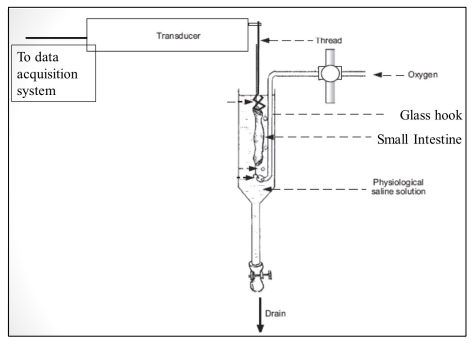
GI Physiology Lab experiment about the effects of acetylcholine (ACh) and atropine on intestinal motility,

Aim of the Experiment :



- Observe the natural (rhythmical) contractions of the small intestine.
- Investigate how acetylcholine (ACh) and atropine modify these contractions.

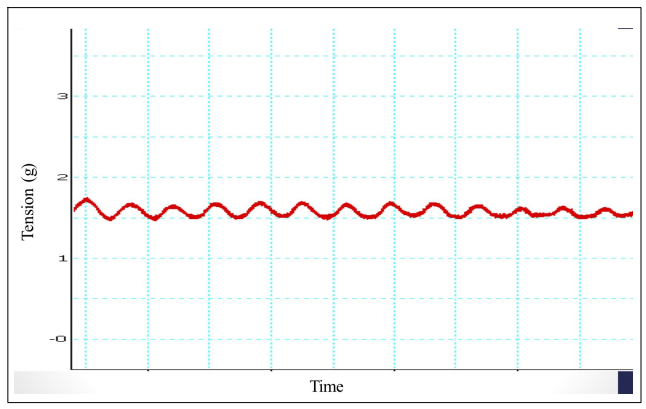
Method Overview :

Small segments of rat small intestine (2–3 cm) are suspended in an organ bath filled with warm, oxygenated buffer (37°C).

A tension transducer is used to measure muscle contractions.

The signals are recorded and displayed as a graph of tension vs. time.

After stabilization (15–20 min), ACh is added, followed later by atropine.



Key Physiology Concepts from the Discussion

1. Phasic (Rhythmic) Contractions :

- The small intestine naturally undergoes periodic contractions and relaxations (phasic motility).
- These are not dependent on nervous or hormonal input — they occur intrinsically due to:

[Slow waves: Rhythmic fluctuations in resting membrane potential] .

The rhythm is determined mainly by the frequency of the "slow waves" .
Generated by Interstitial Cells of Cajal (ICCs) — the electrical pacemakers of the gut.

2. Slow Waves and Spike Potentials :

- Slow waves are regular, slow changes in membrane potential. They set the rhythm, but don't cause contractions on their own.

- Slow waves occur at different frequencies at various points along the gastrointestinal tract. In humans their frequency is 12/minute in the duodenum, 8-9/minute in the ileum. Slow waves set the maximum frequency at which contraction can occur at a particular site.

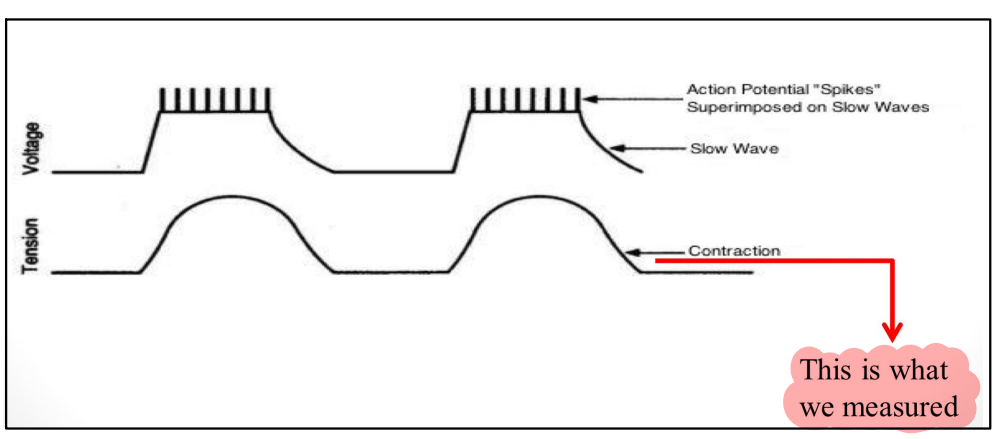
- When a slow wave exceeds −40 mV, it triggers spike potentials (true action potentials) that cause contractions.
- For a contraction to occur, a spike potential must be generated by smooth muscle cells, seen as transient membrane depolarization superimposed on the peak of the slow wave.

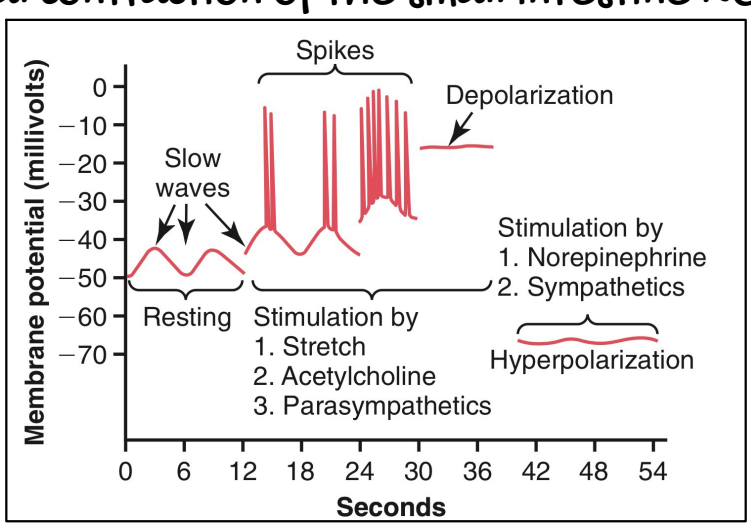
[Spike potentials are stimulated by] :

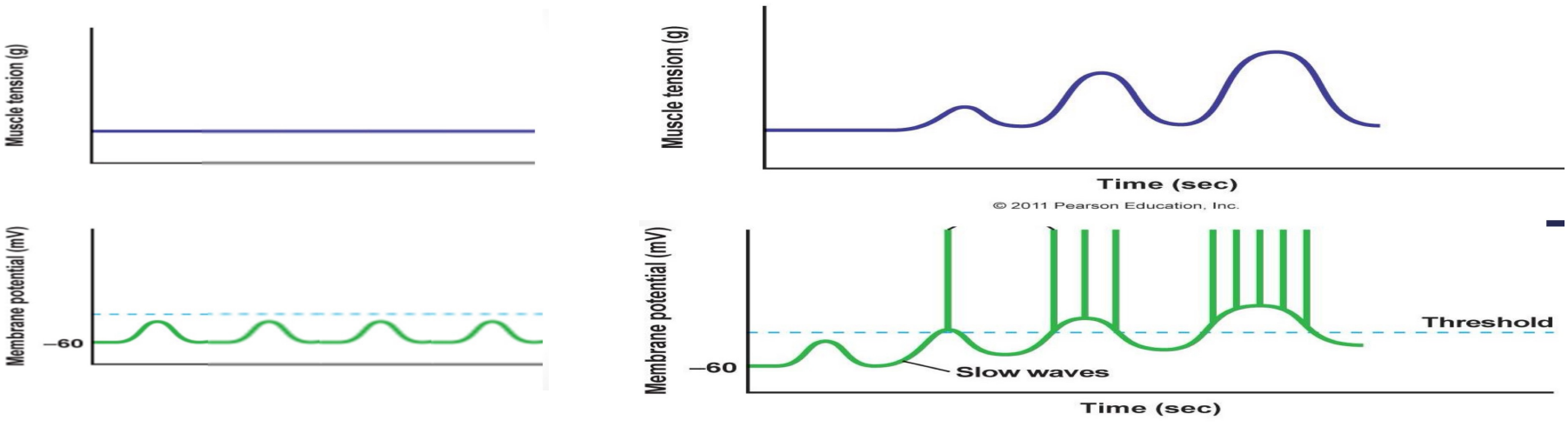
[Stretch \ Acetylcholine \ Some GI hormones]

The higher the slow wave potential rises, the greater the frequency of the spike potentials (1-10/sec)

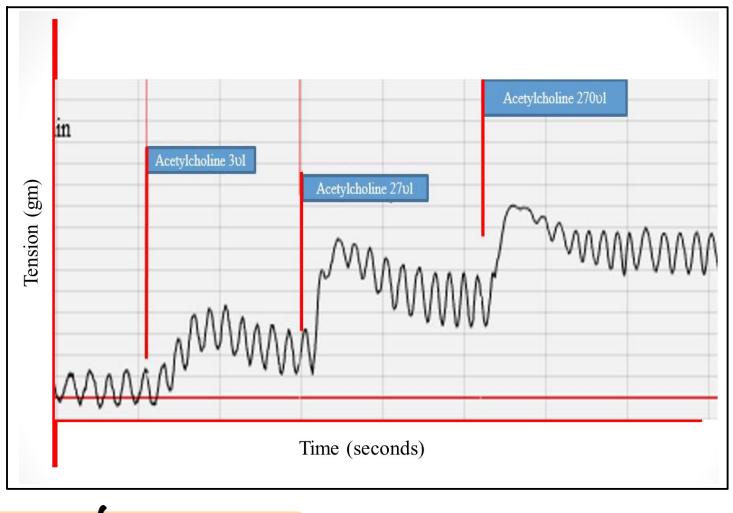
- Remember that in our experiment we measured the actual contraction of the small intestine NOT the slow waves .







Effect of Acetylcholine (ACh) :



ACh is the main excitatory neurotransmitter in the small intestine.

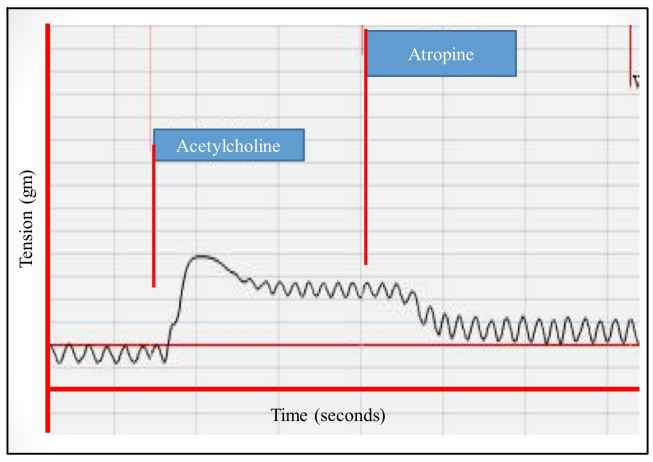
Released by:

- Enteric neurons
- Parasympathetic vagal fibers

[[It increases the contractile force of intestinal smooth muscle by]] :

- Increasing the number of spike potentials NOT by changing slow wave frequency .
- ACh acts via muscarinic receptors (M3) on smooth muscle cells.

Effect of Atropine :



- Atropine is a muscarinic receptor antagonist
- [competitive antagonist]
- It blocks ACh from binding to muscarinic receptors.

[[Result: Inhibition of ACh-induced contractions → intestinal motility decreases]] .

Atropine helps confirm that ACh's effects are receptor-mediated.

[[What You Measured in the Experiment]] :

You did not measure slow waves. You measured actual mechanical contractions, which are the result of spike potentials superimposed on slow waves.

Important Summary Points:

Feature	Acetylcholine (ACh)	Atropine
Type	Excitatory NT	Muscarinic antagonist
Effect	↑ Spike potentials → ↑ Contractions	Blocks ACh → ↓ Contractions
Acts on	Muscarinic receptors (M3)	Blocks M3 receptors
Stimulates	Parasympathetic/ENS release	Used pharmacologically

The Graphs show :

1) An increase in contraction amplitude after ACh.
2) Followed by a decrease or flat baseline after atropine addition.

Take a Home massage

- ACh enhances intestinal motility by increasing the number of spike potentials.
- Atropine inhibits this effect, proving that muscarinic receptors mediate ACh's action.

1. What is the main purpose of adding acetylcholine in the intestinal motility experiment?

A) To inhibit intestinal contractions
B) To stimulate spike potential generation
C) To decrease slow wave frequency
D) To block muscarinic receptors
→ **Correct answer: B)** To stimulate spike potential generation
Explanation: Acetylcholine increases the number of spike potentials, enhancing contraction force.

2. Which cells act as pacemakers for intestinal smooth muscle contractions?

A) Parietal cells
B) Enterocytes
C) Interstitial cells of Cajal
D) Goblet cells
→ **Correct answer: C)** Interstitial cells of Cajal
Explanation: These cells generate slow waves that set the rhythm of GI contractions.

3. What type of contraction pattern is normally seen in the small intestine?

A) Tonic contractions
B) Spasmodic contractions
C) Phasic (rhythmic) contractions
D) Tetanic contractions
→ **Correct answer: C)** Phasic (rhythmic) contractions
Explanation: The experiment focused on rhythmical, periodic contractions typical of the small intes

4. What is the primary effect of atropine in the experiment?

A) Stimulates contraction
B) Increases slow wave frequency
C) Blocks muscarinic receptors
D) Enhances ACh release
→ **Correct answer: C)** Blocks muscarinic receptors
Explanation: Atropine is a competitive antagonist of ACh at muscarinic receptors, reducing contrac

5. What is the electrical basis for triggering a contraction in smooth muscle?

A) Resting potential reaching –70 mV
B) Spike potentials superimposed on slow waves
C) Release of ATP from mitochondria
D) Calcium release from enterocytes
→ **Correct answer: B)** Spike potentials superimposed on slow waves
Explanation: A contraction occurs when a spike potential is generated on the peak of a slow wave.

6. How does acetylcholine affect the frequency of slow waves?

A) Increases it
B) Decreases it
C) Has no effect
D) Stops slow waves completely
→ **Correct answer: C)** Has no effect
Explanation: ACh does not affect slow wave frequency; it increases **spike potential frequency**.

7. What happens when the peak of a slow wave becomes more positive than –40 mV?

A) Nothing happens
B) Contraction is inhibited
C) A spike potential is generated
D) ATP is synthesized
→ **Correct answer: C)** A spike potential is generated
Explanation: This threshold triggers true action potentials, leading to contraction.

8. What did the experiment measure directly?

A) Frequency of slow waves
B) Strength of spike potentials
C) Actual muscle contraction force
D) Membrane potential changes
→ **Correct answer: C)** Actual muscle contraction force
Explanation: The tension transducer measured mechanical contractions, not electrical activity.

9. What is required in the organ bath to maintain tissue viability?

A) Ice-cold water
B) Carbon dioxide
C) Warm, oxygenated buffer
D) Ethanol
→ **Correct answer: C)** Warm, oxygenated buffer
Explanation: The buffer must be at 37°C and oxygenated to mimic physiological conditions.

10. Which receptor type does acetylcholine act on to promote contraction in the small intestine?

A) Alpha-adrenergic
B) Nicotinic
C) Muscarinic
D) Serotonin
→ **Correct answer: C)** Muscarinic
Explanation: ACh promotes contraction via **muscarinic receptors**, particularly M3, in smooth muscl

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