1. INTRODUCTION: BASIC PRINCIPALS OF MOTILITY

1.1. General

The upper gastrointestinal tract transports food that has been eaten allowing digestion and ultimately absorption. The colon in humans has a storage and evacuation function and conserves fluid and electrolytes. In some mammals, i.e. the horse and the rabbit, the colon has a major function in the absorption of nutrients. The sphincters, placed at the junction between different parts of the gut, are specialized areas of smooth muscle, which regulate the forward and backward movement of intestinal contents. The ileocecal valve maintains a lower bacterial count in the small intestine by decreasing reflux of colonic contents into the ileum. The anal sphincter maintains contents in the colon until an appropriate time for expulsion.

In health, the movement of gastrointestinal contents is regulated strictly. This control of smooth muscle contraction is maintained by a complex interaction between excitatory and inhibitory neural stimuli exerted by myenteric nerves and circulating neuropeptides.

1.2. Electrophysiology

The rhythm and rate of contraction of the visceral smooth muscle of the stomach, small intestine, and colon is maintained by the intrinsic contractions of individual muscle cells. The rhythm of the cells is specific for each area of the gut, since each of the major sections of the gut has a different function. Electrical changes in the muscle membrane initiate intracellular biochemical reactions that result in muscle contraction. The intrinsic rate of spontaneous electrical depolarization of the muscle cell varies and is dependent on the location of muscle.

The esophagus has a slow wave rhythm that may be extensively modulated by the enteric nerves (1). In the stomach, the slow waves have a frequency of 3 cycles/min (2). Disturbance
in the generation of slow wave activity will lead to gastric retention (3). The frequency increases in the duodenum to approx 11 cycles/min, with a decrease in the frequency seen in the distal small intestine (4,5). The colon has an irregular slow wave rhythm of 3–6 cycles/min. The control of the slow wave rhythm appears to be set by the interstitial cells of Cajal (ICC) (6,7). Migrating spike bursts may be the electrophysiologic event controlling long distance movement of colonic contents (8,9).

1.3. Contractility

The electrical activity of the colonic smooth muscle initiates a contractile response in each of the layers of the muscular wall. Circular and longitudinal muscles have different functions. Throughout the gut, circular contractions segment the lumen, mixing the contents to expose the mucosa to continually different contents. The longitudinal muscle shortens the bowel, helping to move intraluminal contents forward.

The circular muscle contractions are phasic, which mixes the intraluminal contents and moves the bolus back and forth (10–12). The longitudinal muscle has tonic contractions that shorten the colon causing net forward movement of the colonic intraluminal contents (11–13). The differential contractions of the colonic smooth muscle layers is controlled by the enteric nervous system, as well as differences in the receptor subtypes for both stimulatory (e.g., substance P) or inhibitory (e.g., vasoactive intestinal peptide [VIP]) neurohormones (14,15). The movement of intraluminal contents through the colon is dependent on both contraction and relaxation of the smooth muscle. In addition to phasic and tonic contractions, high amplitude propagating contractions are important for the propulsion of intraluminal contents through the colon (16,17).

The motility patterns are normally coordinated to allow orderly transit of contents through the colon which “fine-tunes” the absorption of salt and water. Slow transit enhances the mucosal extraction of water, causing hard stools and constipation. Rapid transit causes frequent soft stools. Diarrhea associated with colonic motility disorders is low in volume (<400 mL/d), since most intestinal fluid has been absorbed in the small intestine. Table 1 lists the diseases or syndromes that cause disordered colonic motility. Many of the systemic diseases that affect gastric and small intestinal motility also alter colonic motility.

1.4. Sensation

Signals from the sensory afferent nerves help coordinate the colonic contractions through a series of neural reflexes (18). Sensation from these sensory nerves helps the individual monitor and control their bowel function. The sensory system has been examined in detail, and numerous mediators have been identified that control afferent sensory recognition (Table 2) (19). Changes in the tone of the colon will modify the sensory recognition. The increase in rectocolonic tone that occurs after eating increases sensory input that controls local and central nervous system-related reflexes (20). Faulty function in the visceral afferent nerves may be, in part, responsible for many of the symptoms of functional bowel disease.

2. TECHNIQUES TO MEASURE MOTILITY

Clinical and research techniques allow measurement of the different parameters of colonic motility: (i) intraluminal pressure; (ii) tone; (iii) electrical signals; (iv) transit; and (v) visceral sensation. Computerized methods for data acquisition and analysis allow the clinical application of colonic motility measurements, which will hopefully improve our care of these patients.