Chapter 10: Training in Motility and Functional Illnesses

DDSEP Chapter 4: Question 4

The mechanism by which a peristaltic contraction is propagated in the smooth muscle part of the tubular esophagus is:

A. A myogenic mechanism related to progressively more negative membrane potentials toward the distal esophagus
B. A result of sequential activation of the myenteric plexus by the vagus nerves
C. A result of sequential activation of the musculature by the vagus nerves
D. A function of the balance between inhibitory and excitatory myenteric plexus neurons at each esophageal locus
E. Under complete control of the CNS at the level of the medulla

The recommended response is D.

The sequencing of peristalsis is a result of increasing dominance of inhibitory neurons toward the distal esophagus with the effect of delaying the excitation. By experimentally eliminating the influence of the inhibitory neurons, the result is a simultaneous contraction. Removing the vagal influences (and hence CNS influences) altogether does not change the propagation of peristalsis, though it does prevent deglutition-induced primary esophageal peristalsis.
Gastric peristaltic contractions occur normally at a rate of 3 per minute, due to:

A. Vagal excitatory innervation  
B. **Gastric slow waves**  
C. Antroduodenal contraction and coordination  
D. Duodenal pacemaker activity  
E. Rhythmic pyloric contractions

The recommended response is B.

Gastric peristaltic contractions occur at a rate of 3 per minute because the gastric slow waves occur at 3 cpm. Gastric slow waves are also called pacesetter potentials or electrical control activity. These electrical waves set the timing and the propagation velocity of the peristaltic contractions of the stomach. Action potentials and plateau potentials are the electrical correlate of circular muscle contractions. Antroduodenal coordination occurs with the normal emptying of the stomach but does not control the rate of contraction. The pylorus contracts in synchrony with or independently from the antrum and is not responsible for the frequency of gastric contractions. Finally, the fundus is electrically silent and does not have rhythmic electrical pacemaker activity, and the duodenal pacing does not determine the slow wave frequency of the stomach.


Chapter 10: Training in Motility and Functional Illnesses

DDSEP Chapter 4: Question 10

All of the following physiological defects have been described in patients with IBS without fibromyalgia except:

A. Increased colonic motility in response to a meal
B. Prolonged propagated contractions in the small and large intestine
C. Increased visceral sensitivity to rectosigmoid balloon distension
D. Increased somatic sensitivity to tactile and thermal stimuli
E. Altered CNS activation in response to bowel stimuli as measured by PET and functional MRI scanning

The recommended response is D.

A number of both psychologic and physiologic abnormalities have been described in patients with IBS. Present understanding of the basis of IBS has shifted away from motility being the cause to motility disturbances being secondary phenomena. Visceral hypersensitivity is demonstrated in two-thirds of patients with IBS. Interestingly, somatic hypersensitivity has not been found, at least in some studies.

