Intestinal Motility Experiment

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Aim of the experiment

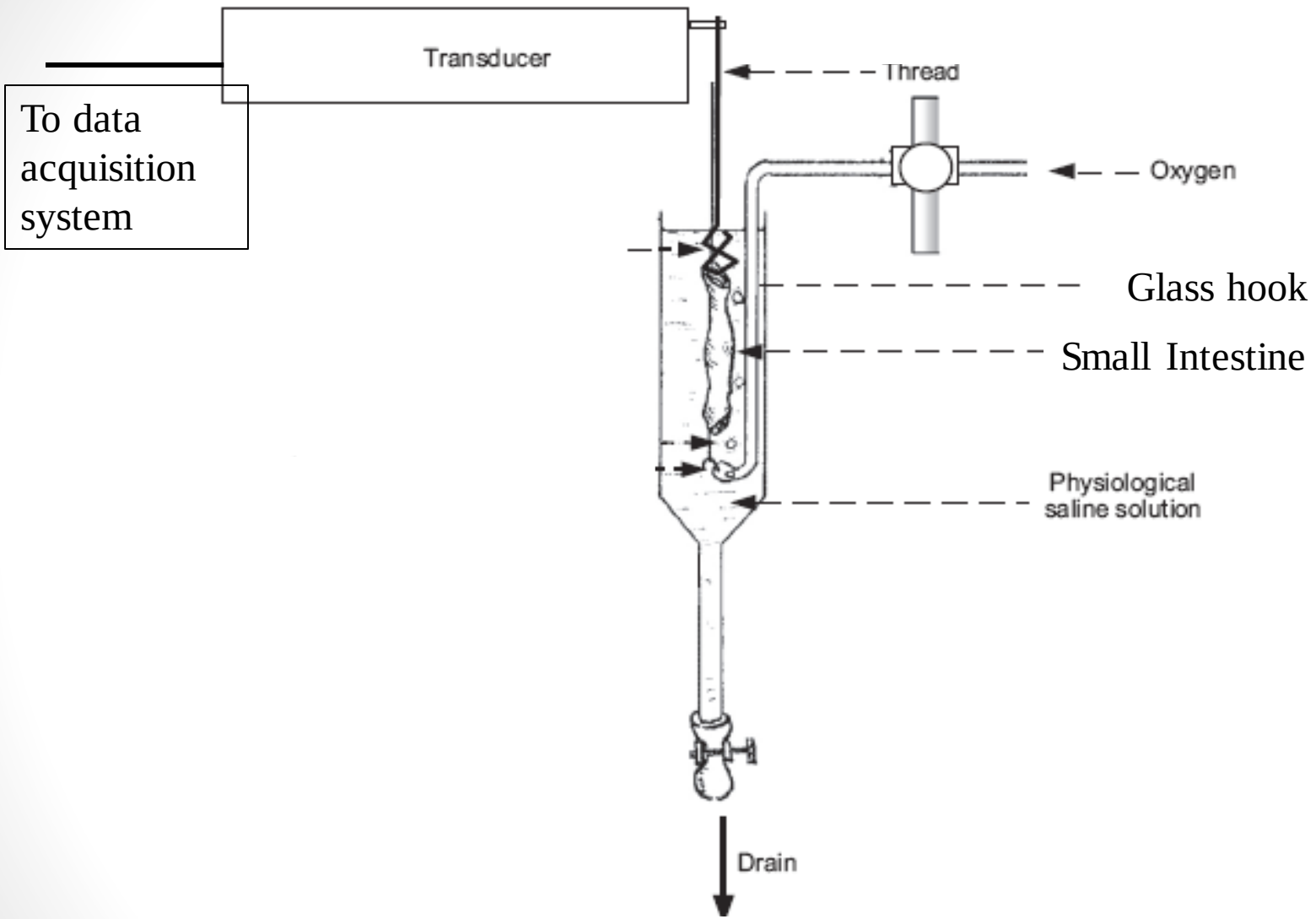
- This experiment investigates the contraction of smooth muscle in the small intestine by :

1. Observing the occurrence of rhythmical contractions
2. The modification of these contractions by acetylcholine and atropine.

Method

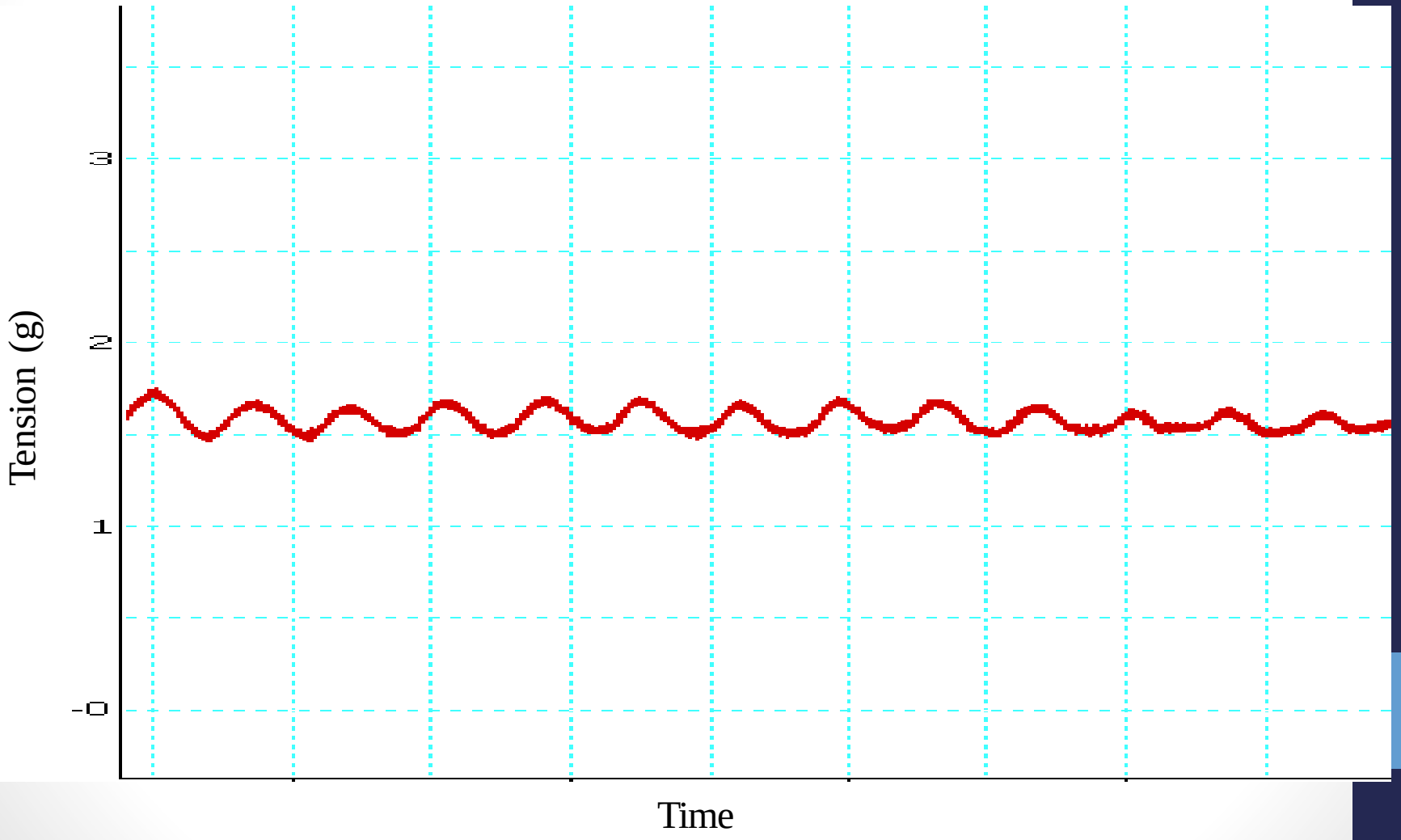
- In our experiment we use the small intestine (SI) of the rat.
- Small pieces (2-3cm) of the SI are hanged vertically by a thread to a glass hook in an organ bath.
- The organ bath contains warm (37^oC) oxygenated buffer. This is essential to maintain the viability of the tissue.
- The SI is connected by a thread to a tension transducer
- The tension transducer converts the mechanical signal generated by the contraction of the small intestine to an electric signal and conveys it to a special software
- The software is capable of displaying a simple graph of tension versus time.

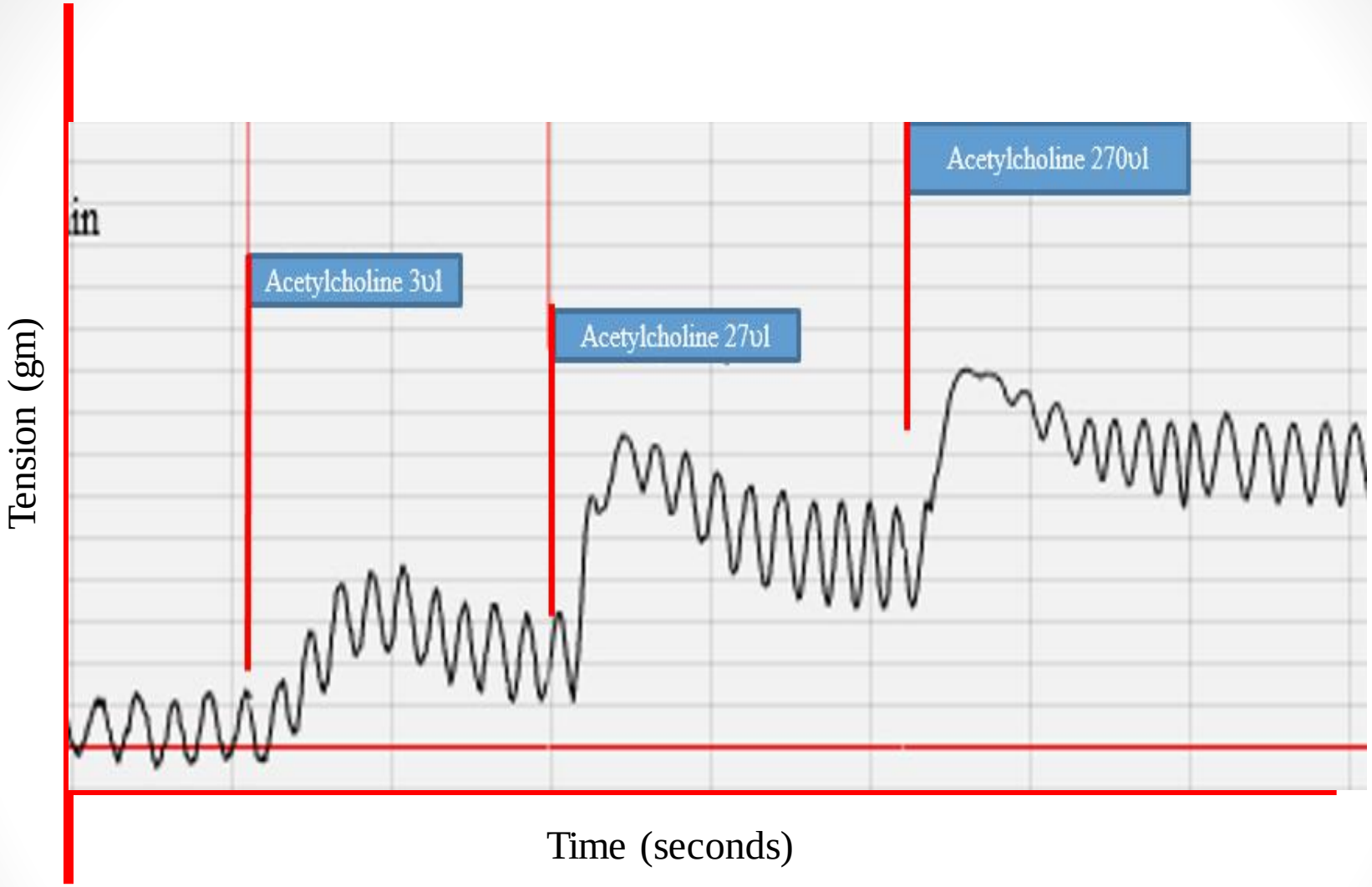
- After hanging the tissue it is allowed to rest for 15-20 minutes to allow the muscle to recover normal function after being handled.
- The tension created by the small intestinal segment is recorded.
- Then Acetylcholine is added to the organ bath.
- Finally Atropine is added to the organ bath.

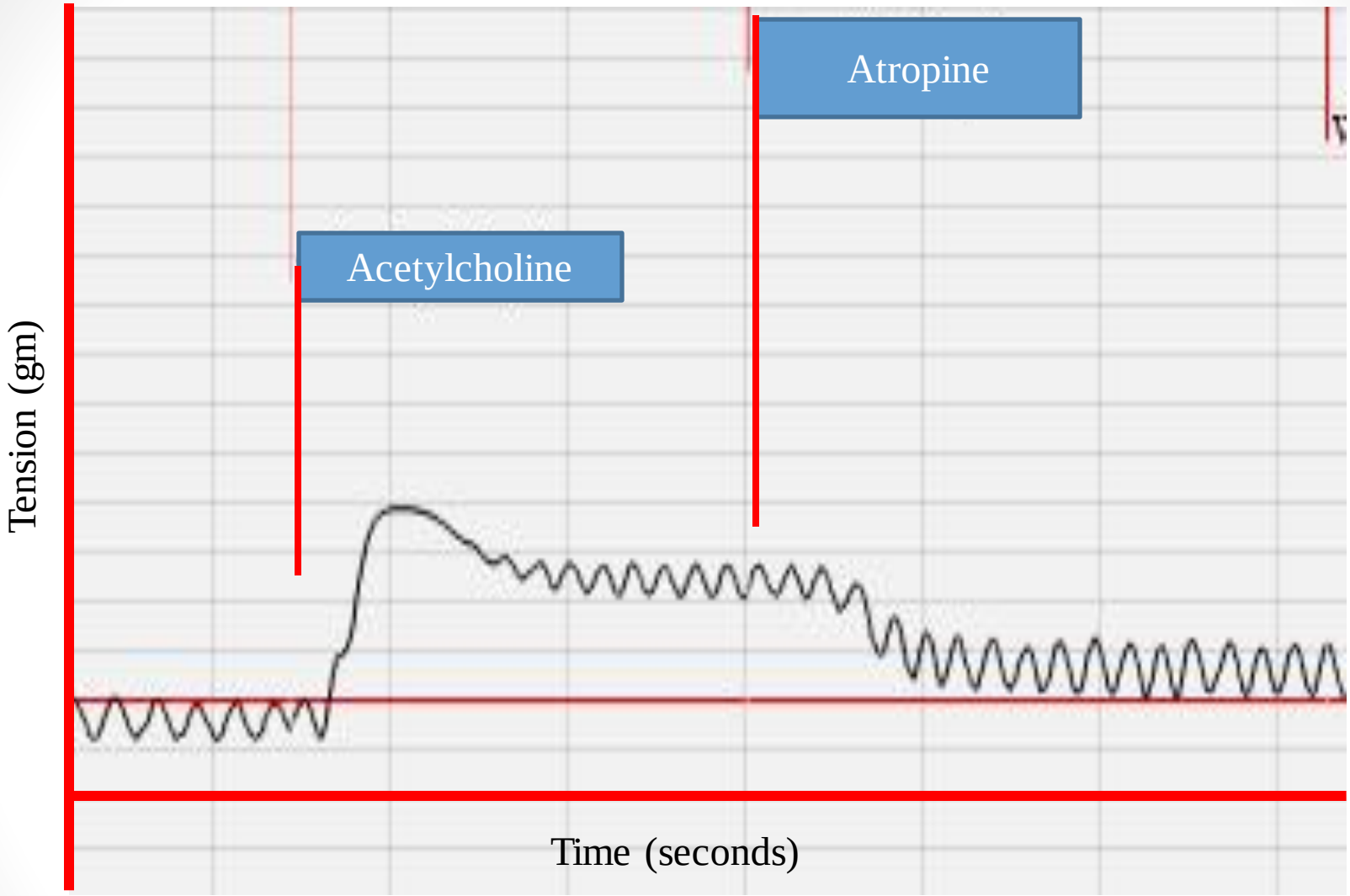


Arrangement of the organ bath, tissue, and pressure transducer.

Results



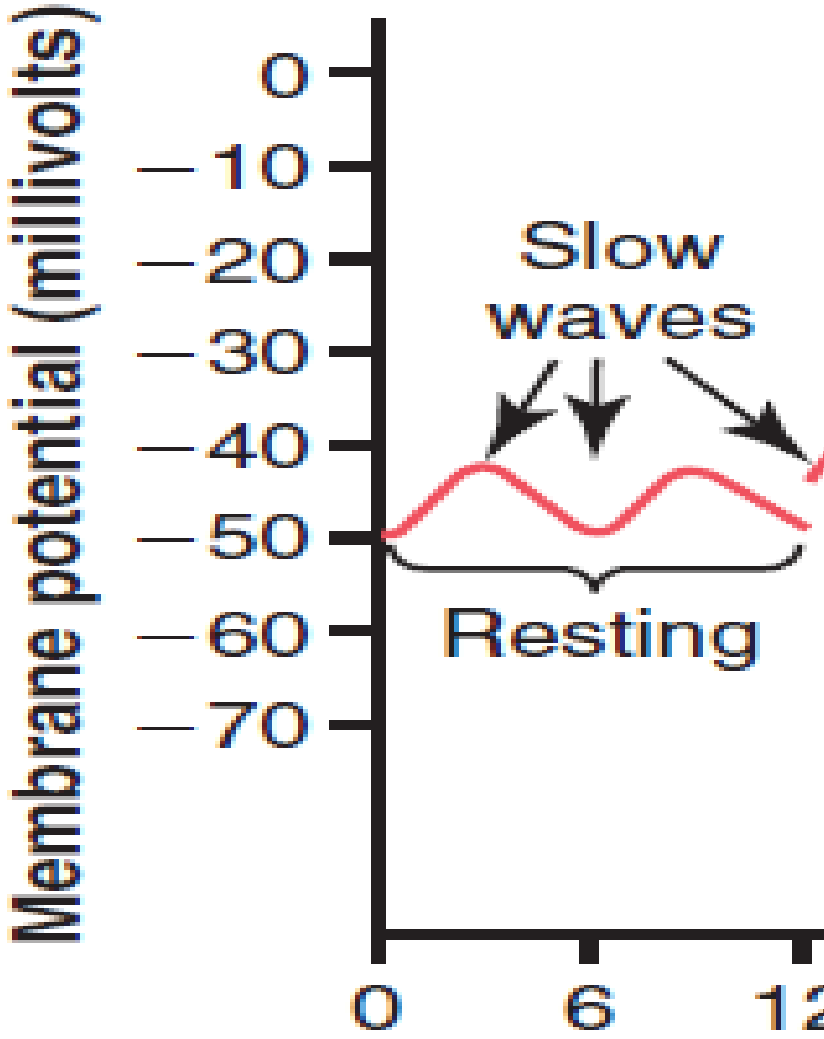

Tension (gm)
in
Acetylcholine 3υl
Acetylcholine 27υl
Acetylcholine 270υl
Time (seconds)

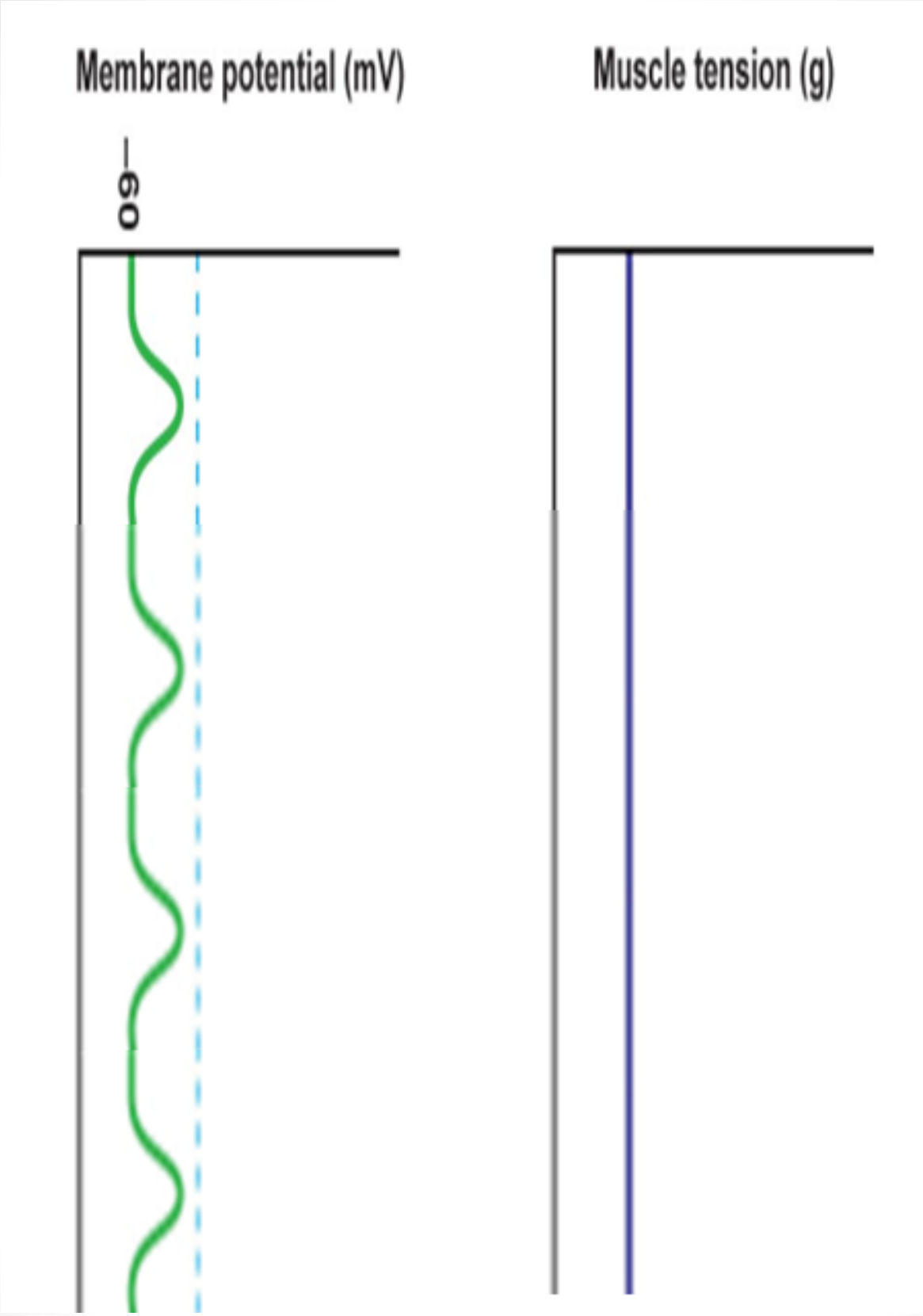

Atropine
Acetylcholine
Tension (gm)
Time (seconds)

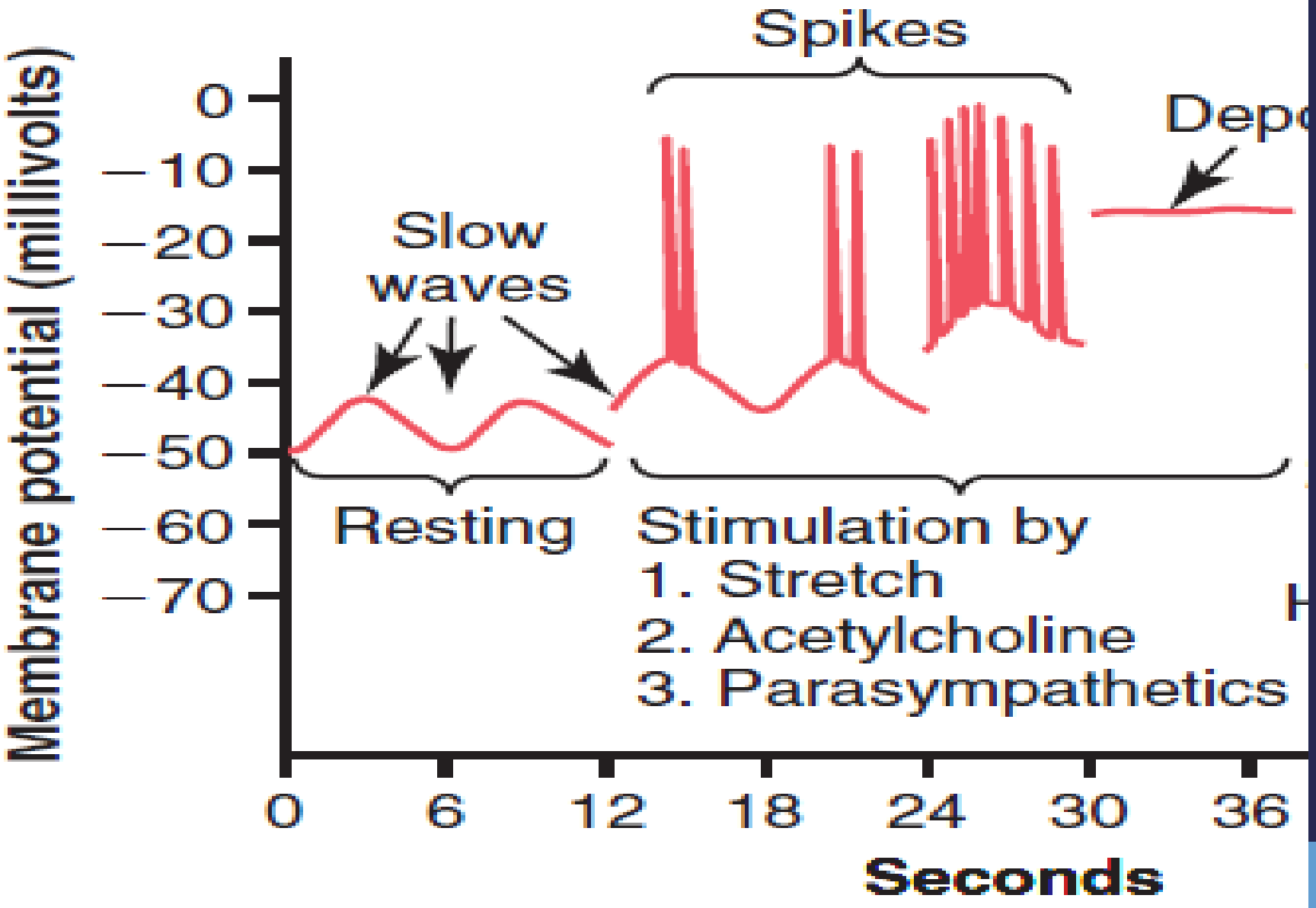
Discussion

- Phasic (Rhythmical)contractions: periodic contractions and relaxations.
- Most gastrointestinal contractions occur rhythmically.
- Smooth muscle fibers in the small intestine contracts rhythmically in the absence of neuronal or hormonal stimulation.
- The rhythm is determined mainly by the frequency of the “slow waves” .
- The slow waves are generated by the interstitial cells of Cajal (ICC), which are believed to act as electrical pacemakers for smooth muscle cells.

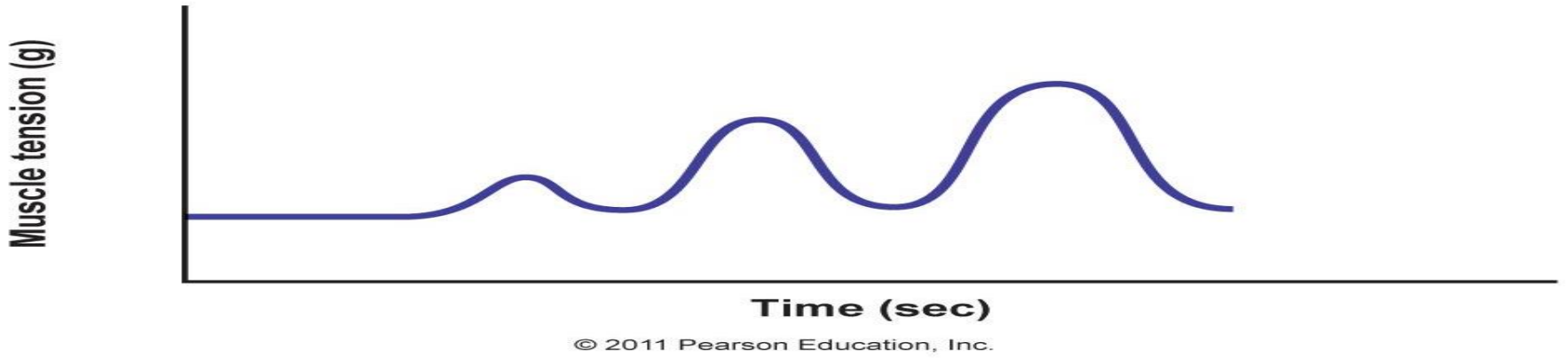
- Slow waves are slow, undulating changes in the resting membrane potential.
- Slow waves occur at different frequencies at various points along the gastrointestinal tract. In humans their frequency is 12/minute in the duodenum, 8-9/minute in the ileum.
- Slow waves set the maximum frequency at which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

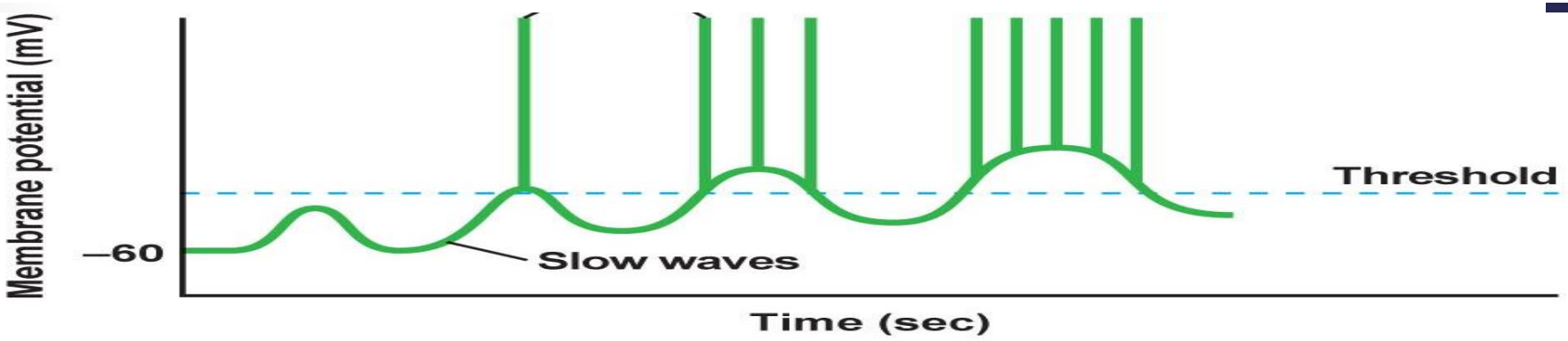



Membrane potential (mV)
-60
Muscle tension (g)

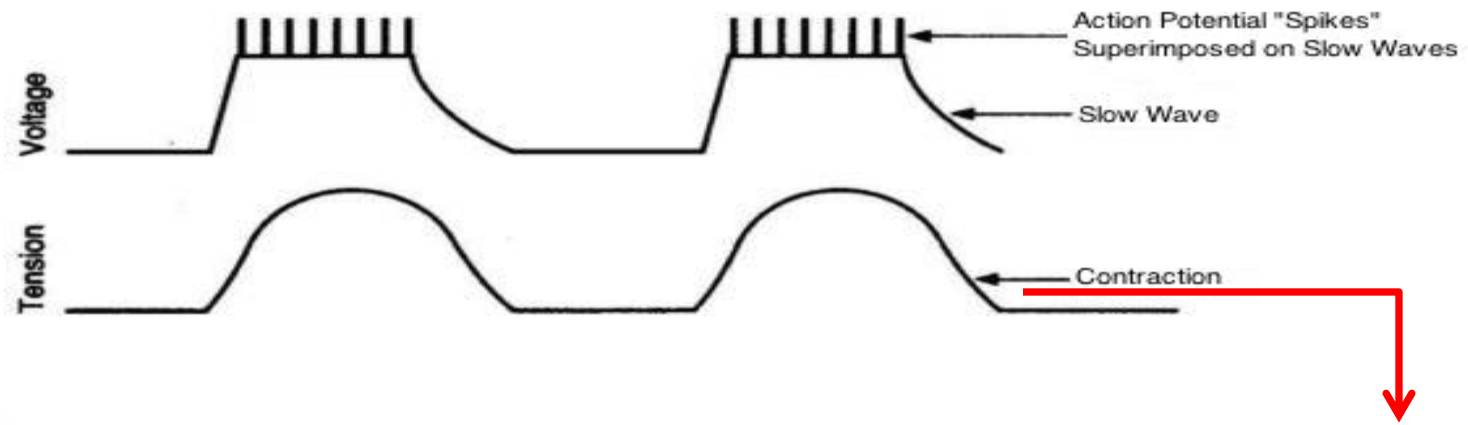

Spikes
Depo
Slow
waves
Membrane potential (millivolts)
0
−10
−20
−30
−40
−50
−60
−70
Resting
Stimulation by
1. Stretch
2. Acetylcholine
3. Parasympathetics
0
6
12
18
24
30
36
Seconds

- 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.
  - They are true action potentials
  - Stimulated by stretch, acetylcholine and some GI hormones
  - Appear when the peaks of the slow waves temporarily become more positive than −40mV.
  - 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 .



- Ach is the major excitatory neurotransmitter in the small intestine
- Secreted by enteric neurons and parasympathetic neurons
- Acetylcholine promotes increased contractile force
  - The increase in contractile force is due to an increase in the number spikes not in the frequency of slow waves.

- Its effect on intestinal smooth muscle cells is mediated through muscarinic receptors
  - Inhibition of the contractile effect of ACh is mediated by adding atropine; a competitive antagonist of Ach at the muscarinic receptor.