Gastro intestinal motility :-

- Wall of GIT → Mucosa ← Mucosal epithelium, Lamina propria, Muscularis mucosa – Thin layer
  → Sub mucosa
  → Muscularis externa → Inner circular, Outer longitudinal

- Most of the GI musculature – muscularis externa
- Smooth muscles of GIT – large bundles – one bundle contains about 1000 smooth muscle cells. These bundles are oriented longitudinally in outer layer of muscularis externa and circularly in inner layer.
- The smooth muscles are interconnected by gap junctions – low resistance communicating junc. Ions & small metabolites can move from one SM to another.

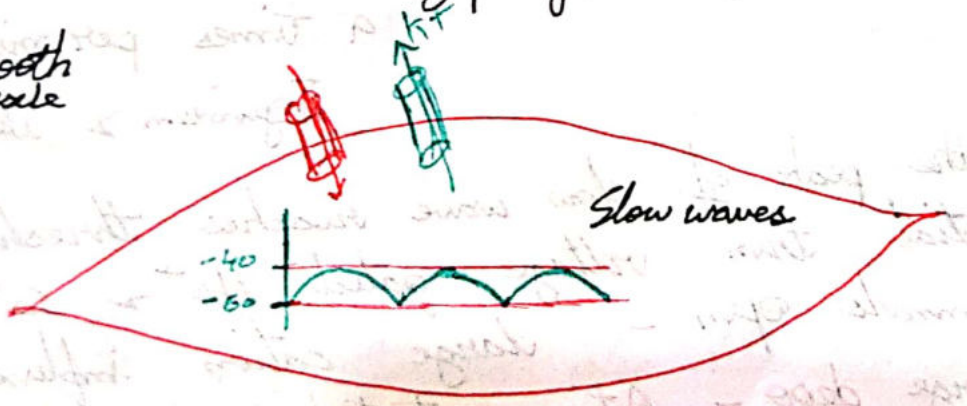
  Bcoz of this any significant electrical activity in one smooth muscle cells will be communicated to neighbouring smooth muscle.
- All the smooth muscles in one bundle are behaving as a network of smooth muscles which are electrically syncitium.
- Gap junctions are also present B/w smooth muscles of different bundles.

- Smooth muscles in GIT are connected by gap junctions and act as electrical syncitium.
  Hence these smooth muscles are called unitary smooth muscles.
  Act as electrical network.

- More gap junctions are present in the circular layer of muscularis externa.

- There are some spl cells which can electrically stimulate the smooth muscles of GIT.
  They are called interstitial cells of Cajal.
  Present B/W longitudinal and circular layer and can produce pacemaker activity
  Their membranes can undergo electrical fluctuations, which spread to the smooth muscles thru gap junctions.

- Smooth muscle



RMP is somewhere B/W −60 mv & −40mv

In the SM of GIT there are 2 theories

① Presence of Ca^{2+} channels :- allows trickling of Ca^{2+} in membranes. to enter causing slight depo

These spl channels allow Ca^{2+} & Na^{+} influx causing slight depo. Once a peak is reached K^{+} channels open - K^{+} efflux - RMP is restored. Again at RMP the Na^{+} Ca^{2+} channels open allowing small amt of cations to enter and the cycle continues.

This pattern of fluctuation of membrane potential of smooth muscle cells is called Slow wave

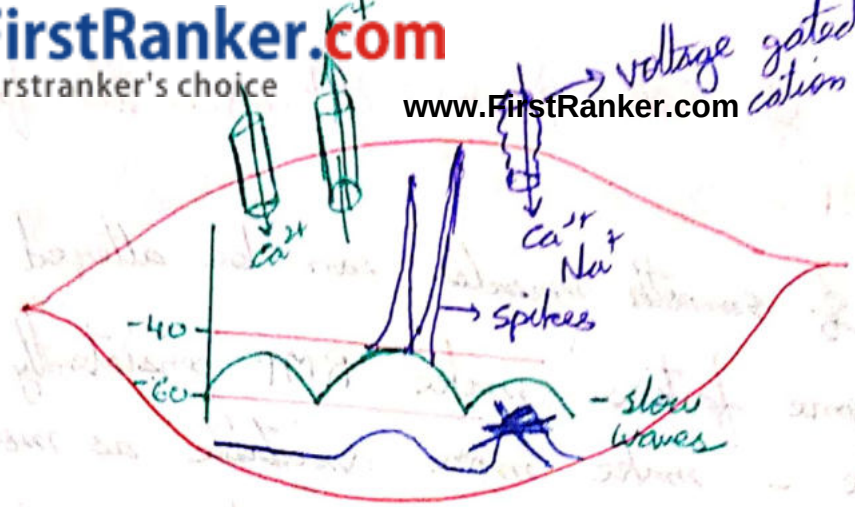
Slow waves are "not action potentials"
↳ inherent tendency of GIT smooth muscles.

Rate of slow waves => 3 times per min in Stomach
12 times per min in duodenum
9 times per min Jejunum & ileum.

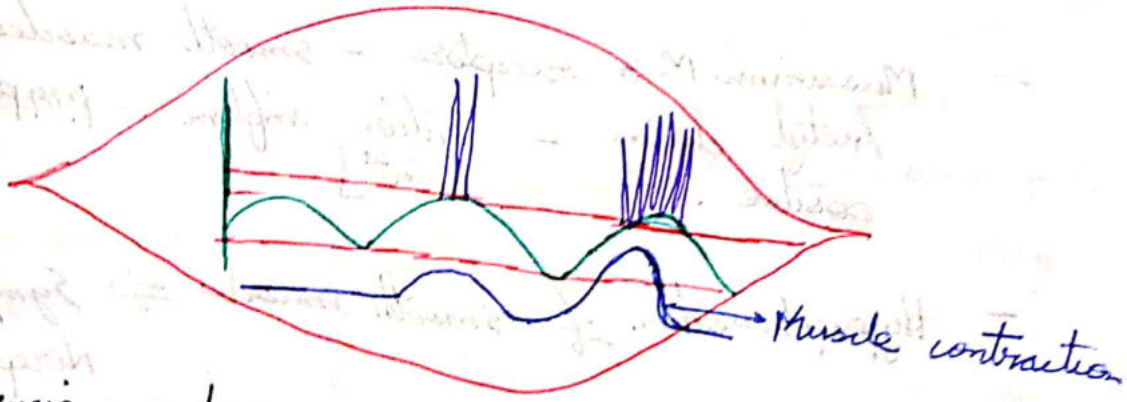
"Don't produce contractions"

- If the peak of slow wave reaches threshold potential then voltage gated Na^{+} & Ca^{2+} channels open - large cation influx - Large depo - Action potential / spike potentials.

Slow waves play a major role in producing spike potentials.



- If the peak of slow waves goes above the threshold potential - multiple spikes.
- Number of spike potential depends on magnitude of slow wave and on duration of slow wave peak at threshold.



Tension prod in smooth muscle is directly prop to the heavy Ca^{2+} influx of spike potentials.

- Frequency of slow waves also determine rhythmic contraction.
- Phasic contraction depends on frequency of spike potentials.

- Strength of ~~of~~ determines the frequency of AP.

- RMP of smooth muscles can be altered.
  If some factors make RMP persistantly more positive - make more excitable as more slow waves reach threshold - excitability is increased.

- When smooth muscles are distended the Ca^{2+} channels become under increasing the cation influx.
  RMP remains persistantly towards Threshold. More slow waves reach threshold - more excitable.

- Muscarinic M 3 receptors - smooth muscles.
  Acetyl choline - cation influx - RMP more positive. $[Ca^{2+}]$

- Hyperpolarisation of smooth muscles ⇒ Sympathetic N:
  Norepinephrine.

⇒ Enteric Nervous System

- Submucosal Plexus or Meissner's Plexus
  ⇓
  In submucosa
  Controlling secretion of glands & Blood flow, absorption, Neuro endocrine cells

- Myenteric Plexus or Auerbach's plexus
  ⇓
  B/W IC & OL
  Regulation of motility of GIT

enteric N.S can work independent of extrinsic N.S

Law of Peristalsis :- Oral side of the bolus must contract like a ring and anal side should be relaxed.

distension of GIT / Any other stimulus eg :- mucosal irritation.

↓

Sensory neuron

↓

Stimulates Neurons in Myenteric plexus.

Myenteric plexus ← Ascending grp of neurons
Descending grp of neurons.

When a neuron in ascending grp is stimulated (By sensory neuron) it prod. stimulatory neurotransmitters acetylcholine and substance P. They further stimulate the ascending neurons.

The neurotransmitter will especially stimulate the circular muscles proximal to bolus.

When descending neurons are stimulated inhibitory neurotransmitters are released NO, Vasoactive intestinal peptide [VIP].

The neurotransmitters especially inhibit the longitudinal muscle, and some inhibit circular muscles as well

- As a result the circular muscles aboral to stimulation relax and oral to it contract. The bolus will be pushed forwards.

- Enteric NS influenced by SNS > PSNS

  ↳ Neurons of ENS can be considered post ganglionic neurons of parasympathetic NS

- Types of Sensory fibres GIT :-
  1. local sensory fibres - local reflexes
  2. Sensory fibres upto sympathetic ganglion - mediated reflexes
  3. Sensory fibres which are taking sensations upto central NS. eg :- medulla.

- Three Reflexes related to GIT :-
  1. Local reflexes - Intrigut reflexes.
  2. a) Gastrocolic reflex ⇒ Reflexes which involve ganglia. These are the reflexes called Reflexes from the gut to prevertebral sympathetic ganglion and back to gut.

     When stomach full ⇒ colin movt (↑).

     b) Enterogastric reflex :- When intestinal lumens are overloaded the stomach is inhibited.

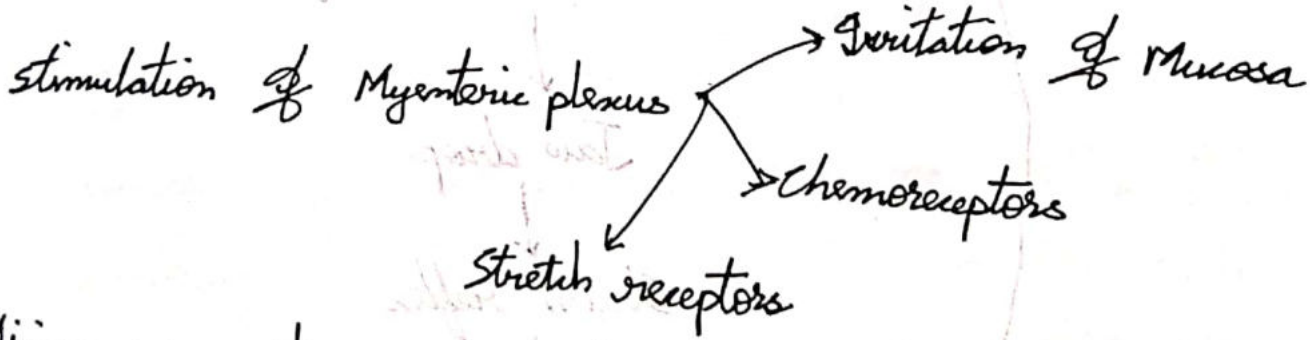
Colonic reflex :- when colon is overloaded ileum is inhibited & ileo caecal valve contracted.

③ Reflexes involving CNS eg:- Vagovagal reflexes.

⇒ Functional type of Movements in GIT :-

① Propulsive moments / Peristalsis
② Mixing movements.

- propulsive moments are inherent tendency of tubes in our body that have smooth muscles and those smooth muscles are interconnected by gap junctions.
- Peristalsis :-
  a) Bolus distends the walls of GIT
  b) stimulation of stretch receptors
  c) stimulation of Myenteric plexus



- Mixing movement :- mix the GI secretions with food bolus
  - localised constriction rings develop at multiple sites simultaneously and divide the GIT into multiple segments.

Congenital absence of Myentric plexus :-
Myentric plexus is absent in some segment of GIT

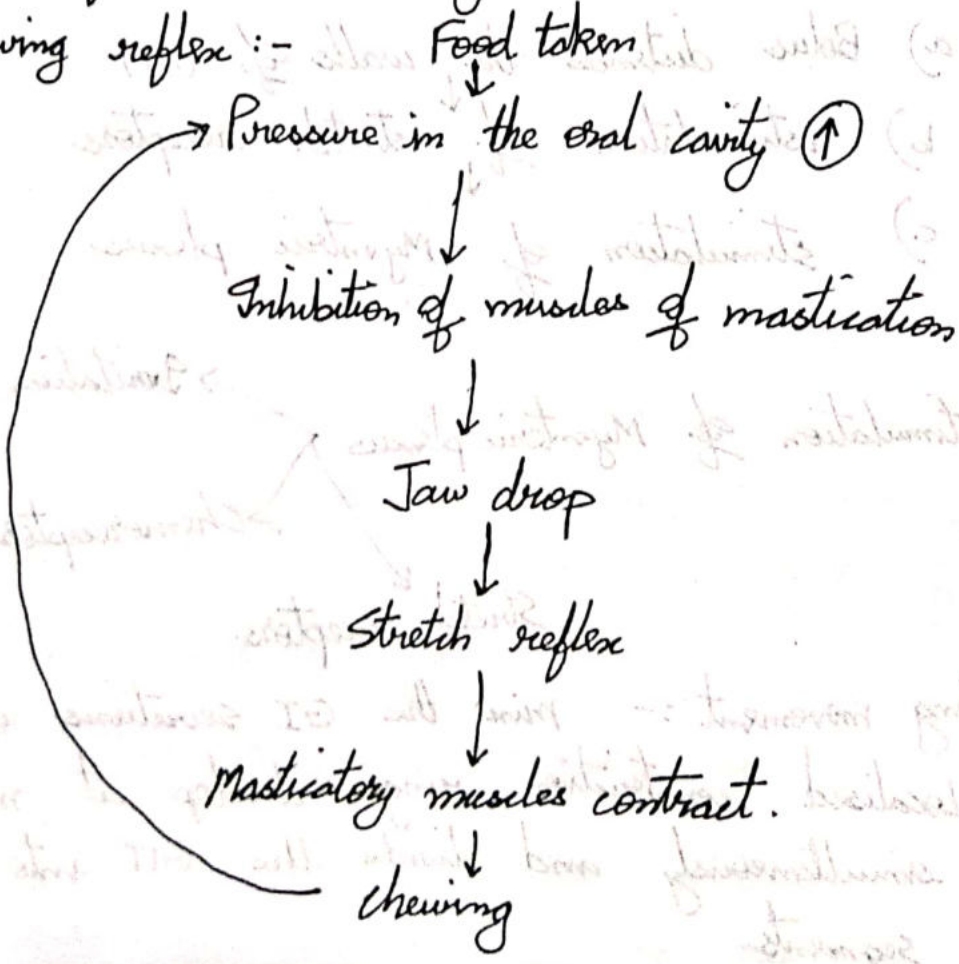
- Atropine - blocks muscarinic receptors.

- Law of Gut :-

Our GIT has myenteric plexus polarised in such a way that whenever any part of GIT is distended, peristalsis prod by myenteric plexus will move more towards anal side and dies out at oral side.

⇒ Mastication / Chewing :-

- Both reflex and voluntary
- chewing reflex :-



Jaw & Chewing :-

1. Mixing of saliva with food
2. Food particles broken down into smaller particles
3. food bolus lubricated.
4. less chance of excoriation of mucosa
5. Increasing surface area of food particles.

⇒ Swallowing :- deglutination

Process of taking the bolus of food from posterior part of oral cavity to the stomach.

I Oral stage :- 1 to 2 sec

- voluntary
- Tongue moved upward
- food bolus pressed against the palate and pushed backward.
- At the opening of oropharynx - many sensory receptors (especially tonsilar pillars) for swallowing purpose - they trigger the swallowing reflex.

II Pharyngeal stage :- less than 6 sec

- Sensory info is sent to CNS thru 5th & 9th CN
- Swallowing reflex center near to respiratory centre in medulla, present in reticular formation very near to Tr. to solitarius nucleus.
  → Activated
- Motor fibres are sent thru 5th, 9th, 10th, 12th cranial nerves.

- Muscles contract in such a way that the food bolus is pushed down into esophagus.
- As soon as swallowing reflex is activated
  1. Soft palate moves upwards, passage to nasopharynx closed.
  2. Vocal cords approximated
     Larynx moves upwards & anteriorly
     Epiglottis closed the larynx.
  3. Upper esophageal sphincter open.
- Food has only one way to enter
- Peristaltic wave is produced in pharyngeal muscles.
- Food bolus enters into esophagus
- Pharyngeal phase of swallowing is completed.

III Esophageal phase :- 8 to 10 sec.

- As soon as bolus comes into upper esophagus it does not produce a new peristalsis but the peristaltic wave which was sweeping over the pharynx continues all over the esophagus. This is called Primary peristalsis.
- lower part of esophagus [lower esophageal sphincter] is thickened and here the smooth muscle has a higher tone.
- Approaching peristalsis relaxes the lower esophageal sphincter and upper part of stomach.

- This is called Receptive relaxation. - relaxed to recieve the food.

- In some conditions lower esophageal sphincter fails to relax - food keeps on accumulating in esophagus. This is called Achalasia. — Myenteric ganglia are absent in lower esophagus / or are destroyed.

  eg :- Trypanosoma cruzi ⇒ causes Achalasia congenital absence.

- If some food is retained within the lumen of esophagus wherever it is present secondary peristalsis are generated there by distension & stretch.