

In this article...

- Anatomy and functions of the large intestine
- Processes of fluid absorption and faeces formation
- Transport of faeces in the large intestine

Gastrointestinal tract 5: the anatomy and functions of the large intestine

Key points

The large intestine comprises the caecum, colon, rectum, anal canal and anus

Haustral and antiperistaltic contractions help it absorb water and electrolytes

The large intestine is where faeces form from food residues, water and bodily by-products

Mass movements push faeces towards the anus and create an urge to defecate

Constipation can indicate serious physiological disturbance or disease

Authors Yamni Nigam is professor in biomedical science; John Knight is associate professor in biomedical science; Nikki Williams is associate professor in respiratory physiology; all at the College of Human Health and Sciences, Swansea University.

Abstract In the large intestine – the final section of the gastrointestinal tract – absorption of water and electrolytes takes place and colonic bacteria complete the process of chemical digestion. The large intestine is also where faeces are formed from the remains of food and fluid combined with by-products of the body. Intestinal content is pushed back and forth by haustral contractions and antiperistaltic contractions, until faeces are finally pushed towards the anal canal by mass movements. This article, the fifth in a six-part series exploring the gastrointestinal tract, describes the anatomy and functions of the large intestine.

Citation Nigam Y et al (2019) Gastrointestinal tract 5: the anatomy and functions of the large intestine. *Nursing Times* [online]; 115: 10, 50-53.

With the exception of ingestion, the small and large intestines carry out all the major functions of the digestive system. This is where the ‘real business’ of digestion takes place. The intestines take up most of the space in the abdominal cavity and constitute the greatest portion of the gastrointestinal (GI) tract in terms of mass and length. Part 4 in this six-part series on the GI tract described the anatomy and function of the small intestine ([Bit.ly/NTGITract4](https://bit.ly/NTGITract4)). Part 5 describes the anatomy and functions of the large intestine, as well as common pathologies that affect both the small and large intestine.

Anatomy of the large intestine

The large intestine is approximately 1.5m long and comprises the caecum, colon, rectum, anal canal and anus (Fig 1). The structure of the large intestine is very similar to that of the small intestine (see part 4), except that its mucosa is completely devoid of villi.

Caecum and appendix

Chyme that has not been absorbed by the time it leaves the small intestine passes through the ileocaecal valve and enters the large intestine at the caecum. On receipt of the contents of the ileum, the caecum continues the absorption of water and salts.

The caecum is about 6cm long and extends downwards into the appendix, a winding tubular sac containing lymphoid tissue. The appendix is thought to be the vestige of a redundant organ; its narrow and twisted shape makes it an attractive site for the accumulation and multiplication of intestinal bacteria.

Colon

At its other end, the caecum seamlessly joins up with the colon, this is the longest portion of the large intestine (Fig 1). Food residue starts by travelling upwards through the ascending colon, located on the right side of the abdomen. The ascending colon bends near the liver at the right colic flexure (or hepatic flexure) and becomes the transverse colon, passing

Clinical Practice Systems of life

across to the left side of the abdomen. Just above the spleen at the left colic flexure (or splenic flexure), the transverse colon becomes the descending colon, which runs down the left side of the abdomen. Before the next bend, the descending colon transforms into the sigmoid colon.

The colon has a segmented appearance; its segments, which are caused by sacculations, are called haustra. The ascending colon, descending colon and rectum are located in the retroperitoneum (outside the peritoneal cavity). The transverse and sigmoid colon are attached to the posterior abdominal wall by the mesocolon.

Rectum, anal canal and anus

Distally, the large intestine opens into the rectum, which is continued by the anal canal. The rectum forms the final 20cm of the GI tract. It is continuous with the sigmoid colon and connects with the anal canal and anus (Fig 2, page 52). The rectum ends in an expanded section called the rectal ampulla, where faeces are stored before being released; the rectum is usually empty since faeces are not normally stored there for long.

The anal canal located in the perineum (outside the abdominopelvic cavity), is 3.8-5cm long and opens to the exterior of the body at the anus (Fig 2). It has two sphincters:

- Internal anal sphincter, which is controlled by involuntary muscles;
- External anal sphincter, which is made of skeletal muscle and is under voluntary control.

Except during defecation, both anal sphincters normally remain closed.

Functions of the large intestine

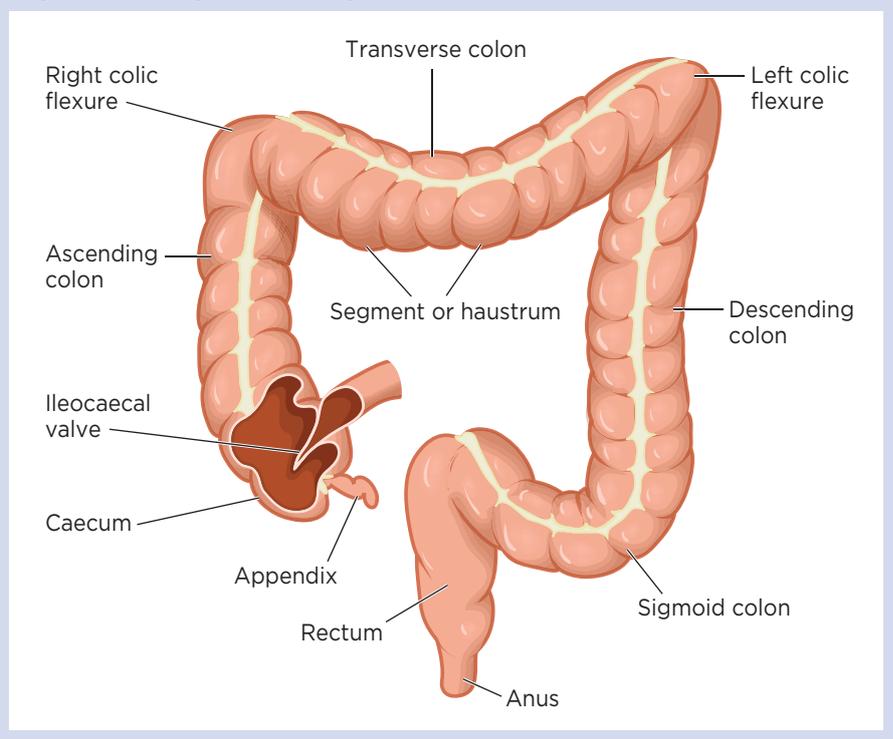
Meals pass from the small to the large intestine within 8-9 hours of ingestion. The small intestine will have absorbed about 90% of the ingested water. The large intestine absorbs most of the remaining water, a process that converts liquid chyme residue into semi-solid stools or faeces. The large intestine has three major functions:

- Absorption of water and electrolytes;
- Formation and transport of faeces;
- Chemical digestion by gut microbes.

Absorption of water and electrolytes

The presence of food residues in the colon stimulates haustral contractions, which occur approximately every 30 minutes and last about one minute each. With each contraction, each haustrum distends and contracts, pushing the food residues into the next haustrum. The contractions also mix the food residues, thereby facilitating the absorption of water.

Fig 1. Anatomy of the large intestine



The large intestine also absorbs electrolytes. Sodium ions are actively absorbed by the action of the sodium/potassium pump; this moves sodium and potassium ions in opposite directions across cell membranes, fostering sodium absorption and potassium loss by releasing the hormone aldosterone.

Antiperistaltic contractions move food residues back towards the ileocaecal valve, slowing transit down and giving more time to the large intestine to absorb water and electrolytes.

Formation and transport of faeces

Of every 500ml of food residue that enters the caecum each day, about 150ml become faeces. These contain mostly bacteria, old epithelial cells from the intestinal mucosa, inorganic waste, undigested food matter and fibre, as well as water to help it pass smoothly through the GI tract. They also contain small quantities of fats and proteins. Their characteristic brown colour is due to the presence of stercobilin and urobilin, breakdown products of haemoglobin from old red blood cells.

Since chyme residue lingers in the large intestine for 12-24 hours, most of the 1.5L of fluid entering the large intestine every day is absorbed, leaving less than 100ml to pass out in the faeces. This small quantity of fluid gives faeces their semi-solid consistency. Faeces are also softened by dietary fibre. Mucus, secreted by goblet cells lining the

entire colon, helps to bind dehydrated chyme and also lubricates the passage of faeces.

Transit in the colon is slow: it takes three days to clear 70% of a meal and complete expulsion of all remnants can take up to a week; transit is faster in men than women (Degen and Phillips, 1996). Normal bowel emptying patterns vary greatly between individuals, from three times per day to three times per week (Walter et al, 2010).

Colonic mass movements

Peristalsis in the ileum forces chyme into the caecum. Distention of the caecum triggers the gastric colic reflex and colonic mass movements begin. Stimulated by stomach distension and colonic irritation, mass movements usually occur three or four times a day, often during or immediately after meals. These strong waves, which can last up to 30 minutes, start midway through the transverse colon. Helped by haustral contractions, they push the now largely dehydrated contents along the colon toward the rectum. Fibre in the diet increases the power of colonic contractions propelling faeces towards the anus.

Colonic mass movements fill the rectum, creating an urge to defecate. It is important to act on this urge, as once the movements have passed, the urge also ceases. If the urge to defecate is ignored for an extended period of time, the rectum overfills, the large intestine absorbs more

Clinical Practice Systems of life

water and faeces become harder and drier. This can cause constipation.

Physiology of defecation

As faeces begin to fill the rectum, the rectal wall stretches, which sends an impulse to nervous centres in the spinal cord to initiate the spinal defecation reflex. This results in the relaxation of the internal anal sphincter, which allows a small quantity of faeces to pass into the anus. The anus detects whether the material is gaseous or solid and acts accordingly. If the material is solid, the external anal sphincter opens up and defecation takes place. However, the external anal sphincter is controlled by voluntary muscles, so it can be consciously restrained to delay defecation until a more convenient time. Children have usually learned this behaviour by the age of two or three years. People with severe dementia may no longer know how to do this.

Faeces are normally passed by contracting the rectal muscles, helped by a voluntary procedure called Valsalva's manoeuvre. This involves contracting the diaphragm and abdominal wall muscles, which increases intra-abdominal pressure and pushes faeces out of the rectum.

If the nerves between the external anal sphincter and the defecation centre in the medulla are damaged – as may be the case after a stroke, in multiple sclerosis, or after spinal injury – the ability to suppress defecation may be lost, resulting in faecal incontinence. Also, with ageing, the ability of the anus to detect whether it contains gas or faeces may become impaired and faecal matter may be treated as gas, causing faecal incontinence.

Chemical digestion by gut microbes

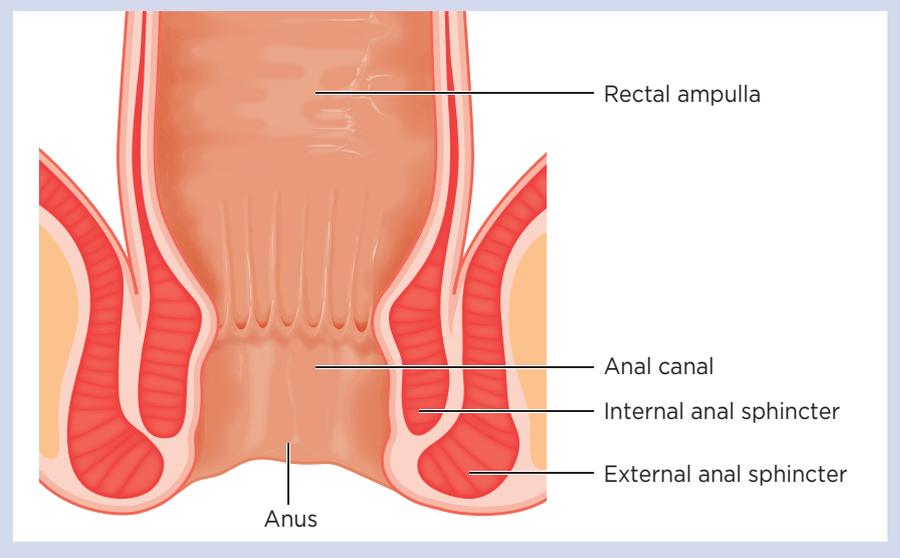
The large intestine does not secrete its own digestive enzymes: in this part of the GI tract, chemical digestion occurs exclusively through the action of millions of colonic bacteria. Through fermentation, these bacteria break down some of the remaining carbohydrates, which releases the hydrogen, carbon dioxide and methane that create flatus (gas). Colonic bacteria also protect the intestine from potentially harmful bacteria coming from the external environment and can synthesise certain vitamins. Their role will be more fully explored in part 6 of this series.

Disorders of the intestines

Lactose intolerance

People with lactose intolerance cannot digest dietary lactose. The undigested lactose ferments in the large intestine, producing gas,

Fig 2. Anatomy of the anal canal



abdominal cramps, bloating and diarrhoea. Symptoms range from mild discomfort to severe pain. One of the gases produced by the bacterial fermentation of lactose in the colon is hydrogen, so people who have lactose intolerance exhale hydrogen. The hydrogen breath test can be used to help diagnose the condition (Argnani et al, 2008).

Coeliac disease

Coeliac disease is an intolerance to gluten, a protein found in wheat, barley and rye. If people with coeliac disease eat gluten, intestinal immune cells (T cells) release inflammatory mediators that cause a flattening of the intestinal mucosal lining, impairing the ability to digest and absorb foods. Symptoms range from mild to severe and include diarrhoea, abdominal pain, bloating and flatulence, indigestion and constipation; in severe cases the condition can lead to malnutrition.

Diarrhoea

Diarrhoea (loose and watery stools) is most commonly caused by gastroenteritis, norovirus or food poisoning but can also be due to food intolerances or allergies, irritable bowel syndrome, inflammatory bowel disease, coeliac disease and diverticular disease.

If the intestines do not absorb fluids, the body can lose several litres of fluid per day, with consequences such as dehydration, loss of electrolytes (potassium and sodium ions) and increased risk of blood clotting. Large losses of potassium ions, for example, can cause cardiac arrest. The only absorption mechanism that is not disturbed by diarrhoea is glucose/sodium co-transport, which means people with diarrhoea can

increase absorption of essential sodium and water in the presence of glucose.

If a person has diarrhoea, it is essential to quickly replenish fluids and electrolytes by administering a solution containing the correct balance of glucose and electrolytes (for example, Dioralyte). Drinks such as lemonade or squash may not contain the correct balance.

If diarrhoea leads to acute hyponatraemia (serum sodium concentration <135mmol/L), this must be corrected promptly. Treatment may include the administration of hypertonic saline, but care must be taken to ensure that blood sodium levels are not allowed to increase too quickly, as this can cause a sudden shift of water in brain cells that may lead to the fatal complication central pontine myelinolysis (Rusoke-Dierich, 2018).

Constipation

Constipation is the infrequent and difficult or painful evacuation of faeces due to slow movement of hard, dry faeces. It may lead to abdominal distension and pain and, if left untreated, faecal impaction and GI obstruction. The condition can be due to irregular bowel habits, a diet low in fibre, and immobility. Certain medications, eating disorders and the overuse of laxatives may also cause – or compound – constipation. Including 20-60g of fibre/day in the diet and drinking one or two glasses of fluid with each meal may help to prevent constipation. Nurses need to bear in mind that constipation can indicate serious physiological disturbance or disease such as diverticulitis, obstruction due to a tumour or paralytic ileus.

Bowel obstruction

A tumour, adhesions in the intestinal walls, foreign bodies or impacted faeces may cause the intestines to become partially or completely blocked and intestinal contents to back up. This may result in abdominal swelling, pain, cramps, vomiting and severe constipation or diarrhoea. Another cause of bowel obstruction is paralytic ileus, a dramatic slowing of the normal peristaltic movement of the intestines. Paralytic ileus can be caused by bacterial or fungal infections, mesenteric ischaemia, appendicitis, abdominal surgery and certain medications.

Diverticulitis

Pea-size pouches called diverticula sometimes form on weakened spots of the intestinal walls as a result of increased pressure; for example, while straining during defecation. They are most common in the sigmoid colon, but both the location and the prevalence varies with age, for example in the US it is reported that the prevalence of diverticulosis was 35% in those younger than 50 years, 40% in individuals 50–59 years old, and 58% in those aged over 60 years (Peery et al, 2016). People who have diverticula but mild or no symptoms are said to have the benign condition diverticulosis. Complications can occur in about 20% of people with diverticulosis, who will develop diverticulitis – an inflammation and infection of the diverticula. This tends to occur when bacteria have built up in diverticula blocked by waste. Diverticular bleeding may occur, as well as chronic injury to the small blood vessels next to the diverticula and colonic obstruction.

Inflammatory bowel disease

Inflammatory bowel disease (IBD) is uncontrolled inflammation and bowel injury in the large intestine resulting in severe discomfort, with symptoms such as abdominal cramps, bloating, gas, liquid motions and diarrhoea. Often there is a severe urgency to defecate and there may be anal/rectal discharge or bleeding. Severe IBD may result in loss of appetite, loss of weight and iron deficiency anaemia.

The two main types of IBD are Crohn's disease and ulcerative colitis. While ulcerative colitis often manifests as continuous areas of inflammation and can usually be cured by removing the affected areas, Crohn's disease tends to cause a patchy distribution of inflamed ulceration that can affect any part of the GI tract, but most commonly the terminal ileum, or the colon, making treatment and surgery more difficult.

Crohn's disease

Until recently, Crohn's disease was thought to be an autoimmune disorder in which the immune system attacked the body's own gut lining. Today, there is evidence to suggest that the immune system overzealously attacks a microbial antigen on the gut lining (Torres et al, 2017). Most people with Crohn's disease are diagnosed before the age of 30. Intestinal obstruction, a common complication, arises from swelling and formation of scar tissue, a thickening of the bowel wall and a narrowed intestinal passage (strictures). Perforation of the bowel may occur as a result of an abscess or fistula.

Ulcerative colitis

Ulcerative colitis is characterised by inflammation and ulceration in the lining of colon and rectum, and rectal urgency that can result in painful, bloody diarrhoea up to 20 times a day. Symptoms may come and go but 5–10% patients have constant symptoms. Perforation is a potential complication, since chronic inflammation and ulceration may weaken the intestine wall to such an extent that a hole may form. This is generally linked with toxic megacolon, an emergency condition where the colon loses all contractile function and gas builds up. Perforation can result in life-threatening peritonitis.

Malabsorption syndrome

Malabsorption syndrome covers a number of disorders in which the small intestine is unable to absorb enough of certain nutrients (proteins, fats, minerals, vitamins and/or carbohydrates) and fluids, resulting in deficiencies, malnutrition and wasting. In patients who have had more than 50% of the small intestine removed, nutrient absorption will be severely compromised.

Appendicitis

If the appendix becomes blocked it becomes inflamed, causing appendicitis. Obstruction causes a pressure build-up, which may compress the blood supply to the gut wall, resulting in ischaemic injury and bacterial infection. The classical symptom is acute pain beginning at the umbilicus and spreading to the right iliac fossa. Nausea, vomiting and possibly fever may ensue. If this is not treated, the appendix may rupture, causing dangerous peritonitis and allowing bacterial infection to rapidly spread through the peritoneal cavity, potentially leading to death within hours. Appendicitis is one of the commonest causes of acute abdominal pain.

Colorectal cancer

Colorectal cancer is the second most common cause of cancer death in the UK (Bit.ly/CRUKMortality) and may be signalled by constipation or diarrhoea, cramping, abdominal pain and rectal bleeding – which may be either visible or hidden in the faeces (occult). Smoking, excessive alcohol consumption and a diet high in animal fat and proteins have been linked to an increased risk of colorectal cancer. Some studies suggest that increasing dietary fibre intake could reduce the risk (Yang and Yu, 2018).

Since most colon cancers arise from benign mucosal growths called polyps, prevention focuses on identifying these polyps. Screening for occult faecal blood is currently offered every two years to people aged 60–74 in most of the UK, although in Scotland it is offered from age 50 and Public Health England last year announced plans to extend its screening programme in stages to cover 50–74 years of age (PHE, 2018). **NT**

● Part 6 of our six-part series on the anatomy and physiology of the GI tract will describe the role of gut microbes in digestion, protection against invading pathogens, and vitamin synthesis.

References

- Argnani F et al (2008) Hydrogen breath test for the diagnosis of lactose intolerance, is the routine sugar load the best one? *World Journal of Gastroenterology*; 14: 40, 6204–6207.
- Degen LP, Phillips SF (1996) Variability of gastrointestinal transit in healthy women and men. *Gut*; 39: 2, 299–305.
- Peery AF et al (2016) Distribution and characteristics of colonic diverticula in a United States screening population. *Clinical Gastroenterology and Hepatology*; 14: 980–985.
- Public Health England (2018) Bowel screening to start at 50. Bit.ly/PHEBowelScreen
- Rusoke-Dierich O (2018) Damage caused by external factors. In: Rusoke-Dierich O. *Diving Medicine*. Cham: Springer.
- Torres J et al (2017) Crohn's disease. *Lancet*; 389 (10080): 1741–1755.
- Walter SA et al (2010) Assessment of normal bowel habits in the general adult population: the Popcol study. *Scandinavian Journal of Gastroenterology*; 45: 5, 556–566.
- Yang J, Yu J (2018) The association of diet, gut microbiota and colorectal cancer: what we eat may imply what we get. *Protein Cell*; 9: 5, 474–487.

CLINICAL SERIES **Gastrointestinal tract series**

Part 1: The mouth and oesophagus	Jun
Bit.ly/NTGITract1	
Part 2: The stomach	Jul
Bit.ly/NTGITract2	
Part 3: The duodenum, liver and pancreas	Aug
Bit.ly/NTGITract3	
Part 4: The jejunum and ileum	Sep
Bit.ly/NTGITract4	
Part 5: The large intestine	Oct
Part 6: Gut microbes	Nov