Introduction

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In normal digestion, food content is propelled through the tract by rhythmic contractions or propulsions called peristalsis.

Impaired peristalsis includes slow propulsions (hypomotility), rapid propulsions (hypermotility), or a combination of both, in some cases there may be a total absence of digestion contractions.

Motility of the G.I.T regulates the orderly movement of ingested material through the gut to ensure adequate absorption of nutrients, electrolytes, and fluids.

The digestive motility includes a number of events:

1- Myogenic events in the smooth muscle cell (myocyte) leading to contractions.

2- Neurogenic events, which coordinate smooth muscle contractions by both intrinsic & extrinsic nerves.

3- Coordinated smooth muscle contractions lead to elevated intra luminal pressure and propulsion.

4- Propulsion, which is the net result of events, one through four ***(Phillips, S.F., 1990).***

Digestive motility disorders may be primary meaning there is no underlying disease causing the problem or secondary when the motility disorder occurs as a result of a disease.

Some digestive motility disorders may be familial (genetic in nature) or idiopathic meaning there is no known cause.

The nervous system modulates motility through both intrinsic & extrinsic neurons ***(Zenilman, M.E., et al., 1993).***

The enteric nervous system integrates information from the periphery & the C.N.S and modulates effector function.

In perivertebral ganglia, afferent inputs from the gut & descending inputs through the symp. branches of the A.N.S are integrated into output that has primarily inhibitory effect on gut motility ***(Mayer, E.A. and Raybould, 1989).***

Autonomic nuclei of symp. & para symp. nerves located in the brain stem integrate inputs from the periphery & the cortex output reaches the gut via the para symp. & symp nerves ***(Mayer, E.A. and Raybould, 1989).***

Motility of the G.I.T is a function of the individual myocyte resting membrane potential, its ability to depolarize spontaneously, the cellular threshold for firing of an action potential, the cell's ability to communicate with others, and extrinsic control of its ionic channels ***(Zenilman, M.E., et al., 1993).***

Motility disorders affecting the oesophagus may be primary and includes: Achalasia and diffuse oesophageal spasm or secondary including: scleroderma, DM & Amyloidosis ***(Sugarbaker, D.J., et al., 1994).*** Oesophageal motility disturbance whether hypomotility as in achalasia or hypermotility as in diffuse oesophageal spasm of oesophagus both characterized by disturbance of oesophagus at lower oesophageal sphincter ***(Sugarbaker, D.J., et al., 1994)***.

Also, disorders motility include Gastro oesophageal reflux, post gastrectomy syndrome whether dumping syndrome, small gastric remnant syndrome, post vagotomy diarrhoea, gastric atony & Roux stasis syndrome ***(Kelly, K.A., 1981).***