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Physiology of lower gastrointestinal tract

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Summary

Background: The lower gastrointestinal (GI) tract, formed from the midgut and hindgut, encompasses the colon, rectum and anal canal.

Aim: The aim of this review is to provide an overview of the anatomy and physiology of the lower GI tract.

Methods: Literature review on anatomy and physiology of the lower GI tract, including normal motility and phases of defecation. It derives its blood supply from the superior and inferior mesenteric arteries while it is innervated by the extrinsic autonomic (the thoracolumbar and sacral nerves) and the intrinsic enteric nervous system. The colon has four layers: mucosa, submucosa, muscularis externa and serosa. The anal canal ends in the internal and external anal sphincters (EASs) involved in continence and defecation. The lower GI tract is predominantly involved in digestion, absorption, defecation and protection. Defecation is a complex process that requires inter-neural (enteric and autonomic nervous systems), neurohormonal and neuromuscular coordination. It has four phases which include basal, pre-expulsive, expulsive and end phase. High-propagating contractions in the colon propel stool to the rectum leading to rectal distention and the recruitment of the recto-anal inhibitory reflex. Once able, the EAS, under full conscious control, is then relaxed allowing stool to be evacuated. Other defecation reflexes include the gastrocolic, gastroileal and coloanal reflexes.

Conclusions: Recent advances provide novel techniques to investigate motility patterns including high-resolution manometry protocols with automated assessments, magnetic resonance imaging techniques for defecography, wireless motility capsules and fecobionics.

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AUTHOR CONTRIBUTIONS

Neha R. Santucci: Conceptualization; data curation; methodology; supervision; writing - original draft. **Alejandro Velez:** Conceptualization; data curation; methodology; writing - original draft.

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1 | INTRODUCTION

The lower gastrointestinal (GI) tract, encompassing the colon, rectum and anal canal, is predominantly involved in digestion, absorption, defecation and protection.[1] These functions require a morphologically intact anatomy and numerous complex physiologic systems to work in synergy. These require inter-neural (enteric and autonomic nervous systems), neurohormonal and neuromuscular coordination.[2] Intrinsic microbial as well as external gut-brain interactions affect the functioning of the lower GI tract. Defecation involves excretion of waste from the body. Continence requires the voluntary control of defecation. Disorders of continence such as constipation, retentive and non-retentive faecal incontinence are often seen in infants and children and need better investigational and management strategies. This review covers the anatomy and physiology of the lower GI tract with a focus on novel techniques to investigate colonic and anorectal physiology including high-resolution manometry testing with new protocols for automated assessments, magnetic resonance imaging (MRI) techniques for defecography, wireless motility capsule and fecobionics.

1.1 | Embryology of the colon and anorectum

The midgut forms the caecum to two-thirds of transverse colon while the hindgut contributes to the distal third of the transverse colon, descending colon, sigmoid colon, rectum and superior part of the anal canal.[3,4] The anal membrane ruptures at the end of the 8th week, allowing communication between the amniotic cavity and the distal digestive tract (Figure 1).

The colonic midgut develops from gestational weeks 6–10.[3] It herniates through the umbilical opening outside of the abdominal cavity, then rotates 90° counterclockwise around the superior mesenteric artery (SMA) axis. Thus, the caudal midgut stays to the left. After physiologic reduction of the umbilical herniation, the large intestine has an additional 180° counterclockwise rotation, returns to the abdominal cavity and has a fixed position. The ascending colon mesentery fuses with the parietal peritoneum on the posterior abdominal wall and disappears, thus, leaving the ascending colon retroperitoneal. The caecal diverticulum appears in the 6th week and develops into the caecum and appendix. The appendix is initially in the caudal midgut loop but becomes the distal end of caecum at birth. The unequal caecal growth leaves the appendix medial to the caecum. However, there is high variability in the location of the appendix. It may be retrocecal (65%–70%), pelvic (25%–30%) and pre- or post-ileal (5%).[5]

The hindgut develops from 6 to 12 weeks.[6] The descending colon mesentery fuses with the left posterior abdominal wall peritoneum and becomes retroperitoneal. The cloaca (terminal part of hindgut) is divided by the urorectal septum into the urogenital sinus and rectum during the 6th week of gestation. The cloacal membrane is composed of the endoderm of the cloaca and ectoderm of the proctodeum (anal pit). By the 7th week, the urorectal septum fuses with the cloacal membrane, giving rise to anal and urogenital membranes, which later form the perineal body in adults. The cloacal sphincter is divided by the urorectal septum to give rise to the anterior and posterior parts. The anterior part forms the superficial transverse perineal, bulbospongiosus and ischiocavernosus muscles while the posterior part forms the

external anal sphincter (EAS). At the end of the 8th week, anal membrane ruptures leading to communication of the anal canal with the amniotic cavity.

2 | ANATOMY OF THE LOWER GI TRACT

2.1 | Normal anatomy of the colon

The colon extends from caecum to rectum and its total length is 3–5 feet (Figure 2).[1] The caecum is the widest and thinnest part of the colon.[1,7] The ascending colon is fixed to the retroperitoneum.[1,7] The transverse colon extends from the hepatic flexure until the splenic flexure and is intraperitoneal and mobile.[1,7] It is attached to the colonic mesentery and the gastrocolic ligament.[1,7] The greater omentum is attached to the superior anterior edge.[7] The descending colon starts at the splenic flexure and is fixed to the retroperitoneum.[7] The lineocolic ligament attaches the spleen to the splenic flexure.[6] The sigmoid colon is the narrowest portion of the colon and remains very mobile. The five layers of colon and rectal wall include the mucosa, submucosa, inner circular muscle, outer longitudinal muscle and serosa.[1] The colonic outer longitudinal muscle is separated into three taeniae coli that converge at the appendix and rectum. The intraperitoneal colon and the proximal one-third of the rectum are covered by serosa.

2.2 | Normal anatomy of the anorectum

The sigmoid colon, the last part of the colon, transitions into the rectum (Figure 3).[1,7] The normal adult rectum is, on average, approximately 12–15 cm in length.[7] At the recto-sigmoid junction, the rectum expands in volume and it reaches its peak volume at a point known as the rectal ampulla, then tapers back down eventually joining the anus.[7] The anorectal junction, the transition point from between the rectum and the anus, is located 1 in (2–3 cm) anterior to the coccyx and, in males, at the level of the prostate.[7] The rectum is devoid of typical colonic anatomic hallmark features—taenia, haustra and plica semilunaris—and does not receive mesenteric projections.[1,7] The taenia originating from the sigmoid converge and fuse shortly above the recto-sigmoid junction and form a continuous longitudinal muscle layer that spans the full rectum.[7] Additionally, the rectum has been noted to have three semilunar transverse folds, termed the *valves of Houston,* that consist of mucosa and some longitudinal muscle involvement; the total number differs and the composition is different based on the location of these folds.[7] There is no known function for these structures; however, it has been postulated that it may play a role in the characterisation of luminal contents (e.g. solids vs. gas).[7]

The anal canal is an average of 2–5 cm long in male adults (shorter in female adults) exiting the body as the anal orifice.[1,7] The anal orifice can be identified as a circumferential area of skin pigmentation and puckering that is formed by the underlying convergence of longitudinal muscle fibres.[7] The anal canal, while small, represents a structure rich in muscular structures (sphincters), vessels and dense neuronal networks. Its attachment to nearby structures is important, as it allows the correct orientation and anchor points to provide a zone of high pressure that accommodates the process of defecation. On the anterior part of the anal canal, the middle portion attaches to the perineal body.[7] Laterally and posteriorly, the canal is devoid of true attachments and this area, full of fatty and

loose connective tissue, acts as reservoir for the canal's expansion during relaxation.[7] On the posterior plane, the anal canal attaches to the coccyx via the anococcygeal ligament—a complicated structure containing a combination of muscle fibres and whose relationship with pelvic floor muscles deserves its own detailing elsewhere.[7]

The anal sphincter complex makes up the bulk, if not the entirety, of the anal canal (Figure 3). The complex is made up of the EAS, the internal anal sphincter (IAS) and a conjoint longitudinal muscle coat.[1,7] The terminal part of the inner circular muscle layer of the large intestine becomes thickened and forms the internal sphincter.[7] The IAS spans most of the anal canal terminating just proximal to the anal orifice and underlying the inter-sphincteric groove. The outer longitudinal smooth muscle layers of the rectum continue into the anal canal and are termed the conjoint longitudinal muscle layer that descends among the IAS and EAS contributing to the general structure and stability of the complex.[7] Unrelated to the muscular layer of the rectum, the EAS contributes most of the bulk and tone to the sphincter complex.[7] It is composed of type 1 slow twitch fibres, and it encircles the entirety of the anal canal. Its attachments include the anococcygeal ligament, the perineal body and the levator ani muscle.[7]

At rest, the IAS is tonically contracted and as part of the recto-anal inhibitory reflex relaxes in response to luminal distention allowing the rectal contents to proceed into the anal canal proper.[1,7] As the luminal contents near the pectinate line, the middle third of the anal canal, they encounter its sensing mechanism and are held here through continued IAS tone and a larger contribution of EAS tone.[1,7] Likewise, the EAS is tonically contracted at rest, but unlike the IAS, it is under complete conscious control.[1,7]

2.3 | Vascular supply and lymphatic drainage of the colon and anorectum

The vascular supply of the colon is highly variable. In general, the SMA supplies the midgut while the hindgut receives its blood supply through the inferior mesenteric artery (IMA, Figure 4).[1,7] The ileocolic branch of SMA supplies TI and the proximal ascending colon.[1,7] The right colic artery supplies the ascending colon, middle colic supplies the transverse colon while the left colic arterial branch of IMA supplies the descending colon.[1,7] The sigmoidal branches supply the sigmoid and the superior rectal artery supplies the proximal rectum.[1,7]

The anal canal is supplied by the terminal branches of the superior and inferior anorectal arteries with a degree of contribution from the median sacral artery.[7] The venous drainage is achieved via the superior and inferior mesenteric veins with additional contributions from the internal iliac veins.[1,7] Lymphatic drainage of the proximal anal canal occurs through the submucosal and intramural lymphatics, while the distal canal is and the EAS drains into the inguinal nodes.[1,7]

2.4 | Neural regulation of the lower GIT

2.4.1 | Innervation of the colon: The nerve supply of the colon comprises the extrinsic autonomic nervous system (Figure 5) and the intrinsic enteric nervous system (Figure 6).[1,7] The sympathetic supply through T6–T12 and L1–L3 leads to tonic inhibition of the

non-sphincteric colonic muscle. This prevents epithelial secretion and decreases splanchnic blood flow. However, it is excitatory to sphincter muscles (especially ileocecal junction and IAS). Norepinephrine is the main neurotransmitter. Preganglionic sympathetic fibres are from lateral horns of T1–T12 and L1–L2 or L3–L4.

Contrarily, the parasympathetic supply includes the posterior division of the vagus nerve to the right and transverse colon and sacral nerves S2–S4 to the left/distal colon. It is predominantly stimulatory. Preganglionic parasympathetics travel superiorly and laterally deep to the peritoneum to join the inferior hypogastric plexus that supplies pelvic organs and proximally up to the splenic flexure. These synapses at ganglia form the myenteric and Auerbach plexuses in the bowel wall.

2.4.2 | Innervation of the anorectum: The IAS and the conjoint longitudinal muscle are supplied by terminal branches of the superior rectal and inferior anorectal vessels and innervated extrinsically by autonomic nerves (Figure 5).[1,7] Specifically, sympathetic L1 and L2 originating fibres and parasympathetic S2 to S4 originating fibres travelling within the inferior hypogastric plexus.[7] Relaxation of the IAS (and contraction of the conjoint longitudinal fibres) is accomplished by stimulation of cholinergic muscarinic and nitrergic receptors leading to the net movement of lumen contents antegrade.[7] Contraction of the IAS and the longitudinal muscle fibres is accomplished by sympathetic stimulation of the alpha-adrenergic receptors.[7] The EAS receives its blood supply from the inferior anorectal vessels, and it is innervated by the inferior anal nerve, a branch of the pudendal nerve.[7]

2.5 | Neurotransmitters involved in the colon and anorectum

Acetylcholine is the primary parasympathetic secretomotor transmitter while noradrenaline mediates the sympathetic input.[8,9] The other autonomic neurotransmitters include vasoactive intestinal peptide (VIP), serotonin and somatostatin. The excitatory enteric neurotransmitters are acetylcholine, tachykinins and enkephalins while nitric oxide and VIP mediate the inhibitory enteric nervous supply.

2.6 | Gut-brain neuronal interactions

The chemo- and mechanoreceptors in the gut wall get stimulated in response to distension and other substrates. These send signals via local afferents to the enteric nervous and through splanchnic and vagal afferents to the CNS. The myenteric and submucosal plexuses in turn send signals via local efferents and CNS via parasympathetic and sympathetic efferents to the gut muscle wall to contract. They also stimulate endocrine cells, secretory cells and blood vessels to facilitate digestion. The feedback loop is regulated by interneuronal connections between the extrinsic and enteric nervous systems (Figure 7).

3 | MICROANATOMY OF THE LOWER GI TRACT

3.1 | Normal microanatomy of the colon

The colon has four layers: mucosa, submucosa, muscularis externa and serosa (Figure 8A–C).

The mucosa of the colon is formed by epithelium, lamina propria and muscularis mucosae. The mucosal thickness increases as it goes from the caecum to the rectum and contains numerous straight tubular glands or crypts of Lieberkühn. The surface epithelium is columnar and does not contain villi. There are absorptive cells at the luminal surface and mucous-secreting goblet cells at the base of the glands. The number of goblet cells is increased compared to colonocytes and increases in number distally. Goblet cell depletion is seen in chronic inflammatory states. Absorptive cells have shorter and fewer microvilli than those in the small intestine. They lack brush border enzymes and do not have any role in digestion. Their main function is absorption of electrolytes and water. The crypts are longer and straighter than those in the small intestine. There are fewer enteroendocrine cells in the colon. Hence, special immunohistological stains are required for proper visualisation under the microscope. Paneth cells are seen scattered in the caecum.

The muscularis mucosa includes several thin layers of smooth fibres oriented in different directions. The layers are penetrated by nerves from the submucosa plexus, which extend vertically into the lamina propria.

The lamina propria is highly cellular, containing lymphocytes, plasma cells, macrophages and scattered eosinophils. Lymphoid follicles are present in the colonic mucosa and may extend through the muscularis mucosae into the submucosa (part of gut-associated lymphoid tissues). Neutrophils are seen occasionally in lamina propria. However, their presence in excess in the surface epithelium or crypts may indicate an active infection or inflammation.

The submucosa is composed of loose connective tissue, with collagen and elastic fibres that connect the mucosa and the muscularis externa layers closely together. It contains blood vessels, lymphatics and specialised nerve ganglions of the Meissner plexus. However, it does not contain any glands.

The muscularis externa has a thin smooth muscle layer with inner circular and outer longitudinal smooth muscle layers. The outer layer is condensed into three longitudinal muscular bands called taeniae coli (seen throughout the colon except the rectum). Contraction of the taeniae coli and circular muscle layer draws the colon into sacculations called haustra, which is characteristic of the colon. The muscularis externa is responsible for the propulsive force of the large intestine. Colonic peristalsis is mediated by the intrinsic myenteric plexus and the extrinsic autonomic neural control. The Meissner plexus resides at the base of the submucosa and consists of two neural networks. The Auerbach plexus lies between the inner circular and the outer longitudinal muscle layers (Figure 8B).

The serosa forms the outermost layer of the colon. It consists of a single layer of avascular, flat, nucleated cells and simple squamous epithelial cells. It lubricates the colon by producing serous fluid.

The appendix is different in some respects from other parts of the colon.[10] The appendix contains thin, finger-like extensions of the caecum characterised by long crypts without villi. The lamina propria and the submucosa of the appendix contain a large amount of lymphoid tissue. Thus, local inflammation can lead to hyperplasia of these lymphoid follicles, causing obstruction of the lumen, bacterial proliferation and subsequent appendicitis. Appendices

epiploicae are lobulated masses of pericolic adipose fat that protrude from the serosal surface of the colon. They are usually 2–5 cm long. They can undergo spontaneous torsion, leading to infarction, which can produce symptoms similar to appendicitis.

3.2 | Normal microanatomy of the anorectum

The lining of the anus is a continuation of rectal columnar epithelium (Figure 9). About halfway through the anal canal, a set of distinctive columns become apparent, termed the anal columns (of Morgagni) that contain a rich network of vessels.[1,7] The columns fuse and meet at the pectinate line where the lining changes from columnar to a tan, non-keratinised stratified squamous epithelium with somatic nerve endings.[7] The centimetre or two proximal to the pectinate line, known as the anal pecten or transition zone, is an anatomically and physiologically distinct area of great importance. This highly specialised region bears a concentration of thermoreceptors, which along with other somatic nerve endings creates a ‘sampling mechanism’ that is crucial in the characterisation of luminal contents prior to defecation.[7] This quickly transitions to the lower anal mucosa and the inter-sphincteric groove lined by keratinised stratified epithelium and indistinguishable from perianal skin.[7]

The rectum does not have mesentery.[7] It is divided into two segments: upper part (rectum proper) and lower part (anal canal), which extends from the anorectal junction to the anus.[1] The rectal mucosa is thicker than other parts of colon, with more prominent veins. It has longer crypts than those in the small intestine and is lined predominantly by goblet cells.[1] The crypts gradually disappear at the level of the anal canal.[1]

4 | PHYSIOLOGY OF THE LOWER GI TRACT

4.1 | Normal physiology of the colon

The primary functions of the colon include absorption of water and electrolytes, mucous secretion, faecal material formation, propulsion and storage and residence of microbiota (Figure 10).[1]

4.1.1 | Absorption: Initial ingested food reaches the caecum in approximately 4h (usually completely by 8–9 h). The colon absorbs up to 5 L of water/day. It removes 90% of fluid from the digested food bolus. Around 1–2 L isotonic chyme that enters the colon is converted to 200–250 mL semisolid faeces. The colon absorbs Na+ against gradient via Na+–K ATP pump (adenosine triphosphate) and can absorb up to 400mEq of Na+/day. With volume depletion, aldosterone increases Na+ conductance in the colon. Potassium (K+) is absorbed by active secretion into lumen and passive absorption. Chloride (CI−) is absorbed by active transport through exchange via HC03-CI exchanger. NH3 forms due to bacteria-degrading protein and urea absorbed (influenced by lumen pH) and then transported to liver.[11]

4.1.2 | Secretion: The colon predominantly secretes mucin, which lubricates the lumen and forms a barrier to microbes and other pathogens.[12]

4.1.3 | Microbiota: Microbiota compose 30% of faecal dry weight. There are 1011–1012 bacteria/g faeces. They mainly consist of anaerobes, especially Bacteroides. *Escherichia*

coli is the most common aerobe.[13] Microbial fermentation of carbohydrates leads to the release of short-chain fatty acids (SCFAs), namely acetate, butyrate and propionate.[14] Butyrate is an important energy source for colonocytes and maintenance of a healthy epithelium. SCFAs facilitate water absorption by non-ionic diffusion. Metabolism of SCFAs by colonocytes provides energy for active sodium (Na+) transport. Endogenous bacteria are also important for bilirubin, bile acids, oestrogen and cholesterol metabolism.[15] They break down carbohydrates and proteins in the colon and produce vitamin K. Based on dietary intake and composition, an average of 400–1200 mL/day of flatus is produced in the colon. The development of immune system is dependent upon colonisation of gut by commensal bacteria by regulating differentiation of T helper cell lineage.[16] For example, *Bacteroides fragilis* colonisation is involved in balance of TH1 and TH2 and differentiation in the gut of regulatory T cells. Development of T regulatory cells in the intestinal immune system is largely determined by bacterial colonisation of gut mucosa. Naive T cells differentiate into colonic TH17 cells by bacteria-derived adenosine triphosphate (ATP). CD70+ dendritic cells expressing P2X and P2Y ATP-sensing receptors induce this differentiation of naive T cells into TH17 cells.

4.2 | Normal physiology of the anorectum

The main goal of the anorectum is to act as a reservoir for stool, with a complex sensing mechanism, and to allow for the complete evacuation of stool through the act of defecation.[1,7] The process of defecation is a highly orchestrated and dynamic operation involving the colonic propulsive force, the coordinated relaxation and contraction of muscles within the anorectum, appropriate timing of IAS and EAS relaxation and the ability of the pelvic diaphragm to place these structures in the optimal emptying position.[1,2,7] Additionally, because of its partial conscious control, it is highly susceptible to environmental, cultural, psychological and psychiatric influences.[2] Especially in children, social expectations, temperament, self-efficacy and developmentally appropriate behaviours such as limit pushing and defiance affect defecation.[17,18] Thus, this learning process of achieving stool continence is the most common time for a child to develop constipation.

4.3 | Normal motility of the colon and the anorectum

The colon is rarely inactive. It has segmental contractions throughout the day which can be isolated or in bursts and rhythmic or arrhythmic. Their main function is to mix the contents of the colon and thus facilitate absorption. These contractions maximise the intestinal mucosa exposure to luminal contents and lead to absorption of water, electrolytes and bacterial by-products (SCFAs).

In adults, the average normal transit time of colonic contents (colonic transit time) is 24 h (ranging from 4 to 50 h).[19] Mass action contraction, which is the colonic contraction that moves contents in a net antegrade fashion, occurs at a rate of approximately 1 cm/h. Despite its name, there has been research using a wireless magnetic motility capsule to indicate that the movement is segmented and often back and forth.[20,21] These mass action contractions are likely a combination of well-established motor patterns such as high-amplitude propagating contractions (HAPCs), low-amplitude propagating contractions

(LAPCs), cyclic motor patterns and colonic pressurisations. These will be discussed in detail in the *Defecation* section below.

Outside of defecation, there are several important reflexes that have been described in the literature and play an important role in the motility of the colon. The following reflexes are pictured in Figure 11 and described in detail below:

a. *Gastrocolic reflex:* This reflex is described as an increase in colonic motility in response to gastric wall distention. Following the consumption of a meal, the number of LAPCs and HAPCs within the colon can be seen in increased frequency within minutes.[22–24] This reflex involves bi-directional communication between the autonomic and the enteric nervous system, as well as numerous neuropeptides such as serotonin, gastrin, cholecystokinin and prostaglandin E1.[25,26] This reflex is often reported on during modern colonic manometry and has been associated with irritable bowel disease process when absent or abnormal.[23,27]

b. *Gastroileal reflex:* Along with the gastrocolic reflex, this reflex works to prepare the GI system to receive stomach contents. The ileocecal valve will be stimulated to relax and adopt a more pliable form in response to stomach distention. This reflex is mediated by similar factors as the gastrocolic reflex.

c. *Coloanal reflex:* The coloanal reflex is intimately associated with the pre-expulsive phase of defecation (described below) and is defined as the relaxation of the anal sphincter complex in response to colonic antegrade contraction activity.[28] Interestingly, this reflex is stimulated by a number of colonic contractile patterns and may originate from any portion of the colon including the proximal portion. These patterns can be full HAPCs (Figure 12) associated with mass action contraction and propulsion of solid contents to simultaneous pressure wave (or pan colonic pressurisations) more associated with forward movement of gas and liquid intraluminal contents.[29–31] In contrast to the recto-anal inhibitory reflex (RAIR) that is associated with proximal IAS relaxation, the coloanal reflex can lead to the relaxation of both anal sphincters.[28,29,31]

4.4 | Defecation—The culmination of normal lower GI motility

Normal defecation begins with the delivery of intraluminal contents through the colonic high-amplitude propagated contractions.[2] As the rectum fills, the wall distention is sensed via afferent fibres that contribute to the urge to defecate.[2] As the rectum continues to fill, the IAS relaxes and allows for antegrade movement of intraluminal contents.[2] This reflex is known as the RAIR which is mediated directly by enteric nervous system and independent of a spinal reflex pathway.[2] IAS relaxation allows for additional anal canal accommodation depending on the consistency of the intraluminal contents that are undergoing periodic sampling.[2] At this point, the rectum will act primarily as a reservoir as the continuation of the defecation process is dependent on conscious and voluntary processes.[2] If defecation is not to occur at this time, the pubic diaphragm and the EAS will hypercontract to prevent accidental incontinence and to move stool away from the distal rectum.[2] Once appropriate, as deemed by behavioural/cultural/environmental cues, the defecation process

will continue.[2] A combination of colonic HAPCs, recto-anal conjoint longitudinal muscle contractions (that shortens and opens the canal, and flattens the anal cushions), intra-abdominal increase in pressure from bearing down, along with the relaxation of the pelvic floor and the anal sphincter complex lead to the successful evacuation of intraluminal contents.[2] Following defecation, the closing reflex leads to the contraction of the EAS and reactivation of the IAS as the recto-anal distention is relieved.[2]

4.4.1 | Four phases of defecation

Basal phase: This phase is known as the non-defecatory state. In this phase, the colon is performing vital homeostatic functions as described above and is noted to have non-propagating activity (Figure 13).[2] This activity is often referred to as the *cyclic motor pattern* and is described as 'rhythmic, propagating contractions' that occur over short distances at a high frequency (2–8 per minute; Figure 14).[32] This activity has been noted to be active by a meal and HAPCs, and to occur during sleep, under anaesthetics and following sacral nerve stimulation.[2] The rectum is largely devoid of contents during this phase. It is kept this way by the intraluminal recto-anal pressure gradient in which the anal canal intraluminal resting pressure exceeds that of the rectal resting pressure.[2] There has also been some recent research to suggest the presence of a 'recto-sigmoid brake' that also contributes to keeping the rectum empty.[33] The *recto-sigmoid brake* is a newly described phenomenon but it has been suggested that it actually represents a primarily retrograde distal cyclic motor-type pattern that is predominantly occurring during the basal phase of defecation with the primary purpose to limit rectal filling.[33]

Pre-expulsive phase: This phase becomes apparent about 1 h prior to defecation.[2] Through a series of experiments, studies have shown that there is increased non-propagating to segmentally propagating contractions with a small overall net antegrade movement of luminal contents towards the rectum.[19,34] At this point, the rectal-sigmoid brake is likely inhibited or dampened to allow for rectal filling and its distention initiates the anorectal sensorimotor activity.[2] As the rectum distends, mechanoreceptors send efferent signals to the sacral parasympathetic neurons that terminate in the spinal cord.[2] There appears to be a threshold in the degree of rectal distention that stimulates conscious perception. This is noted during the sensation portion of most anorectal manometry (ARM) protocols. According to two studies, this conscious perception might be tied to the initiation of rectal contractions.[35,36] Continued distention of the rectum eventually meets another threshold that involves the relaxation of the IAS and the contraction of the EAS which is the RAIR.[2] As the IAS relaxes and the anal canal fills, the intraluminal pressures within the anus and rectum equalise and allow for further antegrade movement of contents.[2] During this phase in defecation, the *sampling reflex* is allowed to occur and, through pelvic parasympathetic neurons and spinothalamic relays with the brainstem and cortex, sensory discrimination of the characteristics of luminal contents occurs.[2] Sophisticated central nervous system interactions with the rectal-anal complex take place and through a feedback loop type of mechanism either defer the next phase of defecation and return the system to *basal phase* (through conscious EAS contraction and subconscious retrograde rectal contractions that reverse the progress of intraluminal contents) or allow the *expulsive phase* to occur.[37,38]

Expulsive phase: As the pre-expulsive phase moves closer to the expulsion phase, the amplitude and frequency of antegrade propagating contractions (APC) increase and originate from subsequently more distal areas within the colon.[34] Two interesting observations have been made over the years. Colonic contractions during this phase are consciously sensed and antegrade movement of intraluminal contents can occur in the absence of measured HAPCs.[34] The intraluminal pressure of the distal colon is observed to increase during this phase and the cyclic motor pattern is absent. Both of these mechanisms are likely to encourage rectal filling.[31,39] It is not until the expulsive phase that the recto-sigmoid pressure gradient is noted to be higher than that of the anal canal pressure. This reversal in pressure gradients occurs through voluntary relaxation of the EAS and increase in intraabdominal pressure (via Valsalva), the modification of the anorectal angle to a more oblique angle (110–155°; through the active process of squatting, the relaxation of the puborectalis complex and the contraction of the levator plate muscles) and the shortening and dilation of the anal canal (through the contractions of the conjoint longitudinal muscle of the anus (CLMA) and the contraction of the pubococcygeus).[2,40–42] The above observations have been made due to diversification of functional assessment tools such as the balloon expulsion test and ‘push’ manoeuvres during ARM, the use of defecography studies and the advent of fecobionics and artificial stools that will be detailed further below.

End phase: Through a yet-to-be-fully elucidated process, the defecation process moves into its final phase known as the end phase. After evacuation of rectal contents is complete, the *closing reflex* is elicited likely by the decreasing distention and traction forces upon the anal canal complex.[43] The *closing reflex* reverses the expulsive phase by contracting the IAS, returning the pelvic floor musculature and the anorectal angle to its resting position (65–108°) and allowing the elongation of the anal canal through the relaxation of the CLMA.[2] It is theorised that colonic motility likely shifts back to its homeostatic pattern of motility. However, no dedicated studies have been done on post-defecation motility.[2]

4.4.2 | Technical advances in the study of defecation

Anorectal manometry: ARM has been a critical tool in the study of human defecation. The past two decades have noted an abundance in its utility for the characterisation and diagnosis of defecatory disorders. With high-resolution sensors and 3D visualisation, several minor details of this complex process have been clarified and enhanced our understanding of continence and faecal evacuation. While the protocols used and the quality of testing vary significantly, a recent review of the current uses, protocols and interpretation recommendations will hopefully lead to more uniform and standardised use.[44] The use of the rectal balloon expulsion test, for example, has been adopted most recently and acts as an easy, cheap way to functionally characterise evacuation and may help predict response to pelvic floor physical therapy, a crucial treatment modality for defecation disorders.[45,46] The London classification is another recent breakthrough that has elevated the utility of ARM to the gold standard for diagnosis of anorectal and defecation disorders. The London classification requires the use of the International Anorectal Physiology Working Group (IAPWG) protocol to populate the four classifications of disorders: disorder of RAIR, disorders of anal tone and contractility, disorders of recto-anal coordination and disorders

of rectal sensation.[47,48] The IAPWG protocol and the London classification of defecatory disorders are detailed in Tables 1 and 2 respectively.

Defecography: The evaluation of defecation disorders often requires a thorough assessment of the rectal, anal and pelvic floor anatomy. While traditional contrast, two-dimensional and cross-sectional imaging is often used as first line to identify anatomical and functional issues in these areas, more sophisticated imaging modalities have become available. Defecography is the umbrella term for radiological exams aimed at the evaluation of the process of defecation and allows for the detailed determination of anatomical and functional abnormalities and variants to help explain or categorise a possible defecation disorder. Aside from providing an anatomical survey, the most useful parameters that can be gleaned from these studies include anorectal angle, anal diameter, degree of rectal emptying and pelvic floor descent.[49] Below is a description of the most used defecography studies, their most common indication and usage and their limitations:

4.4.3 | Barium defecography

Definition: Also known as ‘evacuation proctography’ or ‘conventional’ defecography is a real-time fluoroscopic exam that harnesses the use of X-ray images and barium to assess the anatomy and function of the anorectum and pelvic floor.[50]

Examination: While protocols may vary by institution, it often requires an anticipatory bowel prep and the oral consumption of thin barium 1 h prior to examination. This thin barium is used to opacify the small bowel and help contrast it against the areas of interest. The patient is then given an enema of thick barium paste, and a scout film is taken to assess the anatomy at rest. While seated on a radiolucent commode, the patient is asked to squeeze, strain and defecate while multiple cine clips are taken.

Indication/Use:

- Concern for anatomical abnormalities (previous pelvic/anorectal surgeries, vaginal birth, trauma, etc.).
- Equivocal ARM.
- Dysfunction resistant to pelvic floor physical therapy.

Limitations: Patient discomfort and unnatural/artificial ‘exam’ environment can lead to false positives. Other include lack of accepted testing standards, protocols and interpretation guidelines. Lack of soft tissue enhancement/characterisation limits pelvic floor musculature evaluation.

4.4.4 | Magnetic resonance defecography

Definition: MR defecography, or MRD, is a cross-sectional and multiplanar radiological examination designed to evaluate the anatomy and function of the anorectum and pelvic floor without ionising radiation.[51,52]

Examination: No bowel prep is required and no additional contrast is used (e.g. intravenous and enteral). A distention medium such as ultrasound gel, a potato starch-gadolinium mixture, rectal gel or vaginal gel is instilled into the rectum prior to examination. Positioning during the study varies based on the configuration of the MRI machine. Usually, the patient is supine with the knees bent in a closed machine. If available, an open machine may allow for more physiological positioning. However, due to its scarcity, most protocols are developed for supine positioning in a closed machine. Static images allow for the best resolution needed for the anatomical survey while dynamic sequences are used for defecation-related manoeuvres such as pushing, straining, squeezing and defecation.

Indication/Use:

- Excellent in identifying multicompartment pathology due to its multiplanar capabilities and soft tissue identification.
- Concern for posterior compartment pathologies.
- Functional disorders such as recto-sphincteric dyssynergia.

Limitations: Unphysiological positioning, patient discomfort and unnatural/artificial ‘exam’ environment can lead to misdiagnosis of functional defecation disorders in otherwise normal patients. While the acquisition time is not as long as other MR-based studies, the exam can take longer than a barium/XR-based exam. Availability of machines, trained study personnel and radiologist interpretation experience are the MRD’s most important limitations.

Fecobionics[53]: With the advent of ARM and the balloon expulsion test, the functional examination of defecation is getting closer to the goal of simulating the distensile and tractional forces of stool. The technology behind the fecobionic devices hopes to take the next step in combining the physical properties of real stool and the measurement capabilities of manometric studies. The manufacturers of the device claim that this ‘biomechatronic’ tool can geometrically map the rectum and anus. This provides high-resolution pressure signatures of the defecation process using materials that simulate the consistency and shape of normal stool. The wired device is composed of a soft silicone resin-encased core with a pair of motion processing units (for orientation and bending measurements), three pressure sensors (front, middle—within bag—and back) and an unnumbered set of impedance electrodes. A distensible bag, with attached tubing for filling, covers the middle part of the core, including a single pressure sensor and is said to be able to gather distensibility data (Figure 15). This technology is not yet available commercially and rigorous independent validation studies are still needed. However, this type of technology could solve many of the existing limitations of defecation investigation modalities and, perhaps, lead to a better understanding of the physiology of human defecation.

5 | CONCLUSION

Thus, this paper focuses on the morphology and physiology of the colon, anal canal and rectum. We provide a detailed overview of defecation and motility of the normal colon along with new techniques to investigate these functions. There has been an improved understanding of defecation and continence over the years. There remain gaps

in comprehension of the neurohormonal and brain–gut interactions. There is also a need to better understand the implications of colonic pathophysiology changes in disease states and the extent of their contribution to symptoms.

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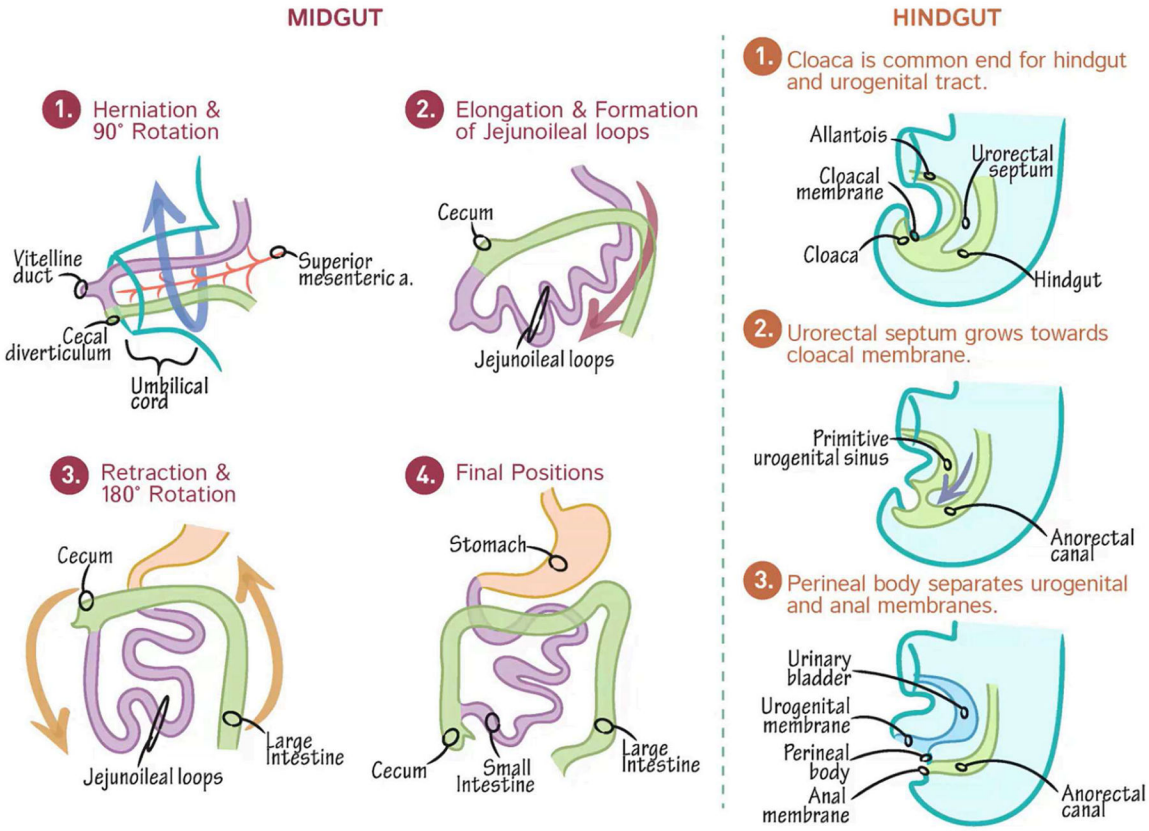

FIGURE 1.

Embryology of the colon and rectum. Midgut and hindgut anatomy, migration and growth during early weeks of development. Image was obtained from Ditki, Medical & Biological Sciences—Embryology Fundamentals: Formation, Growth, & Early Development.

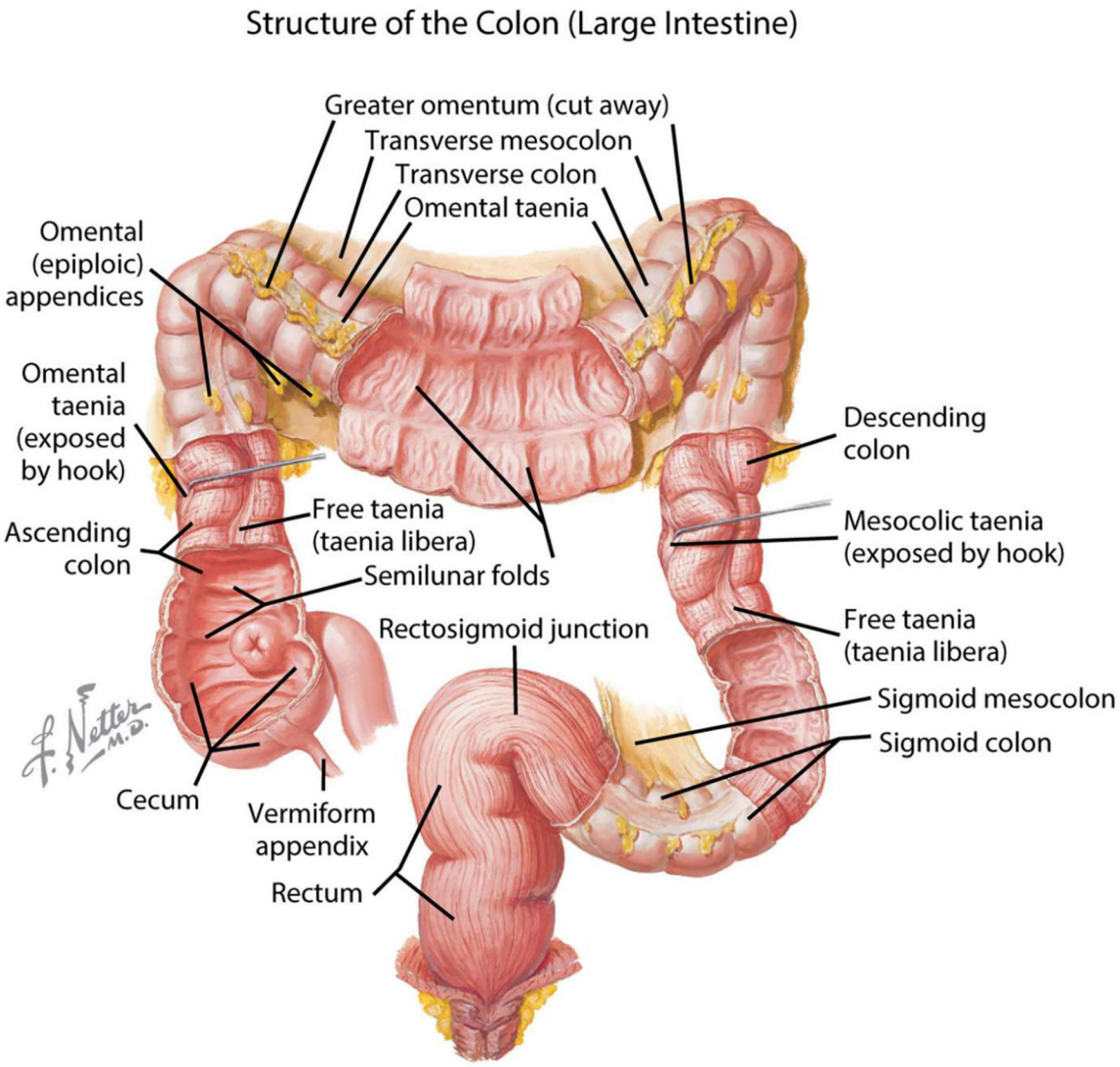

FIGURE 2.

Anatomy of the colon. Cutaway diagram of the colon including major anatomical divisions and terms, as well as cut away portions showing the layers of the colon. Netter illustration used with permission of Elsevier Inc. All rights reserved, www.netterimages.com.

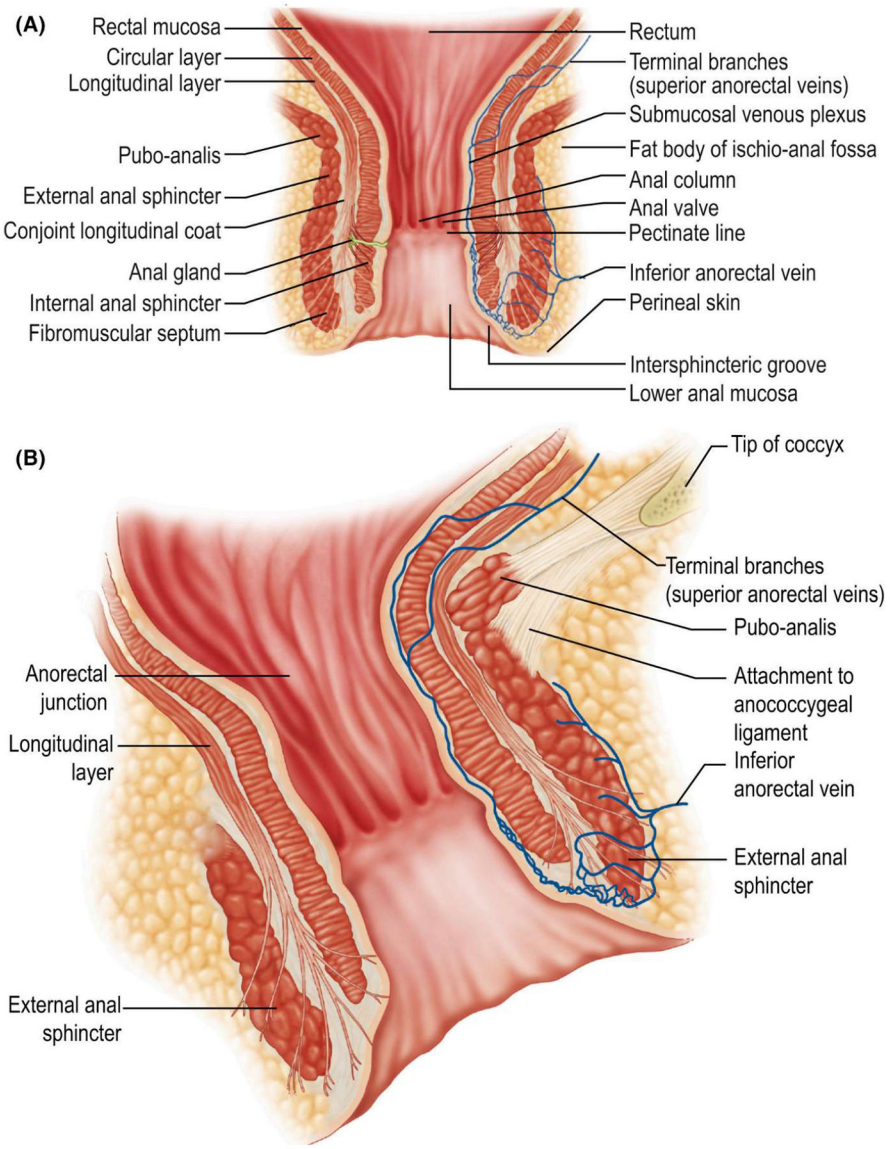

FIGURE 3.

Anatomy of anus. Cutaway illustration of the anal canal including important structures within the sphincter complex. (A) Coronal view of the anus and distal rectum. (B) Sagittal view of the anus and distal rectum highlighting the posterior attachment points to the coccyx. Reprinted from Grey's anatomy 42nd edition[7] with permission from Elsevier (Licence #5705371484150).

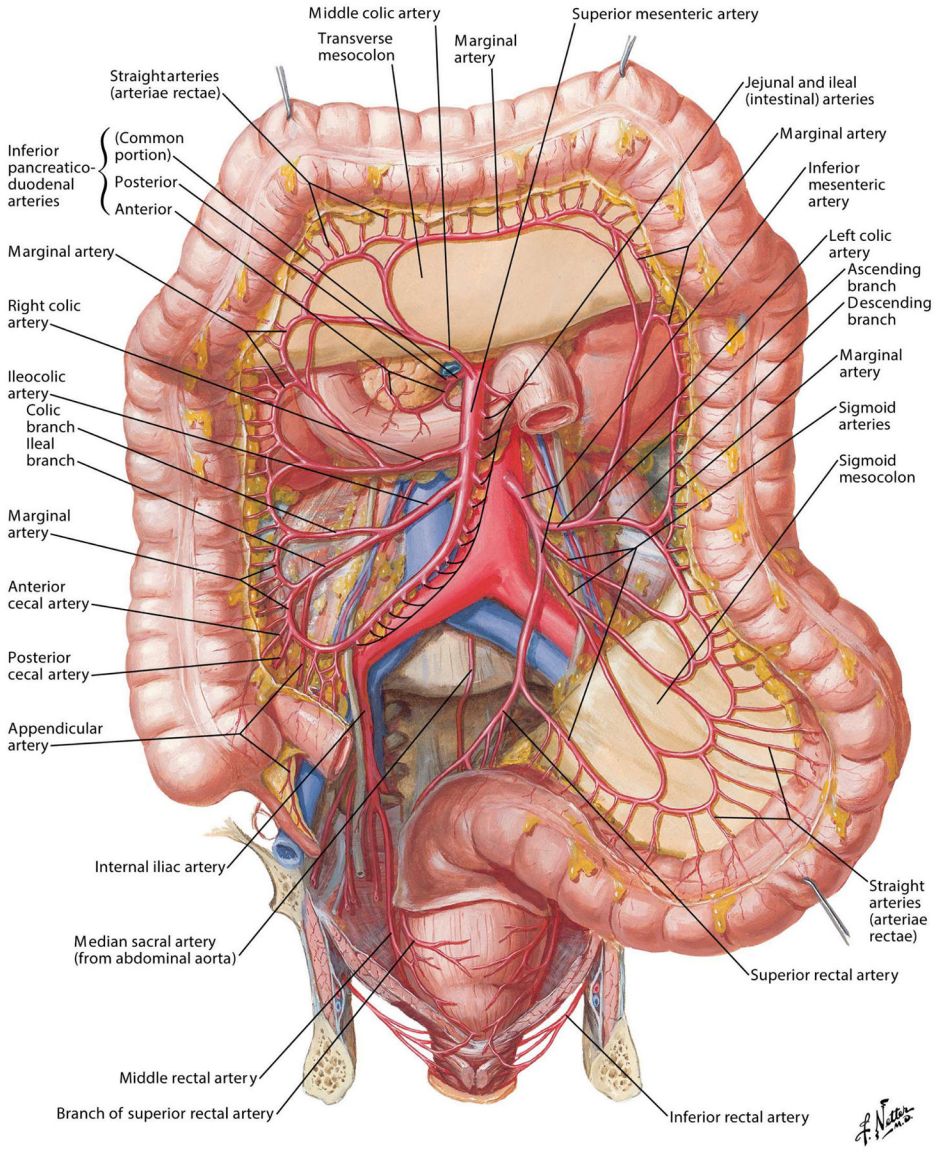

FIGURE 4.

Blood supply of the colon and anorectum. Illustrations showing the major gross anatomical divisions of the colon (top left box), and the arterial blood supply of the colon. Netter illustration used with permission of Elsevier Inc. All rights reserved, www.netterimages.com.

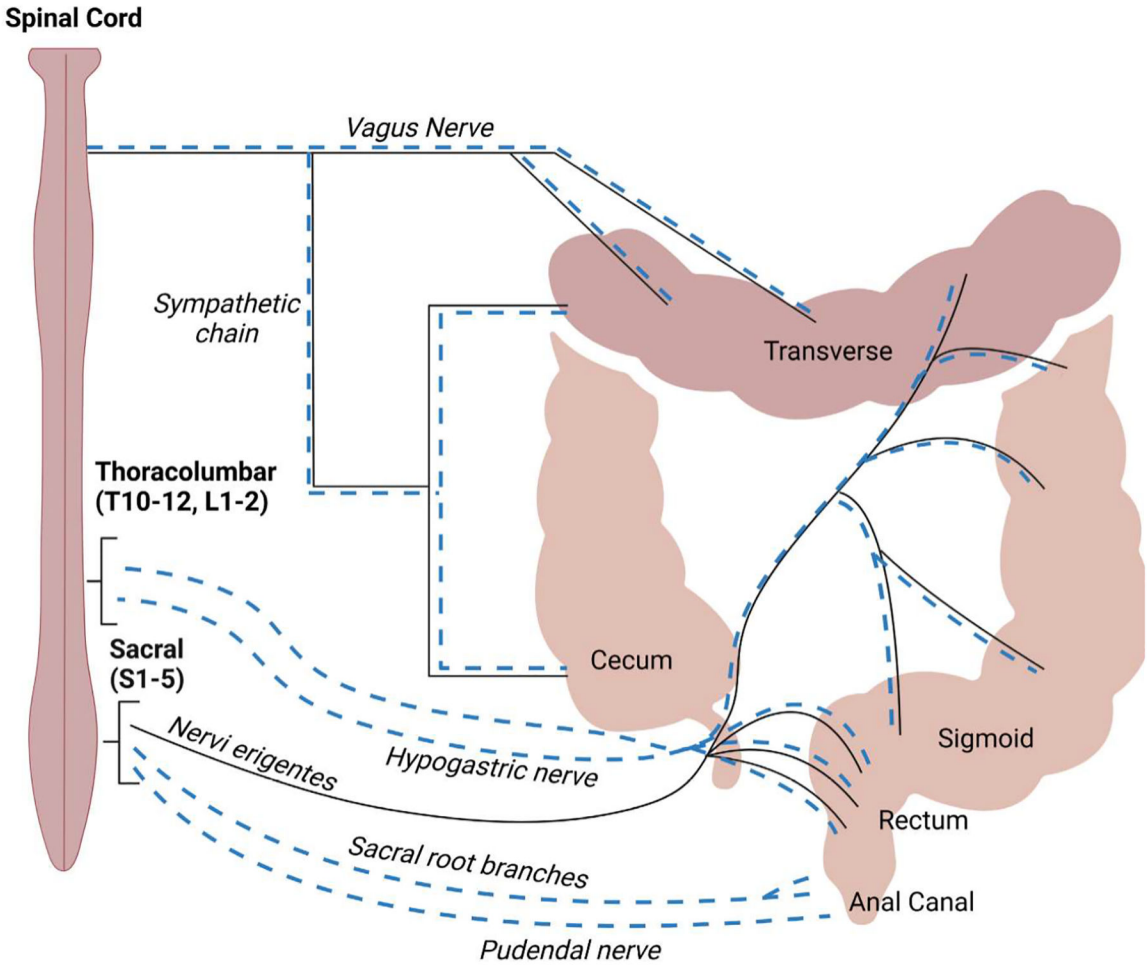

FIGURE 5.

Innervation of the colon and rectum. Illustration noting the sympathetic and parasympathetic innervation of the colon and the anorectum. Illustration constructed using BioRender.

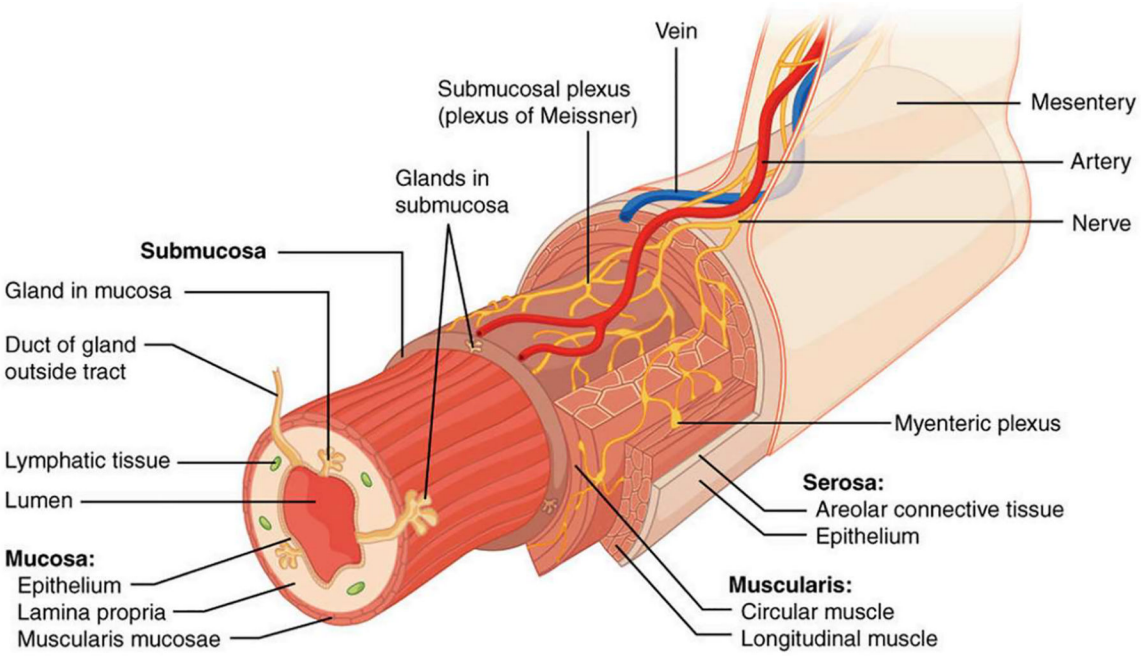

FIGURE 6.

Enteric nervous system. Illustration showing the organisation of the enteric nervous system within the layers of the gut. The myenteric plexus rests between the external muscular layer of the gut within the longitudinal and the circular muscle layers. The submucosal plexus with its inner and outer portions resides between the muscularis mucosa and the deep muscular plexus. Inserts showing respectively stained histopathology slides of the microscopic organisation of the enteric nervous system. OpenStax College, Rice University, CC BY 3.0 https://creativecommons.org/licenses/by/3.0, via Wikimedia Commons.

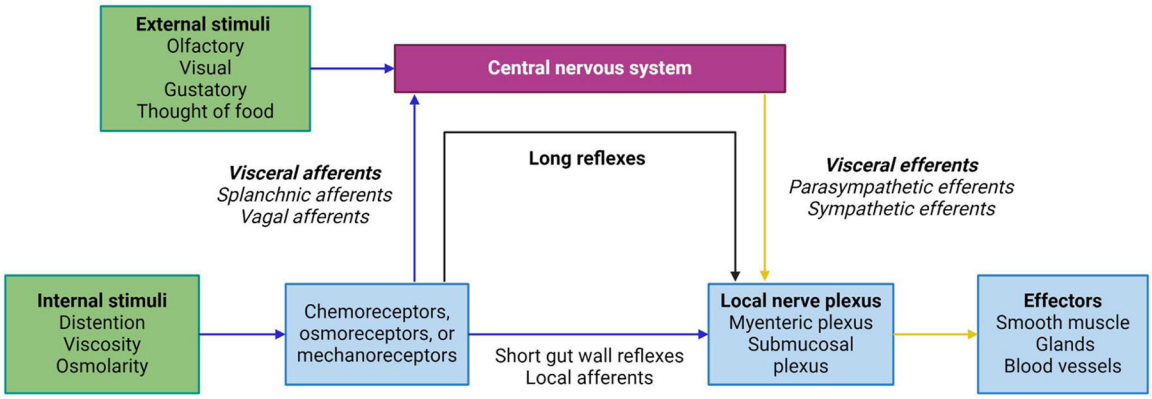

FIGURE 7.

Brain-gut interaction. Illustration showing the schematic organisation of the complex concept behind the interaction of the gut and the brain. The interaction between the gut and the brain is a feedback-type relationship between the central nervous system (CNS), the enteric nervous system (within the gut tube) and the communicating nerve tracts. Illustration constructed using BioRender. Adapted from Human Anatomy and Physiology 10th edition.[54]

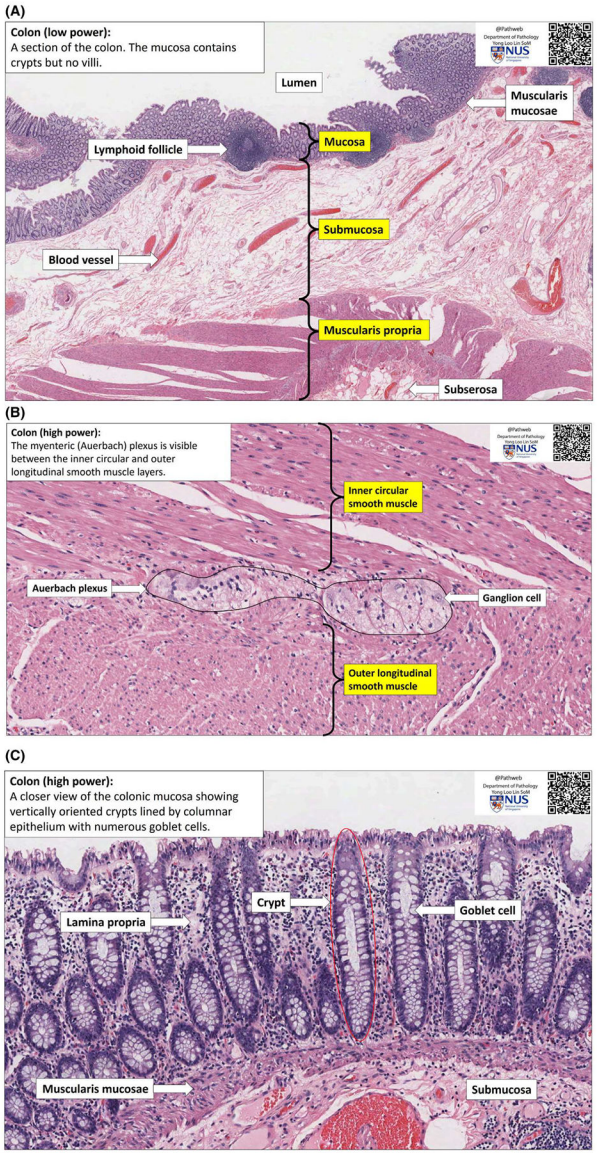

FIGURE 8.

Microanatomy of the colon. (A) Low power magnification showing normal colonic H&E histology highlighting the mucosa, submucosa and muscularis propria. (B) High power magnification showing normal colonic H&E histology highlighting the myenteric plexus within the inner circular muscle layer and the outer longitudinal smooth muscle layer. (C) High power magnification showing normal colonic H&E histology highlighting the organisation of the mucosa including crypts, lamina propria and goblet cells. Used with permission from Pathweb Online Resource, Department of Pathology, Yong Loo Lin School of Medicine, National University of Singapore.

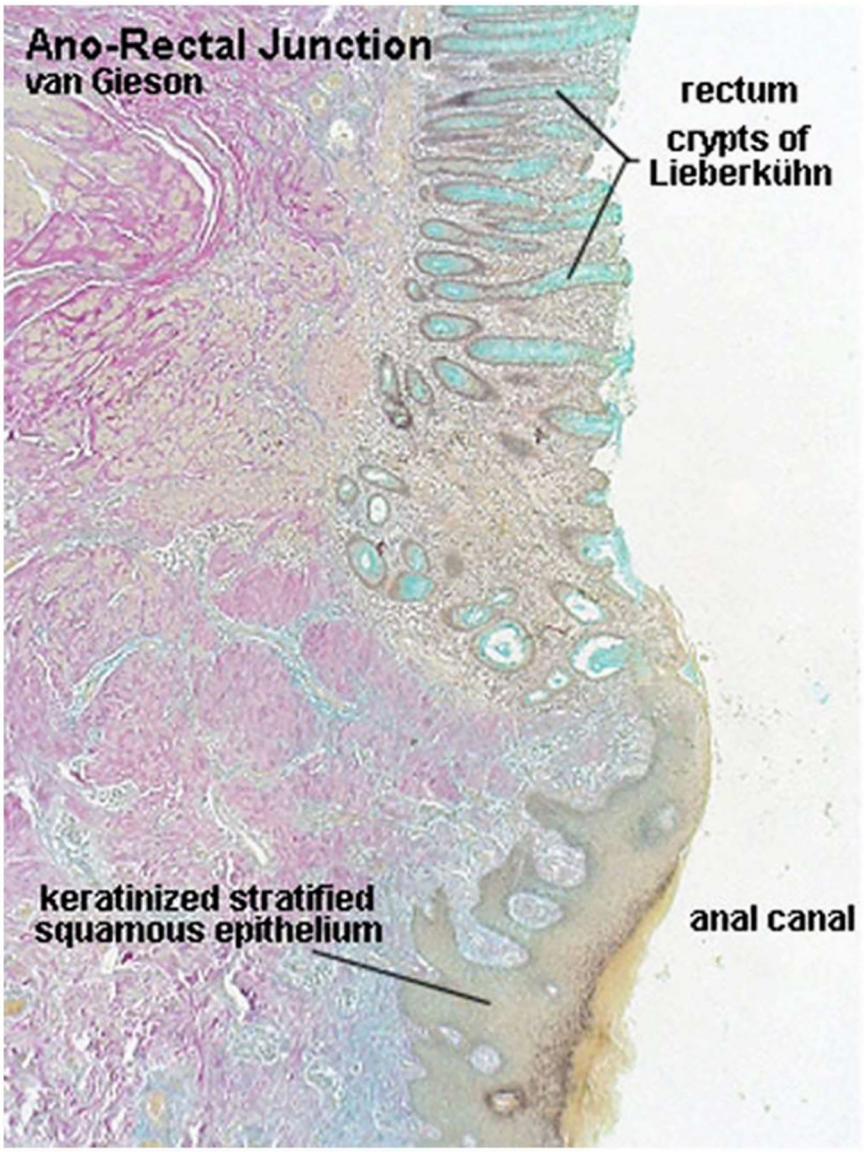

FIGURE 9.

Microanatomy of the rectum. Low power magnification of histopathology slide showing the normal organisation of the anorectal microanatomy. Highlights include the crypts of Lieberkuhn and the transition of epithelium from the rectal columnar epithelium to the stratified epithelium of the anal canal. Copyright Lutz Slomianka 1998–2009. Reproduced, published and distributed for non-commercial purposes as permitted by the original Blue Histology.[55]

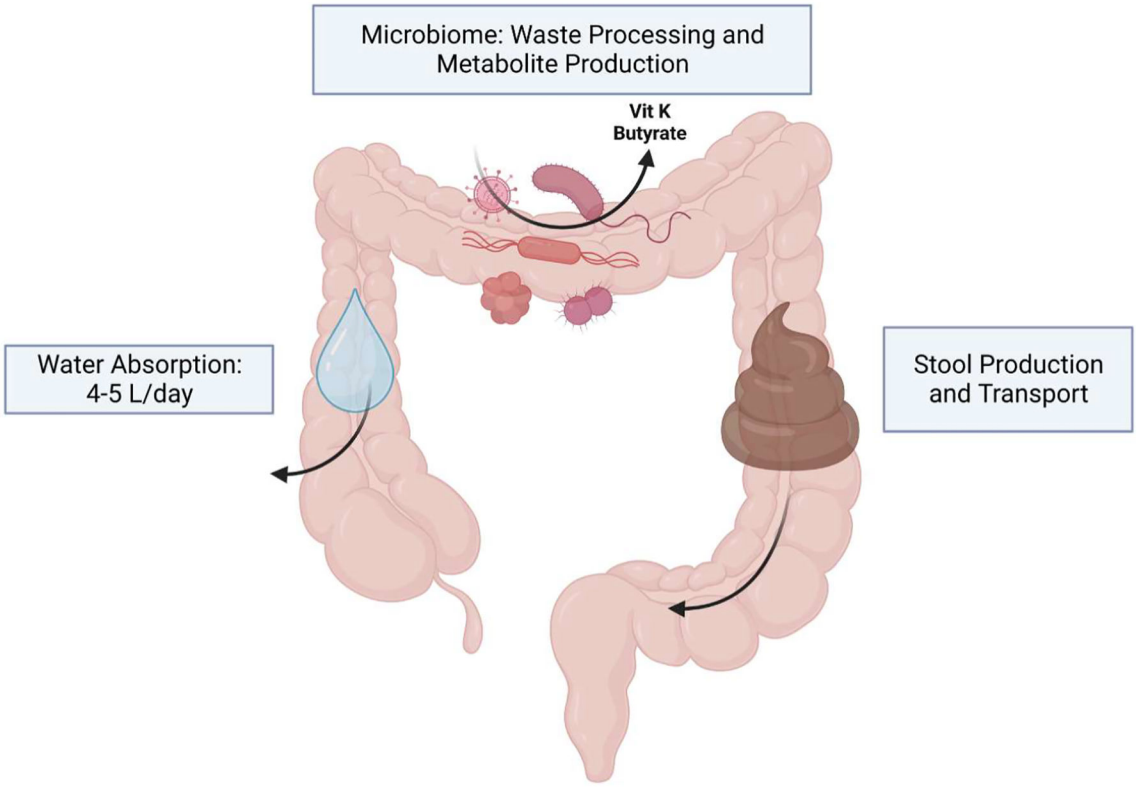

FIGURE 10.

Physiology of the lower gastrointestinal tract. Illustration showing the schematic organisation of the complex concept behind the interaction of the gut and the brain. The interaction between gut and the brain is a feedback-type relationship between the central nervous system (CNS), the enteric nervous system (within the gut tube) and the communicating nerve tracts. Illustration constructed using BioRender. Adapted from Human Anatomy and Physiology 10th edition.[54]

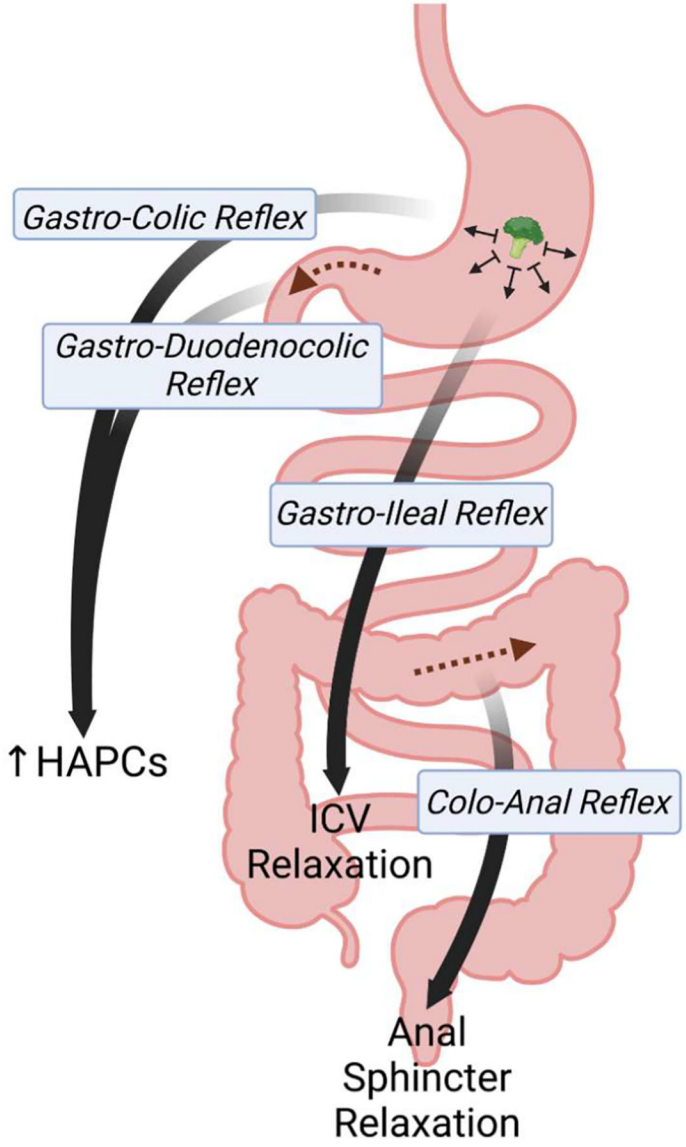

FIGURE 11.

Gastrointestinal reflexes. Illustration showing the known lower GI reflexes as they relate to the process of eating and movement of intraluminal contents. Highlighted are the gastrocolic, gastroduodenal, gastroileal and coloanal reflexes. These are described in detail within the text. Illustration constructed using BioRender.

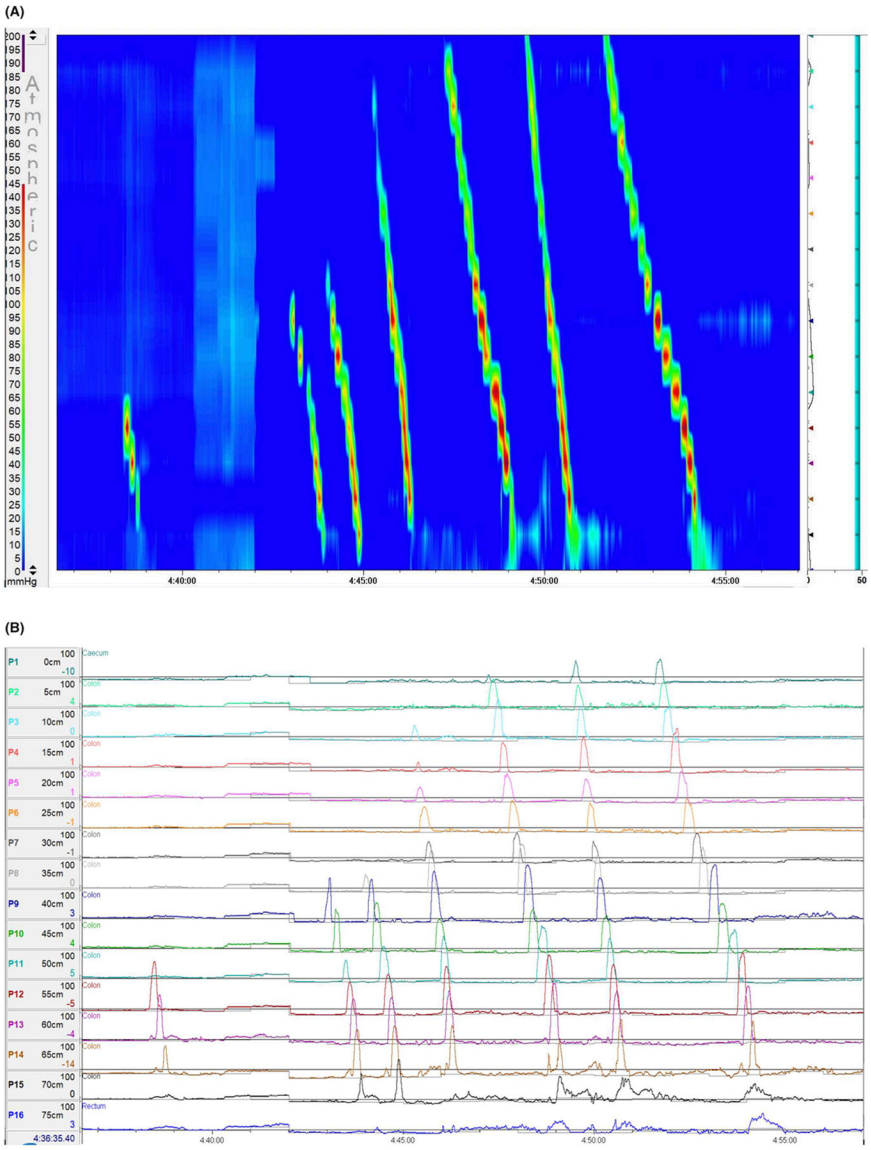

FIGURE 12.

High-amplitude propagating contractions. High-resolution colonic manometry segment displaying normal high-amplitude propagating contractions. (A) High-resolution image and (B) conventional tracings. Original image from de-identified patient.

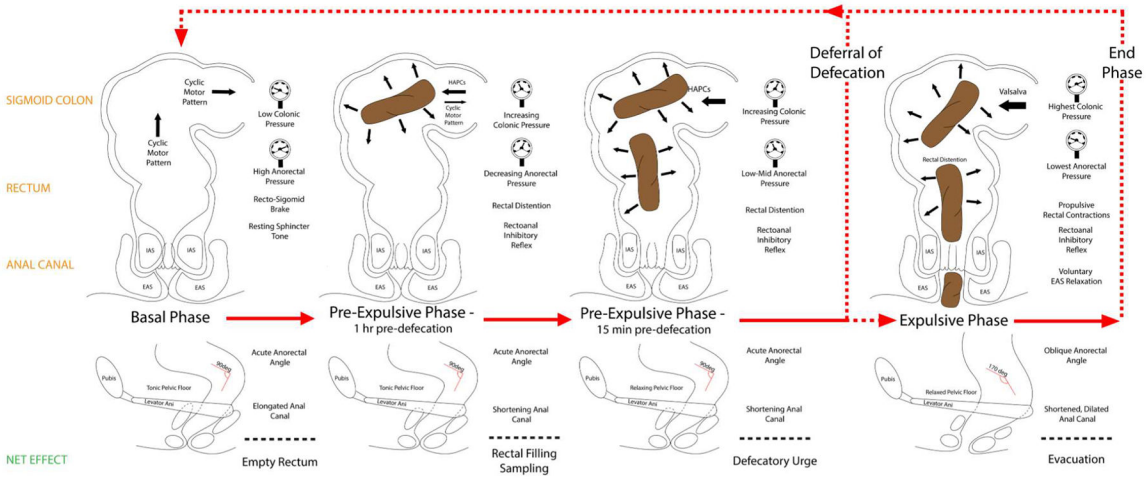

FIGURE 13.

The phases of defecation. Illustration showing the state of the rectum and anal canal, with important structures named, as they accommodate and change through the phases of defecation. The top row of images shows the anorectum as viewed from the sagittal perspective. EAS, external anal sphincter; IAS, internal anal sphincter. The bottom row of images shows a simplified schematic of the anorectum and supporting structures that are important to defecation and how they change during defecation.

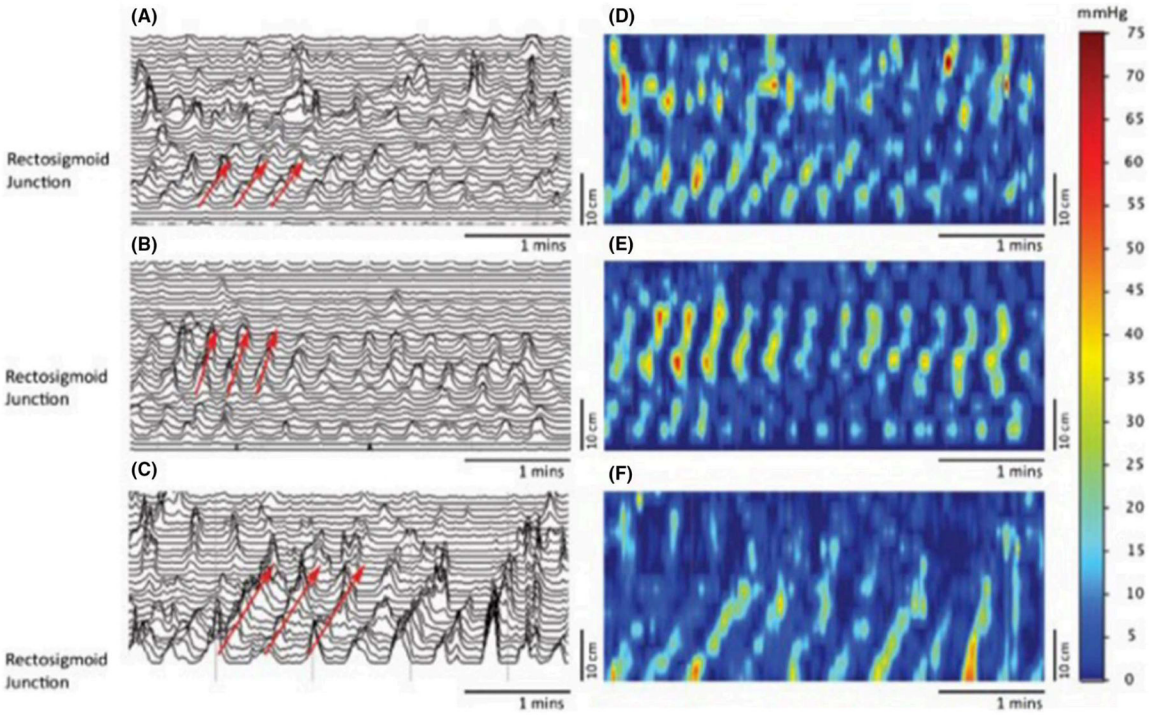

FIGURE 14.

Colonic cyclic motor patterns. Conventional and topographic views of a high-resolution colonic manometry segment showing an example of the cyclic motor patterns noted along the recto-sigmoid junction. Reproduced with permission from publisher John Wiley and Sons (Licence # 5705391489064). Original image source: Lin et al.[33]

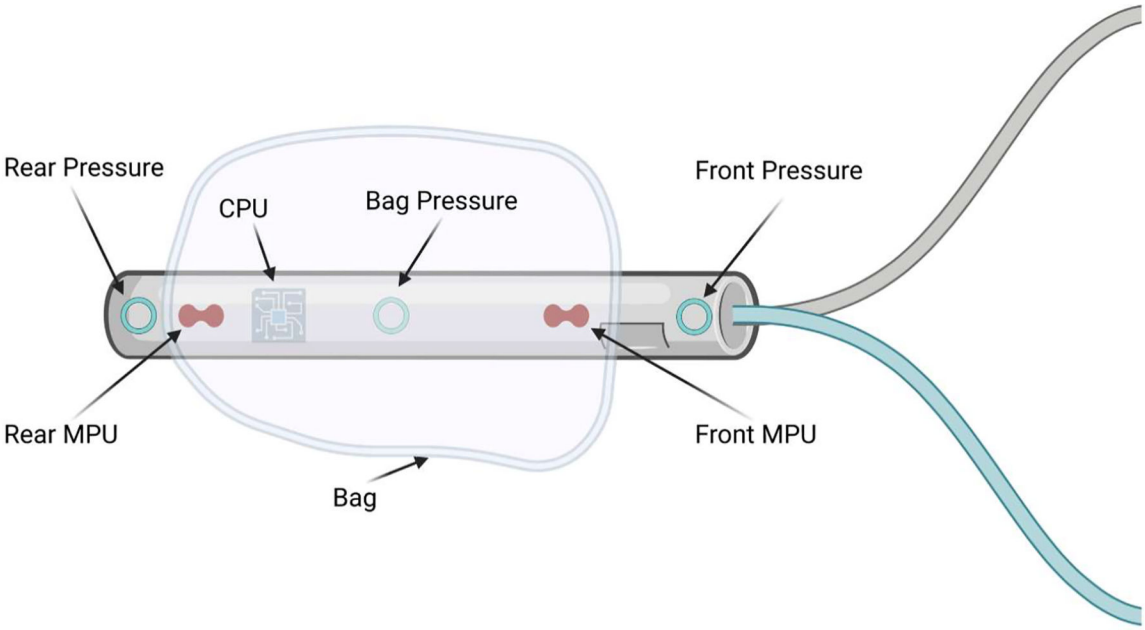

FIGURE 15.

Fecobionics. Illustrated fecobionics prototype device as designed and reported by Hans Gregersen et al.[53] This prototype shows three pressure sensors—two outside and one inside the pressure bag, two motion processing units (MPU) and a central processing unit (CPU). A communication conduit and an inflating catheter for the bag can be seen exiting the device. Illustration constructed using BioRender, adapted from Sun et al.[53]

TABLE 1

Standardised anorectal manometry protocol as published by the International Anorectal Physiology Working Group (IAPWG).

Standardised IAPWG anorectal manometry protocol[47,48]			
Manoeuvre (in order of occurrence)	**Time for manoeuvre**	**Notes**	**Measurement (unit)**
Stabilisation	3min	–	–
Rest	1min	–	Anal resting pressure (mmHg) and presence of ultraslow waves
Short squeeze×3	5s each–1 min 45s	30-s recovery intervals between	Anal squeeze pressure (mmHg)
Long squeeze	30s	–	Endurance of squeeze (s)
Recovery	60s	–	
Cough×2	~1min 30s	30-s recovery intervals between	Rectal/anal pressure (mmHg)
Push×3	15s each–2min 15s	30-s recovery intervals between	Rectal/anal pressure (mmHg)
Rectal sensory testing	Variable	Ramp distention–1–5 mL/s Phasic distention–l0 mL/s	Volume at first sensation, urge and intolerable (mLs)
Recto-anal inhibitory reflex (RAIR)	Variable	Starting volume of 30 mL, many need higher volumes if not elicited	Presence or Absence

TABLE 2

London classification of anorectal disorders assessed by high-resolution anorectal manometry.[47,48]

Classification part	Major finding	Minor finding	Inconclusive finding	Next steps
Part 1–Disorder of the Recto-anal Inhibitory Reflex	RAIR absent: Recto-anal Areflexia	None	None	Exclude aganglionosis with biopsy
Part 2–Disorders of Anal Tone and Contractility ARP–anal resting pressure ASP–anal squeeze pressure	• ↓ARP + ↓ASP: Combined anal hypotension and hypocontractility • ↓ARP + ↓ASP: Anal hypotension • Nml ARP + ↓ASP: Anal hypocontractility	↑ARP: Anal hypertension	None	Adjunctive testing needed
Part 3–Disorders of Recto-anal Coordination BET–Balloon expulsion test RPdP–Rectal pressure during push manoeuvre APdP–Anal pre-pressure during push manoeuvre	None	• Abn BET + ↑RPdP + ↓APdP: Abnormal expulsion with dyssynergia • Abn BET + ↓/↔RPdP + ↓APdP: Abnormal expulsion with poor propulsion • Abn BET + ↓/↔RPdP + ↑/↔APdP: Abnormal expulsion with poor propulsion and dyssynergia	• Abn BET + ↑RPdP + ↑APdP: Abnormal expulsion with normal manometric pattern of recto-anal coordination • Nml BET + Any Part 2 Abn: Normal expulsion with abnormal manometric pattern of recto-anal coordination	Consider defecography studies
Part 4: disorders of rectal sensation RCD–rectal ballon distention	• ↑≥2 RCD: Rectal hyposensitivity • ↓≥1 RCD: Rectal hypersensitivity	None	↑≥1RCD: Borderline rectal hyposensitivity	Consider barostat/ EndoFLIP assessment

Abbreviations, symbols:

↑, Increased or High;

↓, Decreased or Low;

↔, Unchanged; Abn, abnormal; Nml, Normal.