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**Gut Motility and its Control**

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**Abstract**

The gastrointestinal tract is composed of smooth muscle arranged in two layers: longitudinal and circular. Although its activity is influenced by the autonomic nervous system, it is mainly under local reflex control mediated by an enteric nervous system and local hormones. The motility of the gastrointestinal tract has several different well-defined patterns. Its function is to move the gastrointestinal contents through the various phases of homogenization (mixing), digestion, absorption and elimination.

**Keywords** Autonomic nervous system; enteric nervous system; neurotransmitters; peristalsis; segmentation; smooth muscle: longitudinal and circular

**Royal College of Anaesthetists CPD matrix:**

**Learning objectives**

After reading this article, you should be able to:

- understand the basic structure of the gastrointestinal (GI) tract
- understand the hormonal and nervous mechanisms that control GI function
- know about GI motility and how the gut moves its contents along its length

**Structure**

The gastrointestinal (GI) tract is essentially a long muscular tube that stretches from the mouth to the anus. While there is skeletal muscle at both ends of the tube, movement of the material contained within the GI tract is achieved by activity of smooth muscle.

There are two layers of smooth muscle along the GI tract (the outer longitudinal and inner circular muscularis). The outer layer is covered by a serous membrane (the peritoneum) that also forms the
mesentery through which arteries, veins and lymphatics pass (Figure 1). The GI tract is lined by mucosa (and beneath that the submucosa) both of which contain connective tissue, blood vessels and lymphatics (Figure 1). The luminal surface is lined by epithelium, and the mucosa and submucosa are separated by muscularis mucosa (Figure 1). The movement of the material contained within the GI tract depends on the intrinsic properties of the smooth muscle (undertaking rhythmic changes in excitability that varies along the intestine with endocrine and neural regulation). The small and large intestines display significant independent neural control (and are capable of functioning in the absence of extrinsic neural inputs), while the stomach and esophagus depend more on extrinsic neural inputs.

Nervous control

The GI tract has an intrinsic nervous system in the form of two nerve plexuses that allow significant autonomy in function. However, the central nervous system (CNS) provides extrinsic neural inputs that can regulate, modulate and control these functions via sympathetic (postganglionic) and parasympathetic (preganglionic) nerves.

The myenteric plexus, lying between the two layers of smooth muscle, helps control motor activity and the submucosal plexus, lying between the inner circular layer and the (sub)mucosa, helps control intestinal secretion and absorption. There are sensory, motor and interneurons (mostly they are interneurons in the enteric nervous system and not cholinergic having a large number of neurotransmitters see Table 1). Some of these neurotransmitters have paracrine activity and some local endocrine function. The enteric nervous system has as many nerve cells as the spinal cord.

Electrical activity

The smooth muscle along the GI tract has spontaneous and is a functional syncytium. The spontaneous electrical activity tends to be rhythmical, apart from in the oesophagus and proximal portions of the stomach, observed as slow waves that are known as the basic electrical rhythm (BER) where the membrane potentials fluctuates between -65 and -45 mV. The BER is initiated by the
interstitial cells of Cajal (stellate pacemaker cells that send long, branched processes across the intestinal smooth muscle). The fluctuation in the slow wave membrane does not always result in contraction and need to reach a threshold to generate an action and calcium release. The BER rate varies in different parts of the GI tract. In the stomach they happen about 3 times per minute, in the duodenum about 12 times per minute and in the terminal ileum about 8 times per minute. In the colon, the frequency is higher in the sigmoid colon (around 16 times per minute) than in the ascending colon (around 8 times per minute).

External nervous control
The extrinsic nerve supply to the GI tract is via the autonomic nervous system. The vagus supplies parasympathetic fibres to the oesophagus, stomach, small intestine and the large intestine as far as the transverse colon. The descending and sigmoid colons as well as the rectum are supplied by the parasympathetic sacral outflow (S2-4) via the sacral nerves. Parasympathetic fibres form synapses with ganglionic motor cells in the enteric nervous system using acetylcholine as the neurotransmitter. Parasympathetic stimulation generally increases GI tract activity (i.e. gastric, pancreatic and small intestinal secretions, muscle contraction and blood flow). The parasympathetic system also relaxes the gastro-oesophageal sphincter and causes adaptive relaxation of the stomach antrum to receive ingested food.

The sympathetic system inhibits GI tract activity, causing vasoconstriction, decreased motility and reduced secretions. Innervation is via the splanchnic nerves forming synapses in the coeliac, superior and inferior mesenteric ganglia. The preganglionic neurotransmitter is usually acetylcholine, and the postganglionic transmitter is norepinephrine. Postganglionic fibres innervate intestinal blood vessels and secretory glands as well as the enteric neurones, controlling muscle contraction. However, disrupting sympathetic function has little effect on normal GI tract function.
The GI tract has sensory neurones that respond to mechanical, thermal and chemical stimuli. Sensory impulses from abdominal viscera are generally transmitted via the sympathetic nerves, and from the distal colon via the sacral parasympathetic nerves (S2-4).

**Gastrointestinal motility**

The GI tract response to food intake is in the form of a series of reflexes. Some, such as peristalsis, are mediated entirely by the enteric system; e.g. a food bolus results in muscle contraction behind the bolus but muscle relaxation in front allowing it to move forward. Some reflexes (e.g. a food bolus entering the stomach) can require a high level of integration with other parts of the GI tract and involve the paravertebral ganglia or the central nervous system.

There are broadly three patterns of motility observed: peristalsis, segmentation (mixing) and tonic contractions.

**Peristalsis** is the wave of contraction that propels gastrointestinal contents distally along the GI tract, but normally only a few centimetres at a time. It is mediated by the enteric system and initiated by distension of the gut wall. It involves rhythmic contractions of the longitudinal muscles in the GI tract.

**Segmentation** involves multiple simultaneous rhythmic contractions of the circular muscle over discrete segments, causing an appearance similar to a string of sausages. It is characteristic of the fed state and moves the contents slowly towards the colon.

**Tonic contractions** take place in the proximal part of the stomach and in the various sphincters: the gastro-oesophageal sphincter, pyloric sphincter and ileocaecal junction and the internal anal sphincter as well as the sphincter of Oddi, where the bile and pancreatic ducts enter the duodenum. These are under hormonal and nervous control.

In the interdigestive phase (i.e. the periods between meals), there are occasional contractions within the small intestine (but only contracting a few centimetres of the smooth muscle). Migrating motor
complexes (MMC) are waves of electrical activity that regularly cycle through the GI tract during fasting. The MMCs trigger peristaltic waves to facilitate transport of indigestible substances (such as bone, fibre and foreign bodies) from the stomach, through the small intestine, past the ileoecal sphincter and into the colon. The MMC occurs every 45–180 minutes during the interdigestive phase and are easily recognised as rumbling sometimes experienced when hungry. They also serve to transport bacteria from small to large intestine and inhibit migration of colonic bacteria into the terminal ileum.

Feeding stimulates gastrointestinal motility and this is mediated by local hormones and nerves (parasympathetic and enteric), but the precise division of their different roles remains uncertain. Paralytic ileus occurs with intestinal injury/inflammation and is a reflex inhibiting the enteric system that is mediated by sympathetic afferents and efferents. The ileocaecal sphincter is normally kept closed by a reflex arising in the caecum but it opens in response to increased pressure in the ileum (increased caecal pressure causes it to contract and prevent reflux).

**Large intestine**

The large intestine absorbs fluids and electrolytes when forming, storing and periodically eliminating faeces. It can contain the residue of several meals eaten over 1 or 2 days (material usually remains there for between 12 and 48 hours).

Thicker circular smooth muscle around the anal canal forms the internal anal sphincter. It is overlapped at its distal end by the skeletal muscle of the external sphincter, which is under voluntary control. Motor activity in the large bowel consists of segmentation (haustration) to mix and compact the faeces. The contractions last for several minutes - significantly longer than in the small bowel. Peristaltic movements also occur in the colon and can propel the contents in distally and proximally.
Mass movements can occur up to three times per day. Stretch receptors are activated when faeces arrive in the rectum and mass movement in the sigmoid colon is mediated by the enteric system. The external sphincter is under voluntary control and, if circumstances are not appropriate, voluntary contraction will cause the smooth muscle of the colon and rectum to relax.

**FURTHER READING**

Table 1

<table>
<thead>
<tr>
<th>Neurotransmitters/hormones in the enteric nervous system</th>
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</thead>
<tbody>
<tr>
<td>Acetylcholine</td>
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<tr>
<td>Adenosine triphosphate</td>
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<tr>
<td>Cholecystokinin</td>
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<tr>
<td>Dopamine</td>
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<td>Endothelin 2</td>
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<tr>
<td>Enkephalin</td>
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<tr>
<td>Galanin</td>
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<tr>
<td>Gastrin-releasing peptide</td>
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<tr>
<td>Neuropeptide Y</td>
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<tr>
<td>Neurotensin</td>
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<tr>
<td>Nitric oxide</td>
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<tr>
<td>Peptide YY</td>
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<td>Pituitary adenyl cyclase-activating polypeptide</td>
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<td>Serotonin</td>
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<tr>
<td>Somatostatin</td>
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<tr>
<td>Substance P</td>
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<tr>
<td>Thyrotropin-releasing peptide</td>
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<td>Vasoactive intestinal peptide</td>
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Figure 1: Structure of the bowel and mesentry