinal tract. Although usually in the duodenum they have been reported in the stomach near the pylorus.

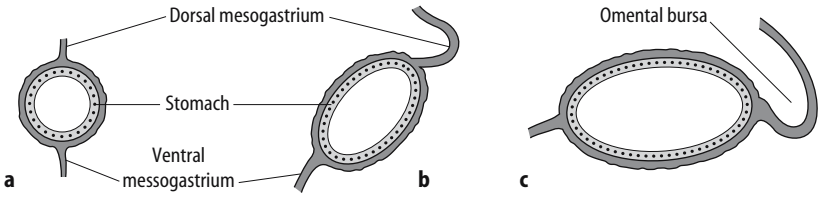

Figure 2.4.a–c The effect of rotation on the ventral and dorsal mesogastrium (**a**, **b**) and the formation of the lesser sac (omental bursa) (**c**). (With permission from Langman's Medical Embryology, 5th edition, Williams & Wilkins, Baltimore, 1985.)

Macroscopic Anatomy

The stomach has two openings, two curvatures, two surfaces and two omenta.

Openings

Gastro-oesophageal Junction

The oesophagus communicates with the stomach via the cardiac orifice, which is situated on the left of the midline at the level of T10. The intra-abdominal oesophagus (antrum cardiacum) is short and conical. After passing through the diaphragm it curves sharply to the left, and becomes continuous with the cardiac orifice of the stomach. The right margin of the oesophagus is continuous with the lesser curvature of the stomach, while the left margin joins the greater curvature at an acute angle (incisura cardiaca).

Gastroduodenal Junction

The pylorus forms the gastric outlet and communicates with the duodenum. It lies to the right of the midline at the level of the upper border of L1 and may be identified on the surface of the stomach by a circular groove (duodeno-pyloric constriction). There has long been disagreement about various aspects of the structure and function of the "gatekeeper" (Greek *pyloros*, from *pyle* = gate and *ouros* = guard). Willis (1682) introduced the term "antrum pylori" (Greek *antron* = cave) to indicate the part of the stomach adjoining the pylorus; no further demarcation was given.

Curvatures

Lesser Curvature (Curvatura Ventriculi Minor)

This extends from the cardiac to the pyloric orifices, thus forming the right or posterior border of the stomach. It is a continuation of the right border of the oesophagus and lies in front of the right crus of the diaphragm. It crosses the body of L1 and ends at the pylorus. A well-demarcated notch, the incisura angularis, is seen distally although its position varies with the state of distension of the stomach. Attached to the lesser curvature are the two layers of the hepatogastric ligament (lesser omentum). Between these two layers are the left gastric artery and the right gastric branch of the hepatic artery.

Greater Curvature (Curvatura Ventriculi Major)

This is directed mainly forward, and is four to five times longer than the lesser curvature. It starts from the incisura cardiaca and arches backward, upward, and to the left; the highest point of the convexity is on a level with the sixth left costal cartilage. It then descends downwards and forwards, with a slight convexity to the left as low as the cartilage of the ninth rib, before turning to the right, to end at the pylorus. Directly opposite the incisura angularis of the lesser curvature, the greater curvature presents a dilatation, which is the left extremity of the pyloric part; this dilatation is limited on the right by a slight groove, the sulcus intermedius, which is about 2.5 cm, from the duodenopyloric constriction. The portion between the sulcus intermedius and the duodenopyloric constriction is termed the pyloric antrum. At its commencement the greater curvature is covered at its origin by peritoneum continuous with that covering the front of the organ. The left part of the curvature gives attachment to the gastrosplenic (lineal) ligament, while to its anterior portion are attached the two layers of the greater omentum, separated from each other by the right and left gastroepiploic vessels.

Surfaces

These change with the degree of gastric distension. When the stomach is empty they may be described as anterior and posterior surfaces, but with distension become anterosuperior and postero-inferior.

Anterosuperior Surface

This surface is covered by peritoneum and lies in contact with the diaphragm, which separates it from the base of the left lung, the pericardium, the seventh–ninth ribs, and the intercostal spaces of the left side. The right half lies in relation to the left and quadrate lobes of the liver together with the anterior abdominal wall. The transverse colon may lie on the front part of this surface when the stomach is collapsed.

Postero-inferior Surface

This surface is covered by peritoneum, except over a small area close to the cardiac orifice; this area is limited by the lines of attachment of the gastrophrenic ligament, and lies in apposition

with the diaphragm, and frequently with the upper portion of the left suprarenal gland. Other relations are to the upper part of the front of the left kidney, the anterior surface of the pancreas, the left colic flexure, and the upper layer of the transverse mesocolon. The transverse mesocolon separates the stomach from the duodenojejunal flexure and small intestine. Thus the abdominal cavity is divided into supra- and infra-colic compartments.

The anterior boundary of the lesser sac (omental bursa) is formed by this surface. This potential space can be accessed via an opening on the free border of the lesser omentum, which contains the common hepatic artery, the common bile duct and the portal vein (the foramen of Winslow).

Parts of the Stomach

The stomach is divided into a pyloric part and body by a plane passing through the incisura angularis on the lesser curvature and the left limit of the opposed dilatation on the greater curvature. The body is further subdivided into the fundus and cardia by a plane passing horizontally through the cardiac orifice. Distally a plane passing from the sulcus intermedius at right angles to the long axis of this portion further subdivides the pyloric portion. To the right of this plane lies the pyloric antrum. At operations, a slight groove may be seen in the serosal surface at the gastroduodenal junction. A small, superficial subserosal vein, lying within this groove and vertically across the front of the gut may be evident. This is the prepyloric vein (of Mayo) and drains into the right gastric vein. At operation, palpation of this area reveals the pyloric ring between the thick walls of the pyloric region and the thin walls of the duodenum.

Omenta

Lesser Omentum

This extends from the inferior and posterior surfaces of the liver to the stomach and proximal 3.0 cm of the duodenum. The free border of the lesser omentum between the porta hepatis and the duodenum contains the hepatic artery, the portal vein, the common bile duct, lymph glands, lymph vessels and nerves. Behind this free edge is the opening into the lesser sac or epiploic foramen (of Winslow). The remainder of the lesser omentum, extending from the left end of the porta hepatis to the lesser curvature, contains the right and left gastric arteries and the accompanying veins, as well as lymph glands, lymph vessels and branches of the anterior and posterior vagus nerves.

Greater Omentum

This is formed along the greater curvature of the stomach by the union of the peritoneal coats of the anterior and posterior gastric surfaces. On its left it shortens into the gastrosplenic omentum, containing the short gastric branches of the splenic artery between its two layers. On the right it is continued for 3.0 cm along the lower border of the first part of the duodenum. From its origin the greater omentum hangs down in front of the intestines as a loose apron, extending as far as the transverse colon, where its two layers separate to enclose that part of the colon. The upper part of the greater omentum contains the greater part of the right and left gastroepiploic arteries and their accompanying veins, lymph vessels, lymph glands, nerve filaments, fat and areolar tissue.

Blood supply

Arterial Supply (Figure 2.5)

The coeliac artery, the artery of the foregut, supplies the stomach by its three branches. It arises from the front of the aorta between the crura of the diaphragm and is a short wide trunk, surrounded by the coeliac lymph nodes and flanked by the coeliac ganglia of the sympathetic system. The main branches are the left gastric artery, the hepatic artery and the splenic artery.

The Left Gastric Artery.

This runs to the left, gives off an ascending oesophageal branch, and supplies the upper part of the stomach. However, it may arise directly from the aorta (5–6.7%), and may provide one or both of the inferior phrenic arteries or a common trunk for the two. Duplicate arteries have been reported and sometimes an enlarged (accessory) branch (8–25% of individuals) is found. This branch may replace the left hepatic artery (11–12% of individuals). The left gastric artery turns downwards between the layers of the lesser omentum and runs to the

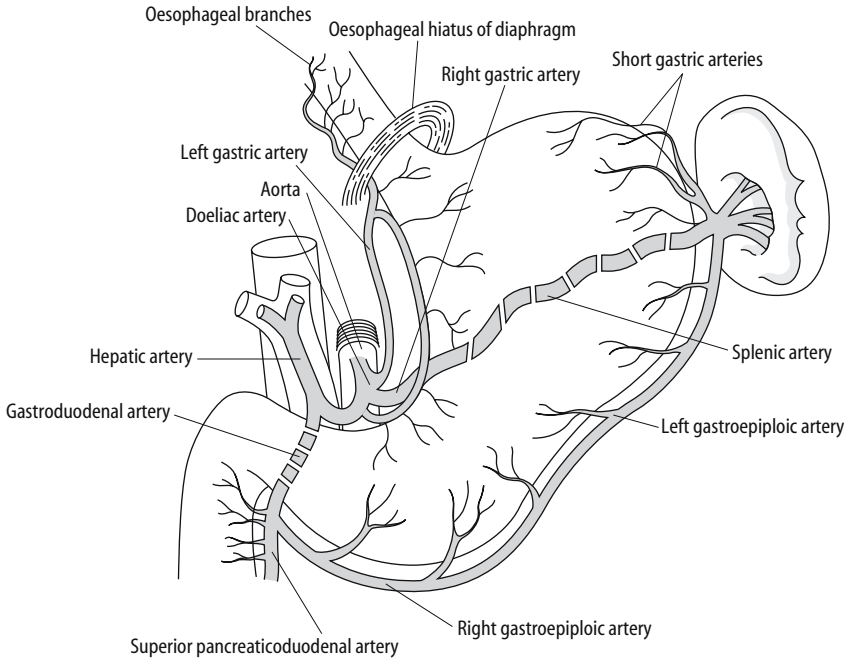

Figure 2.5. The arterial supply of the stomach. (With permission from Clinical Anatomy for Medical Students, 6th edition, RS Snell, p. 207, Fig. 5–14, Lippincott Williams & Wilkins, Philadelphia, 2000.)

right along the lesser curvature. Having divided into two parallel branches, these divide further supplying the anterior and posterior gastric walls. These vessels anastomose freely with arteries from the greater curvature. Around the incisura angularis, the two main branches then anastomose with the two branches of the right gastric artery. The hepatic artery may arise directly from the left gastric.

The Hepatic Artery

This is the second branch of the coeliac trunk and passes downwards as far as the first part of the duodenum. At the opening into right border of the lesser sac it turns forwards (epiploic foramen) and curves upwards between the two layers of the lesser omentum towards the porta hepatis, to supply the liver. The gastroduodenal and right gastric arteries are given off as it turns into the lesser omentum. The right gastric artery passes to the left between the two layers of the lesser omentum, and runs along the lesser curvature of the stomach before dividing into two branches that anastomose with the branches of the left gastric artery. It also gives off branches to the anterior and posterior gastric walls, anastomosing with branches from the right gastroepiploic artery. The gastroduodenal artery descends behind the first part of the duodenum, which it supplies by multiple small branches. The terminal divisions are the superior pancreaticoduodenal artery, supplying the second part of the duodenum and head of the pancreas, and the right gastroepiploic artery. The right gastroepiploic artery passes along the greater curvature of the stomach between the layers of the greater omentum and gives off branches to the anterior and posterior gastric walls before anastomosing with the left gastroepiploic artery.

The Splenic Artery

This passes to the left along the upper border of the pancreas, behind the peritoneum and the stomach, to supply the spleen. Division into the terminal branches close to the spleen is called a magistral splenic (~1–2 cm from the hilum), but earlier division is called a distributing splenic. During its course it gives off branches to the pancreas; just before entering the splenic hilum it gives off the short gastric

arteries supplying the gastric fornix, and the left gastroepiploic artery. The latter passes downwards and to the right along the greater curvature of the stomach, between the two layers of the greater omentum, to anastomose with the right gastroepiploic artery at the mid-portion of the greater curvature. It gives off branches to the anterior and posterior gastric walls, which anastomose with branches of the gastric arteries along the lesser curvature. These arterial arcades ramify through the submucosa, forming a rich arterial network from which branches arise to supply the mucous membrane. Therefore the mucosa is not supplied by end arteries, with the possible exception of the mucosa along the lessercurvature, which appears to receive its arterial supply directly from branches of the right and left gastric arteries.

Multiple variations of the splenic artery are reported. Commonly it may divide into two branches that reunite with the splenic vein passing through the loop thus formed. It may give rise to branches normally derived from other vessels, such as the left gastric, middle colic and left hepatic. The short gastric arteries may arise from the gastroepiploic artery, the splenic artery proper, the splenic branches of the splenic artery, or any combination thereof. Similarly the left gastroepiploic artery may originate from one of the splenic branches. In a third of cases the dorsal pancreatic artery may also arise from the splenic artery.

Multiple small branches from the hepatic and gastroduodenal arteries supply the first 2 cm of the duodenum. This part of the duodenum occupies the embryological transition zone between the coeliac and superior mesenteric vascular supplies, and the vessels, which supply it vary considerably in their size and mode of origin. This variation in blood supply may partly account for the frequency of ulceration.

The coeliac trunk may lack one or more of its main branches. These may arise from the aorta or the superior mesenteric, either independently or in conjunction with another branch. The following variations have been reported:

1. Hepatosplenogastric trunk
2. Hepatosplenic trunk (hepatic and splenic)
3. Hepatosplenomesenteric trunk (hepatic, splenic and superior mesenteric)
4. Hepatogastric trunk (hepatic and left gastric)
5. Splenogastric trunk (splenic and left gastric)
6. Coeliacomesenteric trunk (superior mesenteric in conjunction with hepatosplenogastric trunk)
7. Coeliacocolic trunk (middle or accessory middle colic arising from the coeliac trunk is extremely rare).

A posterior gastric artery, a branch of the splenic, is reported to be present in 48–68% of individuals and forms another source of the blood supply to the superior portion of the posterior gastric wall. It may also supply a superior polar artery to the spleen. These vessels have a "hidden" posterior location and may be overlooked, leading to the possibility of dangerous bleeding if damaged.

Venous Drainage

The gastric veins are similar in position to that of the arteries along the lesser and greater curvatures. These veins drain either directly or indirectly into the portal system. The major veins are:

1. ***Left gastric vein***. This runs to the left along the lesser curvature, receiving the oesophageal veins below the oesophageal hiatus in the diaphragm. It usually drains directly into the portal vein at the superior border of the pancreas.
2. ***Right gastric vein***. This runs along the lesser curvature to the right towards the pylorus. Posterior to the first part of the duodenum it joins the portal vein. It also receives the prepyloric vein which receives the veins from the first 2 cm of the duodenum.
3. ***Left gastroepiploic vein***. This passes to the left along the greater curvature and with the short gastric veins drains into the splenic vein or its tributaries. The splenic vein is joined with tributaries from the pancreas as well as the inferior mesenteric vein; these ultimately form the portal vein with the superior mesenteric vein.
4. **Right gastroepiploic vein**. This runs to the right as far as the head of the pan-

creas. Usually it joins the superior mesenteric vein and thus drains into the portal vein. However, considerable variations may occur and the right gastroepiploic may enter the portal vein directly, or it may join the splenic vein. There is no gastroduodenal vein.

Lymphatic Drainage

The gastric lymphatics arise in the subepithelial interglandular tissue of the mucosa. They pass outwards between the glands to communicate with each other in the periglandular plexus and from here the channels proceed into the subglandular plexus between the glands and muscularis mucosae. Short vessels passing through the muscularis mucosae form the submucous plexus. Larger vessels draining this plexus then pass through the muscular coats before communicating with the networks among the muscle fibres, and opening into the subserous plexus. From this subserosal plexus, valved collecting vessels radiate to the curvatures of the stomach to enter the omenta.

The lymphatics of the stomach can be divided into three systems:

1. *Intramural*. This consists of three networks; submucosal, intermuscular and subserosal. The submucosal lymphatic channels communicate freely throughout the submucosa of the stomach and to a lesser degree with the submucosal lymphatics of the duodenum; they also communicate freely with the intermuscular and subserosal networks.
2. *Intermediary*. This consists of numerous small channels between the subserosal network and the extramural collecting systems.
3. *Extramural*. This consists of four major zones of lymphatic drainage, corresponding to the arterial supply of the stomach. Ultimately all zones drain into the coeliac nodes around the coeliac arterial trunk on the anterior aspect of the aorta.

The lymphatic drainage of the stomach can be divided into four zones [1]. (Figures 2.6 and 2.7):

- *Zone 1*. This comprises the upper two-thirds of the lesser curvature and a large part of the body of the stomach. These drain into the left gastric nodes lying along the left gastric artery. These nodes are joined by lymphatics coming down from the lower part of the oesophagus, and their efferents proceed to the coeliac nodes.
- *Zone 2*. This is from the distal part of the lesser curvature, including the lesser curvature of the pyloric region, to the suprapyloric nodes along the right gastric artery. Efferent channels from the suprapyloric nodes drain to the hepatic and ultimately to the coeliac and aortic nodes.
- *Zone 3*. This zone includes the pyloric part of the stomach as well as the right half of the greater curvature. The lymphatics from these areas drain into the right gastroepiploic nodes in the gastrocolic ligament, lying along the right gastroepiploic vessels, and into the pyloric nodes on the anterior surface of the head of the pancreas. The direction of lymph flow is from above downwards, towards the pylorus and the nodes between the head of the pancreas and second part of the duodenum. From these groups, collectively called the subpyloric glands

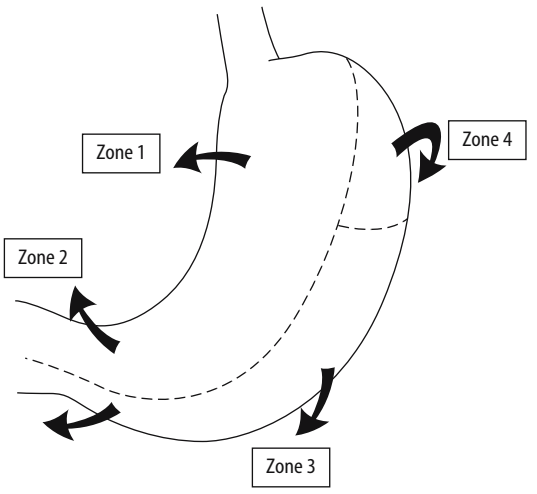

Figure 2.6. Zonal drainage of the gastric lymphatics. (With permission from Last's Anatomy, 10th edition, p. 245, Fig. 5.27, CS Sinnatamby (ed), Churchill Livingstone, London, 2001.)

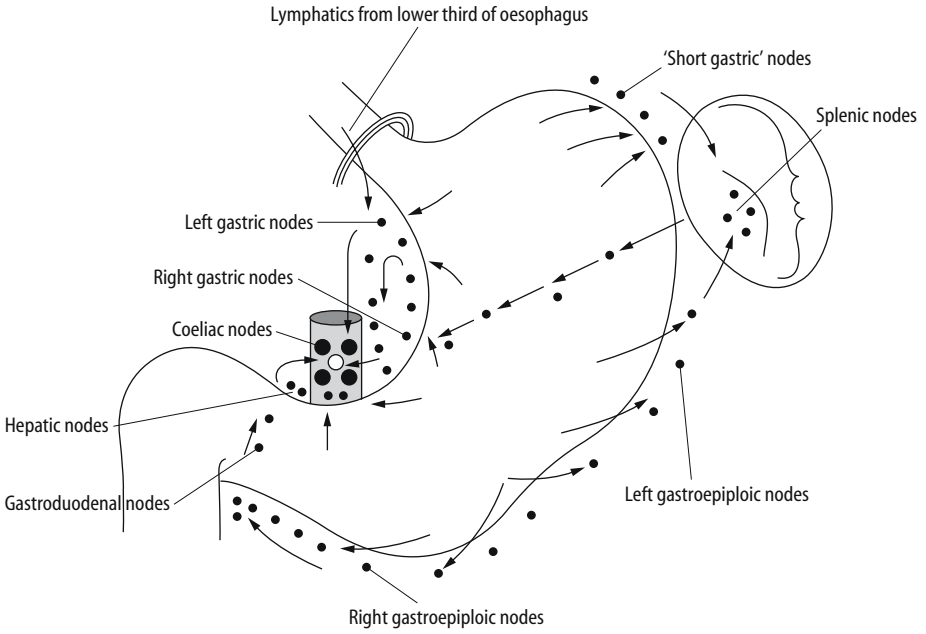

Figure 2.7. The nodal lymphatics. (With permission from Clinical Anatomy for Medical Students 6th edition, RS Snell, p. 207, Fig. 5–15, Lippincott Williams & Wilkins, Philadelphia, 2000.)

(which also drain the first part of the duodenum), efferent vessels pass along the gastroduodenal artery to the hepatic nodes along the hepatic artery, and thence to the coeliac nodes.

- *Zone 4*. This comprises the left half of the greater curvature and the gastric fornix. The lymph vessels from here pass to the left gastroepiploic nodes, lying along the left gastroepiploic artery. These drain to the pancreatico-lienal nodes along the splenic artery, before terminating in the coeliac nodes.

Nerves

The autonomic nervous system consists of two components, cholinergic – mostly parasympathetic, and adrenergic – mostly sympathetic nerves. However, a third component of the autonomic system, which is neither cholinergic nor adrenergic, has been recognised within the gastrointestinal tract – the peptidergic system. They release a purine nucleotide as the active substance. An increasing number of peptides that are released have been recognised and this has led to the concept of a three-part autonomic control system consisting of cholinergic, adrenergic and peptidergic nerves. These peptides are unique with a dual localisation in endocrine cells and peripheral nerves in the walls of the gastrointestinal tract.

Parasympathetic Nerve Supply

The anterior and posterior vagal trunks and their branches form the parasympathetic nerve supply to the stomach. Afferent fibres are also present in the vagi.

Anterior Vagus

This is derived mainly from the left vagus nerve but also includes fibres from the right vagus and also some sympathetic fibres from the splanchnic nerves. It enters the abdominal cavity through the oesophageal hiatus in the diaphragm. It is usually single but may be divided into multiple trunks. Having given off several fine branches to the lower end of the oesophagus and cardiac part of the stomach, the anterior trunk breaks up into its main branches. Latarjet's classic description of the nerves is that

three main sets of branches are present [2] (Figure 2.8) These are:

- Set 1. This consists of four to five direct branches, emanating "one below the other" to supply the upper part of the lesser curvature. These nerves do not form a plexus. A few filaments from the sympathetic supply join these direct branches via the coeliac plexus. One of the branches in this group is very distinct and Latarjet called it the "principal anterior nerve of the lesser curvature". It innervates the area from the cardia to the pylorus.
- Set 2. Branches from the vagal supply to the liver. There are usually three to five nerves and they descend in the lesser omentum, on to the superior margin of the pylorus and first part of the duodenum.
- Set 3. These consist of vagal filaments from the hepatic branches. These accompany the sympathetic nerves along the right gastroepiploic artery and provide vagal fibres to the inferior margin of the pylorus.

Latarjet divided the nerves of the anterior vagus into two distinct functional divisions. The first division, consisting of the direct branches, supplies the fornix and body, i.e. the "reservoir" part of the stomach. The second division, through the hepatic branches, supplies the pylorus and first part of the duodenum, i.e. the "sphincteric" part of the stomach.

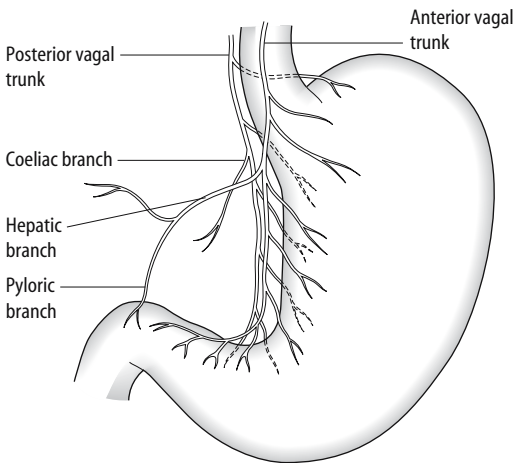

Figure 2.8. The anatomy of the nerves of Laterjet. (With permission from Clinical Anatomy for Medical Students, 6th edition, RS Snell, p. 246, Fig. 5–28, Lippincott Williams & Wilkins, Philadelphia, 2000.)

Posterior Vagus

This is mainly formed by fibres from the right vagus nerve and enters the abdomen posterior to the oesophagus. After entering the abdomen it divides into two main branches: the coeliac and the posterior gastric. It then continues along the lesser curvature innervating the posterior gastric wall although only extending to the incisura angularis. The lowest branch is sometimes referred to as "the posterior nerve of Latarjet". These nerves do not innervate the pylorus and prepyloric region.

Sympathetic Nerve Supply

This is derived almost entirely derived from the coeliac plexus. The gastric branches of the coeliac plexus accompany the vessels supplying the stomach – the left gastric, hepatic and phrenic arteries. Others accompany the splenic, right gastric and gastroepiploic vessels. Fibres from the coeliac plexus accompany the left inferior phrenic artery, pass anterior to the lower oesophagus and communicate with the anterior vagus before being distributed to the cardia and fornix. Other fibres travel with the left gastric artery and divide into three groups:

1. Those passing with the oesophageal and superior branches of the left gastric artery to the cardia and proximal part of the body of the stomach. These communicate with branches of the anterior and posterior vagal trunks.
2. Those passing with the main branch of the left gastric artery along the lesser curvature to supply the anterior and posterior surfaces of the body of the stomach and antrum.
3. Those passing through the lesser omentum towards the porta hepatis. These communicate with hepatic branches of the anterior vagal trunk

Fibres from the coeliac plexus pass along the hepatic artery and are distributed with its branches. They reach the pyloric region of the stomach with the right gastric and right gastroepiploic arteries.

Preganglionic sympathetic fibres end in the coeliac ganglia. The efferent fibres emerging from the coeliac ganglia to accompany the arteries are postganglionic. Afferent visceral fibres from the stomach travel the same course in reverse, to ganglion cells in the posterior spinal nerve roots. However, these do not synapse in the sympathetic ganglia.

Peptidergic System

Peptidergic cells are derived embryologically from neuroectoderm and are referred to as APUD cells because they synthesize monoamines through a process of amine precursor uptake and decarboxylation (APUD). They are also referred to as neuroendocrine cells. A large number of biologically active peptides have been detected in these APUD cells within the gut. These peptides include gastrin, vasoactive intestinal peptide (VIP), somatostatin, enkephalin, neurotensin and substance P.

Most of these monoamines have several molecular forms or sizes, e.g. gastrin-14, gastrin-17, gastrin-34. Some are released into the circulation, producing their biological effects in distant target organs (endocrine), whilst others act locally in the vicinity of their site of origin (paracrine) and some function as neurotransmitters (neurocrine).

Microscopic Anatomy

The wall of the stomach and the proximal 3.0 cm of the duodenum are composed of four coats. From without inwards these are the serous, muscular, submucous and mucous coats. The mucous coat is separated from the luminal contents by a layer of gastric mucus.

Serous Coat (Adventitia)

This is formed by the peritoneum, which is a thin layer of loose connective tissue covered with mesothelium. It is attached to the muscular coat, except at the greater and lesser curvatures, where it is continuous with the greater and lesser omentum respectively. Owing to its peritoneal attachments the proximal 3.0 cm of the duodenum, i.e. the proximal half of the first part of the duodenum, the duodenal bulb, is mobile. It shares the peritoneal covering of the pyloric region of the stomach and is unlike the remainder of the duodenum, which is retroperitoneal.

Muscularis Externa

Since the time of Willis (1682), there has been disagreement about the muscular layers of the stomach. The muscularis externa is composed of smooth, unstriped or involuntary fibres and is made up of three layers: an external longitudinal, middle circular, and an inner oblique layer. These inner oblique fibres are arranged in inverted U-shaped bundles over the anterior and posterior gastric walls. They loop over the fornix and extend as far as the incisura angularis. Hence these fibres have no effect on the distal stomach. In this area, including the pyloric region, the muscularis externa is composed of outer longitudinal and inner circular layers.

Submucous Coat

This is a layer of loose areolar tissue with some elastic fibres that lies between the muscularis mucosae and the muscularis externa. It is rich in mast cells, macrophages, lymphocytes, eosinophilic leucocytes and plasma cells. Within this layer the vessels and nerves divide before entering the mucous membrane. It contains arteries, veins, lymphatics and Meissner's nerve plexuses. These plexuses form part of the autonomic nervous system and contain postganglionic sympathetic fibres as well as pre- and postglanglionic parasympathetic fibres.

Unlike the duodenum (glands of Brunner), in the stomach the submucous layer does not contain any glands. However, it is wider than that of the duodenum and extends into the rugae of the stomach, forming the core of each mucosal fold.

Mucosa

This consists of three components, the muscularis mucosae, the lamina propria, and the epithelial lining.

Muscularis Mucosae

This is a thin layer of smooth muscle that forms the border between the mucosa and submucosa. It has outer longitudinal and inner circular fibres and fibres extend from the inner layer through the lamina propria around the gastric glands and toward the gastric lumen. These may compress the glands and aid their emptying.

Lamina Propria

This layer consists of a delicate network of collagenous and reticular fibres and a few fibroblasts or reticular cells. It lies between the muscularis mucosae and the surface epithelial cells with their glands and extends into the area between the necks of the glands forming a basement membrane. It is thin in the fundus and body, where the gastric glands are numerous and closely packed, but is more prominent in the cardiac and pyloric zones. It also contains plasma cells, mast cells, eosinophilic leucocytes and lymphocytes. Local accumulations of lymphocytes may occur in the cardiac and pyloric regions. Strands of smooth muscle from the muscularis mucosae traverse this layer, which also contains fine capillaries, lymphatic vessels and nerve fibres.

Epithelial Lining

A layer of simple columnar cells covers the entire luminal surface of the mucosa. However, the surface contains numerous tubular invaginations – gastric pits or foveolae. The pits are deeper in the pyloric region than elsewhere in the remainder of the stomach, extending at least halfway to the muscularis mucosae. They are V-shaped, tapering off into the glands that open into them.

The mucus-secreting columnar cells lining the luminal surface and the pits are joined by tight junctions. This may act as one of the mechanisms to protect the underlying layers against luminal acid. The supranuclear portions of the cells contain dense, homogeneous, spherical or ovoid granules consisting of a type of mucigen, which upon release into the lumen gives rise to the layer of mucus that covers the luminal surface of the mucosa. In the cells of the gastric pits, the granules become progressively less abundant at deeper levels, and in the bottom of the pits they form only a thin layer immediately beneath the cell surface. These cells continue into the necks of the gastric glands. Under physiological conditions, the surface mucous cells are continuously desquamated into the lumen and are completely replaced every 3 days. Newly formed cells appear in the deeper parts of the foveolae and in the necks of the glands; these are slowly displaced upward and continually replace those lost on the surface.

Mucosal Zones

The mucous membrane of the entire stomach is lined by glands that open into the gastric pits. The blind ends of the glands extending into the mucosa are slightly expanded and coiled, sometimes dividing into two or three branches (Figure 2.9). The gastric mucosa can be divided into three zones, based on the predominant cell types within the glands (Tables 2.1 and 2.2).

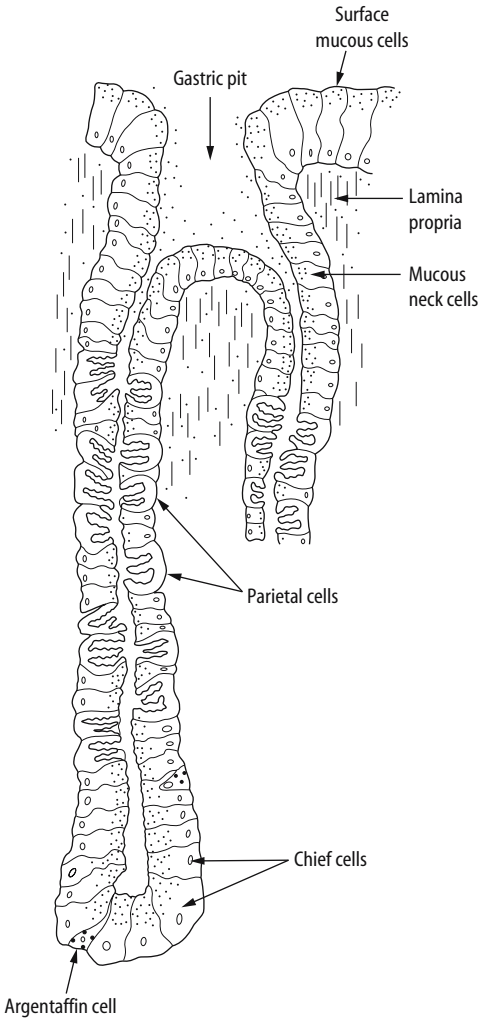

Figure 2.9. The gastric glands. (Reproduced with permission from Applied Physiology for Critical Care, MA Glasby, CL-H Huang, 1st Edition, 1995, Fig. 34.1, p. 337, Butterworth Heinemann Oxford.)

Table 2.1. Summary of the mucosal zones

Oxyntic zone	These glands produce nearly all the enzymes and hydrochloric acid secreted in the stomach as well as producing mucus
Cardiac zone	These glands secrete mucus
Pyloric zone	These glands secrete mucus. They also produce endocrine, paracrine or neurocrine regulatory peptides by virtue of the APUD cells contained in their glands

Table 2.2. The secretory epithelial cells and their roles

Four major types of secretory epithelial cells cover the surface of the stomach and extend down into gastric pits and glands:

Mucous cells: secrete alkaline **mucus** that protexts the epithelium against shear stress and acid

Parietal cells: secrete **hydrochloric acid**

Chief cells: secrete **pepsin**, a proteolytic enzyme

G cells: secrete the hormone **gastrin**

Cardiac Zone

This is a narrow, ring-shaped area around the gastro-oesophageal junction, containing the cardiac glands. These glands have wide lumina and shallow pits and are composed of mucus-secreting cells. This zone may contain a few APUD cells that synthesise monoamines. In the transitional area, where this zone is continuous with the oxyntic zone, a few parietal cells may be present. The glands of the cardiac zone secrete mucus.

Oxyntic Zone

This comprises the proximal two-thirds or more of the stomach. The glands are known as fundic glands, proper gastric glands or principal gastric glands. One of their most important properties is the secretion of gastric acid. The term oxyntic (Greek: acid- forming) is also used as an indicator of this glandular zone. The mucosa here is much deeper than in the cardiac zone and contains a greater number of glands. The pits are shallow, but the glands extending from the bottoms of the pits are longer than the pits are deep.

Each principal gastric gland is composed of four kinds of cells:

1. Chief, zymogenic or peptic cells. Their secretory granules contain the precursors of pepsin.
2. Parietal or oxyntic cells. These are most numerous in the necks of the glands, but do not border directly onto the lumen, being separated from it by the peptic cells. They are triangular in shape, with the apex projecting towards the lumen between the sides of two peptic cells. These cells are intensely acidophilic, and contain the gastric proton pump mechanism that produces the hydrochloric acid. They may also contain intrinsic factor.
3. Neck mucous cells. These cells resemble the mucous cells of the cardiac and pyloric zones. They lie between the parietal cells in the necks of the glands but are smaller than the surface mucous cells. Their mucigen granules are larger and less dense than those of the surface cells.
4. Neuroendocrine cells. These are small, granulated cells that occur sporadically within the gastric mucosa. They synthesise and store serotonin (5-hydroxytryptamine, 5-HT). They are much more numerous in the pyloric zone.

Pyloric Zone

This comprises the distal third of the stomach and extends further along the lesser curvature than the greater. The pits are the deepest within the stomach and extend into the mucous membrane for half its thickness. These glands branch more extensively and the tubules are coiled. They contain the following types of cells:

1. Mucous cells. These are similar to the neck mucous cells of the oxyntic glands and constitute the majority of cells in the pyloric glands. They have a pale cytoplasm containing indistinct granules, the nucleus is often flattened against the base of a cell, and short microvilli covered by a layer of mucus are present on the luminal surface.
2. Parietal cells. A few isolated parietal cells may be present among the mucous cells. Parietal cells also occur in the transitional region between the pyloric and oxyntic zones.

3. Neuroendocrine cells. These cells are much more numerous in the pyloric than in the cardiac and oxyntic zones although when compared with the mucous cells they are still relatively few in number. With light microscopy they have been called enterochromaffin cells. With electron microscopy their cytoplasmic granules are clearly visible after staining with chromium or silver salts. On the basis of their staining reactions, the cells have been divided into two types: argentaffin cells, in which the granules reduce silver without pretreatment, and argyrophilic cells, in which a reducing substance is required before the granules will react with silver.

The mucosal zones of the stomach are not sharply defined, the glands of one region mingle with those of the adjoining region and intermediate glands may be present between the mucosal zones.

Physiology

Gastric Secretions

The cells of the gastric glands secrete about 2500 ml of gastric juice daily. This contains a variety of substances and gastric enzymes, whose role is to kill ingested bacteria, aid protein digestion, stimulate the flow of bilary and pancreatic juices and provide the necessary pH for pepsin to begin protein degradation (Table 2.3).

Mucus Secretion

The most abundant epithelial cells are mucus-secreting columnar cells, which cover the entire luminal surface and extend down into the glands as "mucous neck cells". These cells secrete bicarbonate-rich mucus that coats and lubricates the gastric surface, and serves an important role in protecting the epithelium from acid and other chemical insults. It is made up of glycoprotein subunits bound by disulphide bonds and forms a water-insoluble gel that is impermeable to H^+ ions. Production is stimulated by luminal acid and vagal activity, and is increased by prostaglandins. Therefore aspirin non-steroidal anti-inflammatory drugs (NSAIDs) increase the damage to the stomach by inhibiting prostaglandin formation as well as by crystallising out in the gastric cells. Bicarbonate is also secreted from parietal cells. These epithelial barrier cells are very adherent due to tight junctions between them. After epithelial disruption the cells migrate along the exposed basement membrane to fill in the defect and then stick tightly together. Gastric cells also can turn over rapidly in response to injury, as there is a rich mucosal blood flow providing oxygen, bicarbonate and nutrients and removing acid. Blood flow is normally increased simultaneously with acid secretion and is reduced by aspirin and alcohol.

Table 2.3. Contents of normal (fasting) gastric juice

Cations:	Na^+, K^+, Mg^{2+}, H^+
Anions:	Cl^-, HPO_4^{2-}, SO_4^{2-}
Pepsins:	I–III
Gelatinase	
Mucus	
Intrinsic factor	
Water	

Pepsinogen Secretion

The chief cells secrete pepsinogens, contained in zymogen granules. These are the precursors of the pepsins (proteases) in gastric juice. Once secreted, pepsinogen I is activated by the presence of gastric acid into the active protease pepsin. This is an endopeptidase that is largely responsible for the initiation of protein digestion into smaller peptides and polypeptides. It splits the long amino acid chains in the region of peptide bonds containing aromatic amino acids. It acts at pH 1.5–2.5 and above pH 5.4 is inactivated. It is released mainly by vagal stimulation but also by histamine gastrin secretion, alcohol, cortisol, caffeine and acetazolamide. Pepsinogen release may also occur during periods of hypoglycaemia and prolonged increased intracranial pressure.

Hormone Secretion

The principal hormone secreted from the gastric epithelium is gastrin, a peptide that is important in control of acid secretion and gastric motility (see below).

Other Secretions

Gastric epithelial cells secrete a number of other enzymes, including an acid-resistant lipase and

gelatinase. The lipase hydrolyses triglycerides of medium- and short-chain fatty acids into glycerol and free fatty acids.

Intrinsic factor, a glycoprotein secreted by parietal cells, is necessary for intestinal absorption of vitamin B_{12}. It acts by combining with the vitamin B_{12} and is necessary for its attachment to receptors in the terminal ileum. Lack of intrinsic factor due to reduction in parietal cell mass following gastric surgery, or the production of antibodies to the cells, called pernicious anaemia, leads to megaloblastic anaemia. Secretion of intrinsic factor occurs following vagal, gastrin or histamine stimulation of the parietal cells.

The Formation and Secretion of Gastric Acid

Stimulation of the parietal cells results in acid secretion (Figure 2.10). These cells contain multiple tubulovesicular structures within their cytoplasm that on stimulation move to the mucosal membrane and fuse with it, producing a microvillous appearance that increases the surface area. This results in the presence of the H^+-K^+ ATPase that transports the H^+ onto the luminal surface. This secretion is isotonic with other fluids and its pH is <1.

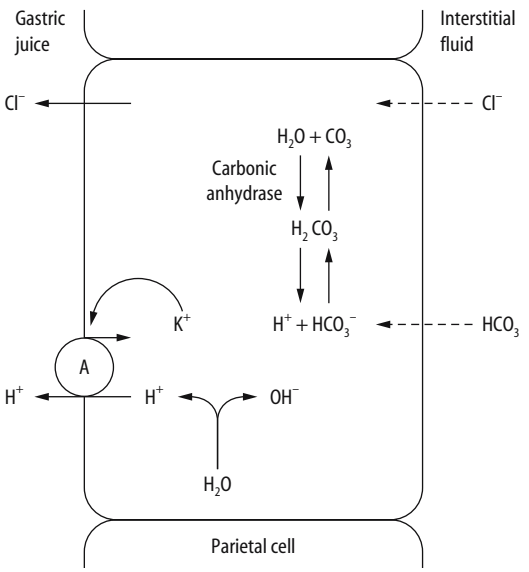

Figure 2.10. Hydrochloric acid production from the parietal cell.

The H^+ is obtained from the ionisation of water, which is then actively transported into the gastric lumen in exchange for K^+ that has been recycled from the membrane. Chloride ions are also actively transported into the gastric lumen. The resulting OH^- ion is neutralised by the carbonic acid buffer system to form a bicarbonate ion that diffuses into the interstitium to be replaced by a further Cl^- ion. There is a HCO_3^-–Cl^- exchange mechanism within the interstitium, but Cl^- also enters the cell with Na^+. The carbonic acid is replenished by the hydration of CO_2, which is produced by cellular metabolism from the abundance of carbonic anhydrase within the mucosa. After a meal this results in the development of a negative respiratory quotient; thus arterial CO_2 is higher than venous and the gastric venous return is alkaline with a high HCO_3^- content.

Gastric Hormones

Gastrin

Experiments in the early twentieth century using injected extract of pyloric mucosa stimulated secretion of gastric acid and pepsinogen. This action was thought to be hormonal in origin and the active substance was called gastrin [3]. However, this theory was initially disputed because this action was similar to that of histamine and the isolation of gastrin was not performed until the late 1960s when two related heptadecapeptides were identified from hog antral mucosa. These heptadecapeptides were isolated in the pyloric zone. The highest density of gastrin-producing G cells occurs in the distal 3.0 cm of the stomach, where the concentration of gastrin is 500 times higher than in the body of the stomach. The first part of the duodenum also contains a significant level of G cells. These cells originate from neuroectoderm together with other cells of the APUD series.

Microscopically they are piriform in shape and located in the mid and deep zones of the pyloric mucosal glands. Electron microscopy shows that they possess microvilli extending into the lumen and that secretory granules are present in the basal parts of the cells. This allows for secretion of hormone into the bloodstream in response to luminal stimuli.

There are two main types of gastrin, gastrin I and gastrin II, produced predominantly by the

G cells of the pyloric mucosal zone. Other sources are the duodenal G cells, D cells in the islands of Langerhans in the pancreas, and isolated G cells in the proximal-acid-producing region of the fornix and body of the stomach.

Gastrin 17 (17 amino acids) is the predominant form in the pyloric antrum, and is further subdivided into a non-sulphated gastrin I and a sulphated gastrin II form. There is also a "big" gastrin (G34) containing 34 amino acids and a "big big" gastrin with many more amino acids. "Mini" gastrin containing 14 amino acids can also be isolated, but is less active than G17. The common factor in all the molecules is the C-terminal tetrapeptide Tyr-Met-Asp-Phe-NH_2.

Fasting levels of gastrin are increased by achlorhydria associated with pernicious anaemia, any form of surgical vagotomy, Zollinger–Ellison syndrome (gastrinoma), chronic renal failure and massive small bowel resection.

Its physiological and pharmacological effects are summarised in Table 2.4.

Somatostatin

Somatostatin is a tetradecapeptide that was initially found to inhibit the release of growth hormone (GH) from the pituitary gland. However, it has subsequently been identified widely in the central nervous system, the gastrointestinal tract and other organs, with the highest concentration being found in the pancreas. In the stomach it is found in the pyloric and oxyntic mucosal zones but not in the cardiac zone. Within the pancreas it is isolated from the islet D cells.

Table 2.4. The physiological and pharmacological effects of gastrin

Gastrin causes:	Parietal cells to stimulate acid secretion
	Pepsin and intrinsic factor secretion
	Increased mitotic activity in the stomach and small bowel mucosa
	Contraction of the lower oesophageal sphincter
	The release of insulin, glucagon and calcitonin
	Pancreatic stimulation and bile flow
	Small bowel secretion
	Gastric and small bowel motility to increase
	The gastrocolic reflex

Although initially thought only to suppress the secretion of growth hormone, it also possesses a wide variety of inhibitory actions on other pituitary and extrapituitary secretions. It suppresses the release of thyroid-stimulating hormone by the pituitary, the release of glucagon, insulin and exocrine secretions by the pancreas, the secretion of cholecystokinin, motilin and secretin by the intestine, and the secretion of gastrin, gastric acid and pepsin by the stomach.

Somatostatin suppresses gastric acid secretion by direct action on the parietal cells of the cardiac and oxyntic mucosal zones. Thus by lowering the pH, it also inhibits the secretion of gastrin through a feedback loop of low pH suppressing both gastric acid and gastrin secretion.

Vasoactive Intestinal Peptide (VIP)

Vasoactive intestinal peptide (VIP) is a polypeptide with strong vascular effects isolated from small intestine. It has been subsequently demonstrated in central and peripheral neurones, suggesting a neurotransmitter function. In the peripheral autonomic system VIP nerves occur in various regions, including the superior and inferior mesenteric ganglia, and the submucous (Meissner's) and myenteric (Auerbach's) plexuses of the intestinal wall. Structures believed to exert a sphincteric function receive a particularly rich supply of VIP nerves, more so than the smooth muscle of adjacent regions. Among these are the oesophagogastric junction, the pyloric "sphincter", sphincter of Oddi, internal anal sphincter, and the openings of the ureters and urethra into the trigonum of the bladder.

In the stomach these nerves are found around oxyntic and pyloric mucosal glands. In the duodenum VIP (and substance P) is present in nerves in the villi and muscularis mucosae and around blood vessels and between the lobules of Brunner's glands. Its actions include vasodilatation, thus lowering blood pressure, increased cardiac output, glycogenolysis and relaxation of smooth muscle. In the stomach there is significant inhibition of gastric secretion associated with VIP release. It probably acts as a neurotransmitter in a paracrine, rather than in an endocrine, way. The VIP neurones have been shown to be under dual (both vagal and splanchnic) control of the autonomic system.

Substance P

In the gastrointestinal tract nerve fibres and neurones containing substance P (11-amino-acid peptide) are encountered along its entire length. However, they are least prominent in the oesophagus and upper part of the stomach, but the highest concentrations occur in the duodenum. These neurones are located mainly in the myenteric plexuses. Here the nerve fibres richly innervate the circular musculature. However, the longitudinal muscle contains only a sparse network of fibres. Substance P may also be vasoactive as the nerve fibres are also found in close contact with blood vessels.

In the stomach substance P is found in the oxyntic zone in a few, thin fibres only and in fibres interconnecting in the pyloric antrum. In the duodenum substance P (and VIP) is present in nerve networks in the villi as well as in the muscularis mucosae and around blood vessels. It has been found to cause contraction of the muscularis mucosae.

Other Gastric Hormones

Encephalin

These are endogenous opiate-like compounds forming two pentapeptides, endorphin and enkephalin. They can be isolated throughout the gastrointestinal tract, although the highest concentration is found in the pyloric antrum. The role of these peptides is not clear.

Galanin

Galanin may act as a regulatory factor in the control of gastrointestinal motility. It is usually found in close association with VIP-containing nerves.

Neurotensin

This is secreted by N cells in ileal mucosa. However, small traces occur in the pyloric mucosal zone. The neurotensin level rises after a meal, but its function is still unclear. It may inhibit pentagastrin-stimulated gastric acid and pepsin secretion after a meal as well as delaying gastric emptying, resulting in the controlled release of chyme into the small intestine.

Absorption from the Stomach

The stomach absorbs very few substances. Fats are not absorbed. Polypetides are absorbed only slightly. Sugars are absorbed to an extent and this varies with the sugar and its concentration. Galactose is most readily absorbed followed by glucose, lactose, fructose and finally sucrose. Low concentrations of sugars are absorbed very slowly. Ethyl alcohol is absorbed fairly rapidly as are other lipid-soluble compounds including aspirin and other NSAIDs. These substances are also well-recognised causes of gastric irritation and their use (especially overuse) is commonly associated with development of gastritis and gastric ulcers. The stomach absorbs water readily with half the ingested volume absorbed in about 20 minutes.

Regulation of Gastric Secretion and Motility

Gastric function is classified into three phases in which secretory and motor activities are closely linked.

Control of Gastric Acid Secretion

Acid secretion may be divided into two phases interprandial, when acid secretion is 1–5 mmol/h, and stimulated where acid secretion is maximally 20–35 mmol/h. This is further subdivided into cephalic, gastric and intestinal phase. Normal subjects maximally secrete 0.5 mmol/h/kg body weight)

Interprandial

Resting secretion occurs in the absence of all intestinal stimulation. However, to abolish all gastic acid secretions, a bilateral vagotomy (truncal) and excision of the pyloric antrum would be necessary.

Stimulated Secretion

The Cephalic Phase (Figure 2.11). The cephalic phase is initiated by seeing, smelling and anticipating food. These influences act on the limbic system and hypothalamus and these nuclei stimulate the dorsal motor nucleus of the vagus. This stimulus is transmitted thought the vagus nerve to the enteric nervous system, resulting in release of acetylcholine in the vicinity of G cells and parietal cells. Binding of acetylcholine to its receptor on G cells induces secretion of the hormone gastrin, which, in concert with acetylcholine and histamine, stimulates parietal cells to secrete small amounts of acid. Additionally, a low level of gastric motility is induced.

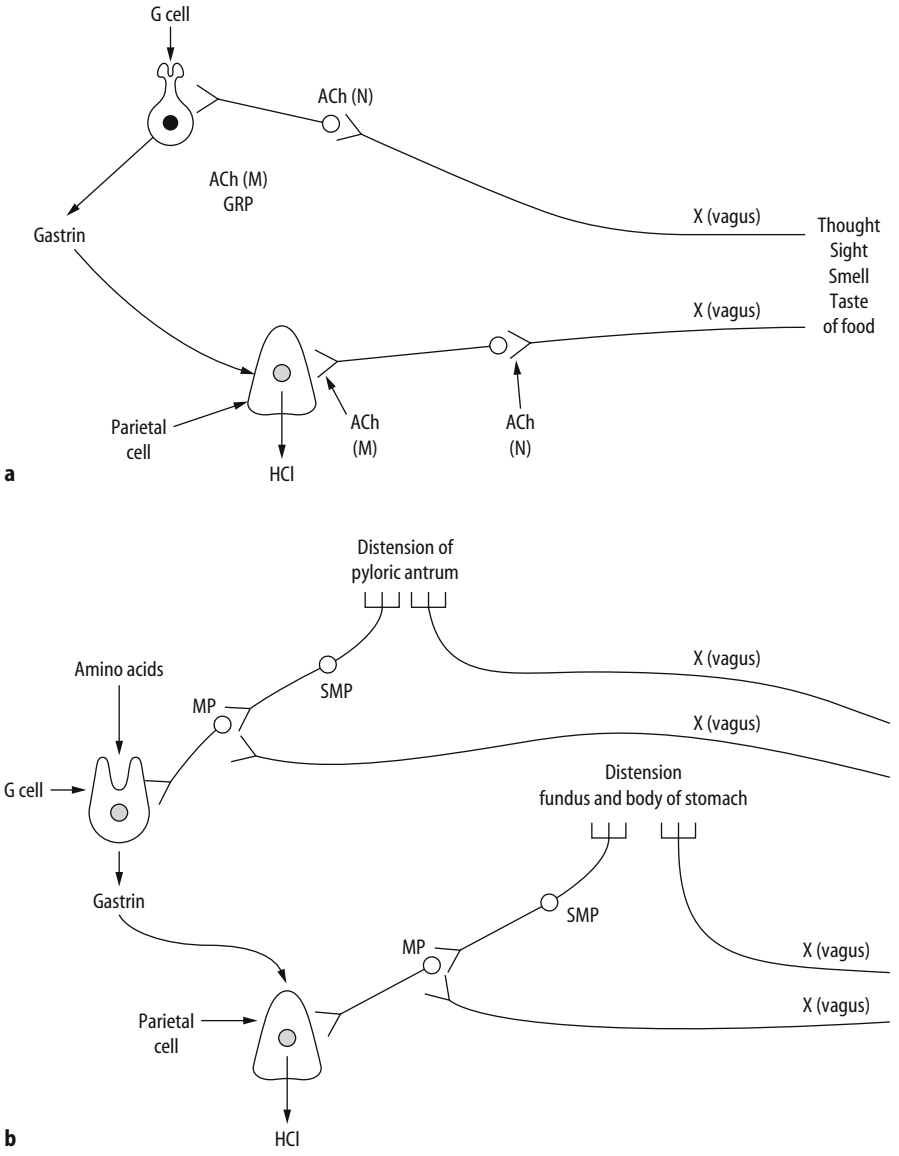

Figure 2.11. The cephalic (**a**) and gastric phases (**b**) of acid secretion. X, vagus; ACh (N), acetylcholine (nicotinic receptor); ACh (M), acetylcholine (muscarinic receptor); GRP, gastrin-releasing peptide; SMP, submucous plexus; MP, myenteric plexus. (Reproduced with permission from Applied Physiology for Critical Care, MA Glasby, CL-H Huang, 1st edition, 1995, Fig. 34.3, p. 340, Butterworth Heinemann, Oxford.)

The release of acetylcholine and bombesin (gastrin-releasing peptide) initiates gastrin release from the G cells. The gastrin passes via the portal circulation to stimulate the parietal cells. It potentiates the effect of vagal stimulation, thus resulting in increased acid secretion. The parietal cells also have H_2 receptors (histamine) stimulated by the release of histamine from mast cells close to the parietal cells. The histamine sensitises the parietal cell to the action of gastrin and acetylcholine. The H_2 receptor blockers (cimetidine and ranitidine) act on these receptors, thus reducing acid secretion.

The Gastric Phase (Figure 2.11). When food enters the stomach several additional factors come into play, foremost among them being distension and mucosal irritation. Distension excites stretch receptors and irritation activates chemoreceptors in the mucosa. These events are sensed by enteric neurones, which secrete additional acetylcholine, further stimulating both G cells and parietal cells. Gastrin from the G cells feeds back to the parietal cells, stimulating it even further, mediated by vagovagal reflexes through the dorsal motor nucleus. Additionally, activation of the enteric nervous system and release of gastrin cause vigorous smooth muscle contractions. The net result is that secretory and motor functions of the stomach are fully turned on – acid and pepsinogen are secreted, pepsinogen is converted into pepsin and vigorous grinding and mixing contractions take place.

However, acid secretion may be inhibited during the gastric phase by local mechanisms. If the antral pH falls to 1–1.5, inhibition of gastrin release occurs. This is mediated by two mechanisms – the effect of luminal acid on the microvilli of the G cell and the stimulation of somatostatin from D cells in the antrum, which acts inhibits directly on the G cells and parietal cells by a local paracrine effect.

The Intestinal Phase. As chyme is emptied into the small intestine control is necessary to limit gastric empting. This probably allows the duodenum time to neutralize the acid and efficiently absorb incoming nutrients. Hence, this phase of gastric function is dominated by the small intestine sending inhibitory signals to the stomach to slow secretion and motility. Two types of signals are used: nervous and endocrine. Distension of the small intestine, as well as chemical and osmotic irritation of the mucosa, is transduced into gastric-inhibitory impulses in the enteric nervous system – this nervous pathway is called the enterogastric reflex. Fat and carbohydrate in the chyme cause the release of GIP (gastric inhibiting peptide), which inhibits gastrin secretion. Secondly, enteric hormones such as cholecystokinin and secretin are released from cells in the small intestine and contribute to suppression of gastric activity. Gastrin also causes the release of calcitonin from the C cells of the thyroid gland, which inhibits further release of gastrin via a feedback loop.

Collectively, enteric hormones and the enterogastric reflex put a strong brake on gastric secretion and motility. As the ingesta in the small intestine is processed, these stimuli diminish, the damper on the stomach is released, and its secretory and motor activities resume.

Gastric Motility and Hunger Contraction

Resting Electrical Activity Within the Stomach

A pacemaker in the longitudinal muscle close to the greater curve of the cardia controls the frequency of contractions. It depolarises at a rate of 3/minute, and each wave – the gastric slow wave (or basal electrical rhythm) – increases sodium permeability across the cell membrane and the impulse spreads through the longitudinal and circular muscles via low resistance junctions. These junctions make up about 12% of the membrane surface.

In the empty stomach (approximately 50 ml volume), the resting potential is low (–50 mV) and although the waves pass at a rate of 3/minute, not all of these waves are equal in amplitude and do not set off an action potential. However, when the critical firing level is passed the resulting action potential sets off an excitation–contraction coupling and a contraction spreads throughout the stomach.

Intragastric Pressure

Intragastric pressure remains relatively constant at 5 mmHg (0.7 kPa) because as food passes into the stomach, the musculature of the fundus and body relaxes via a feedback loop – receptive relaxation. In addition, as wall tension rises so does the radius, thus keeping the intragastric pressure constant (law of Laplace). However, above 1000 ml, the radius cannot increase in size so the wall tension and intragastric pressure rises.

Thus volumes above 1000 ml lead to stimulation of stretch receptors within the stomach wall.

Gastric Tone

In most instances gastric hypotonicity is of idiopathic origin and presumably of little clinical significance. More severe degrees, sometimes progressing to acute gastric dilatation, may occur in a variety of conditions, e.g. post-

operatively, after severe trauma and in electrolyte disturbances.

A short, transversely situated, "steerhorn" stomach, on the other hand, is now known to be the result of gastric hypertonicity. In these cases immediate emptying of liquid barium usually commences in the erect position, before the onset of peristalsis or cyclical contractions of the pyloric sphincteric cylinder.

Control of Gastric Motility

As the volume of the stomach passes 1000 ml the intragastric wall tension rises. This activates stretch receptors, again through a vagovagal reflex arc, which cause depolarisation in the longitudinal and circular smooth muscles. However, this leads to every slow wave being above the critical firing level. An action potential is therefore propagated with each slow wave and a contraction passes from the fundus through the body to the pyloric antrum. These now occur three times per minute and the force of the contraction also increases along the stomach. Initially low in the fundus, where the muscular layer is thinnest, at the pylorus intragastric pressure may reach 40–50 mmHg (5.3–6.7 kPa).

The pyloric sphincter is not a high-pressure zone and is open in the resting phase. As a contraction wave arrives it contracts. However, since the canal was open before the rise in intragastric pressure some of the chyme passes through the pylorus (5–15 ml) before the gastric slow wave reaches it. When the slow wave reaches the antrum and pylorus they contract together – terminal contraction, and the pylorus closes. This phase of the contraction acts to recirculate or "churn" the gastric contents. Only when duodenal pressure drops due to relaxation does the pyloric pressure drop.

Factors Modifying Gastric Motility

Both vagal stimuli and gastrin increase antral motility and influence emptying. However, after a truncal vagotomy, the force of the antral pump is reduced and gastric emptying time is prolonged, hence the need for a drainage procedure after a truncal vagotomy.

The force of the pump is also moderated by the volume and composition of the chyme. Hormonal and neuronal mechanisms also regulate gastric emptying. These include duodenal distension – via a vagal feedback loop, increased duodenal osmolarity – via osmoreceptors, the presence of acid in the duodenum – via a local enteric neuronal pathway, and the release of GIP and chalesystokinin by fat in the duodenum.

In addition, sympathetic stimuli reduce gastric emptying via the limbic and hypothalamic nuclei.

Hunger Contractions

When the stomach is empty there can be periods of increased gastric motility several hours after a meal. With stimulation of the hypothalamus the individual feels hungry. This leads in a rise in vagal stimulation and causes increased gastric motility. Contraction of the empty stomach leads to a rise in intragastric pressure that can stimulate tension and pain receptors in the gastric wall, simulating mild pain or discomfort.

Changes in Physiology and Function Related to Disease

Nausea, Retching and Vomiting

The mechanism of vomiting in mammals is complex and in spite of experimental studies some aspects are still not fully understood. It is usually accepted that the vomiting sequence consists of three successive phases: nausea initially, followed by retching, often leading to forcible expulsion of gastric contents through the mouth, i.e. ejection or vomiting. During these stages a coordinated sequence of movements occurs, involving, amongst others, the upper small bowel, stomach, oesophagus, diaphragm, voluntary abdominal muscles and glottis. The complex movements of the ejection phase occur with extreme rapidity. The action is controlled by the vomiting centre present bilaterally in the medulla oblongata at the level of the olivary nuclei, and close to the tractus solitarius at the level of the dorsal vagal nuclei. A complex pathway mediated via efferents in the fifth, seventh, ninth, tenth and twelfth cranial nerves leads to contraction of the intercostal muscles, diaphragm and abdominal muscles. Afferent impulses pass via the vagus (tenth cranial nerve) and sympathetic nerves to the vomiting centre. However, other impulses reach via the labyrinth, the limbic system and the chemoreceptor trigger zone. This is situated in the lateral wall of the fourth ventricle.

Nausea is the conscious recognition of the subconscious excitation of an area known as the medulla oblongata closely associated with the vomiting centre and can be initiated by impulses from the gastrointestinal tract, the lower brain in association with motion sickness or cortical impulses. Vomiting without the prodromal phase of nausea can occur, indicating that only certain portions of the vomiting centre are associated with it.

The Process of Vomiting

If the upper gastrointestinal tract becomes excessively irritated, over-distended or overstimulated, vomiting may occur. Initially antiperistaltic waves begin and may occur as far down as the ileum. These waves travel at 2–3 cm/s; thus within a few minutes a large volume of intestinal contents may be pushed back into the stomach and duodenum causing distension. This may result in retching.

Retching Phase

The retching phase is characterized by a series of violent spasmodic abdomino-thoracic contractions with the glottis closed. During this time the inspiratory movements of the chest wall and diaphragm are opposed by the expiratory contractions of the abdominal musculature. At the same time movements of the stomach and its contents take place. Whereas a patient will complain of disagreeable sensations during nausea, speech is not possible during retching. The characteristic movements furnish a ready diagnostic sign of the retching phase.

The Vomiting Act

Once the act of vomiting has been triggered, a deep inspiratory movement occurs, with elevation of the hyoid bone, which opens the upper oesophageal sphincter and closes the glottis, the soft palate rises to close the posterior nares and then a downward contraction of the diaphragm with simultaneous contraction of the abdominal wall muscles raises intragastric pressure. With the sudden relaxation of the lower oesophageal sphincter, expulsion occurs.

Questions

1. Outline congenital abnormalities.
2. Describe lymph drainage of stomach and relate this to surgical excision.
3. Describe nerve supply.
4. Name cells of the gastric wall and their functions.

References

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Further Reading

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