Intestinal Motility Experiment

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As we already know, we have 2 types of contractions in the GI tract:

1) Tonic contractions, and these occur only at the sites of sphincters
2) Rhythmic/Phasic contractions, and we're trying to see how they happen in this experiment

Remember, we talked about slow waves, which are maintained by Interstitial Cells of Cajal (ICC), these slow waves are not true action potentials, they're only undulating changes of resting membrane potential, and **they don't cause contractions by themselves!**

The rhythmic contractions occur by having the "always happening" changes of resting membrane potential without reaching the threshold and causing any of these contractions, and on top of them, the "Spike potnetials" these are true action potentials that happen on top of a slow wave causing a contraction, the signal for initiating a spike potential can be caused by:

1) Stretch
2) Chemicals; including Ach, certain Gi hormones like Motilin

And as we said that spike potentials happen on top of the slow waves, then we can say that these slow waves set the frequency at which contractions can happen!

Slow waves are fixed and is different depending on:

1)The part of the Gi tract
2)The species

As an example, In humans, the slow waves frequency in the duodenum is 12 per minute, the terminal ileum's frequency is 8-9 per minute, and in other species like rats, it can reach upto 25-30 per minute!

Aim of the experiment

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

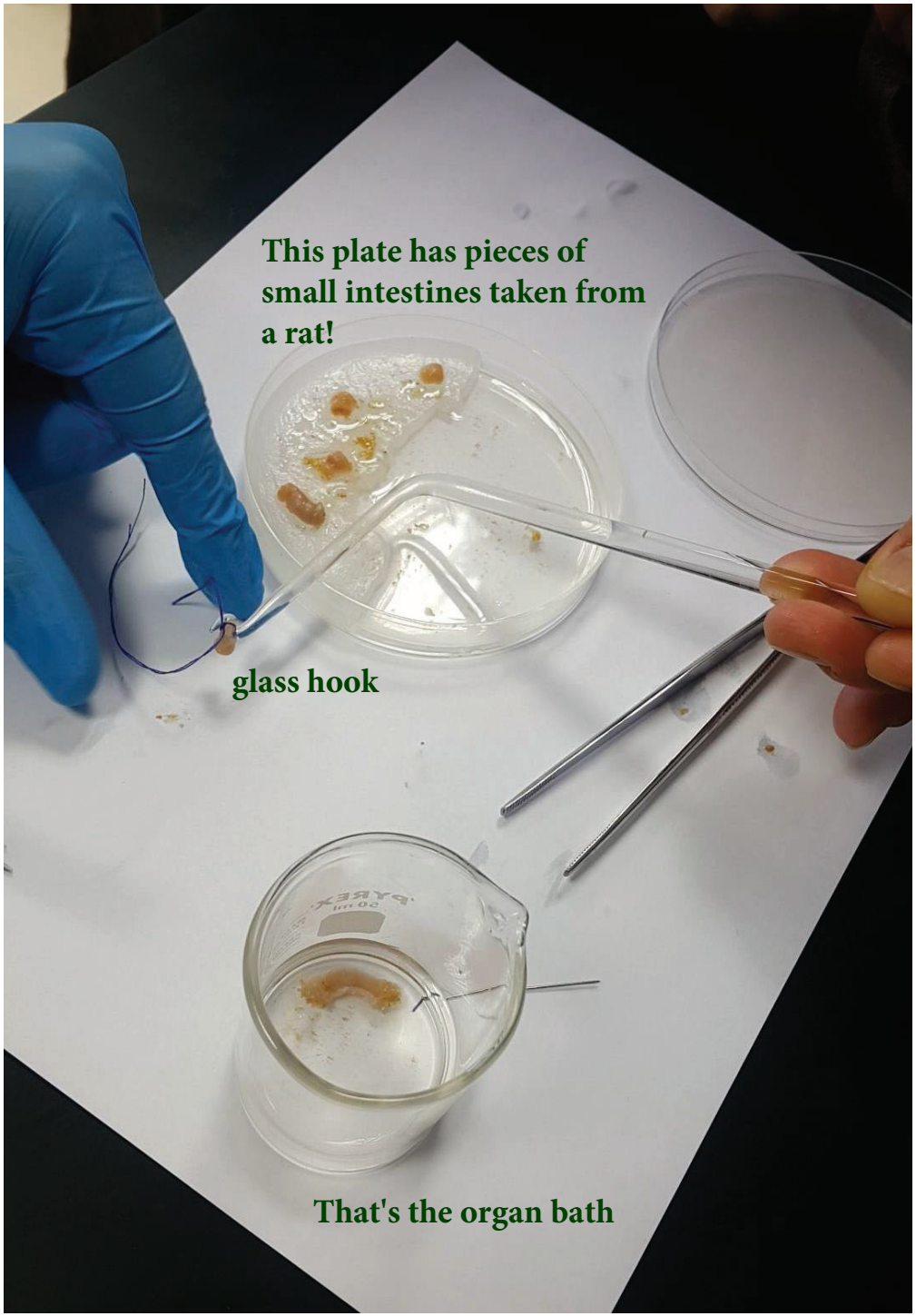
1. Observing the occurrence of rhythmical contractions
2. The modification of these contractions by acetylcholine and atropine.

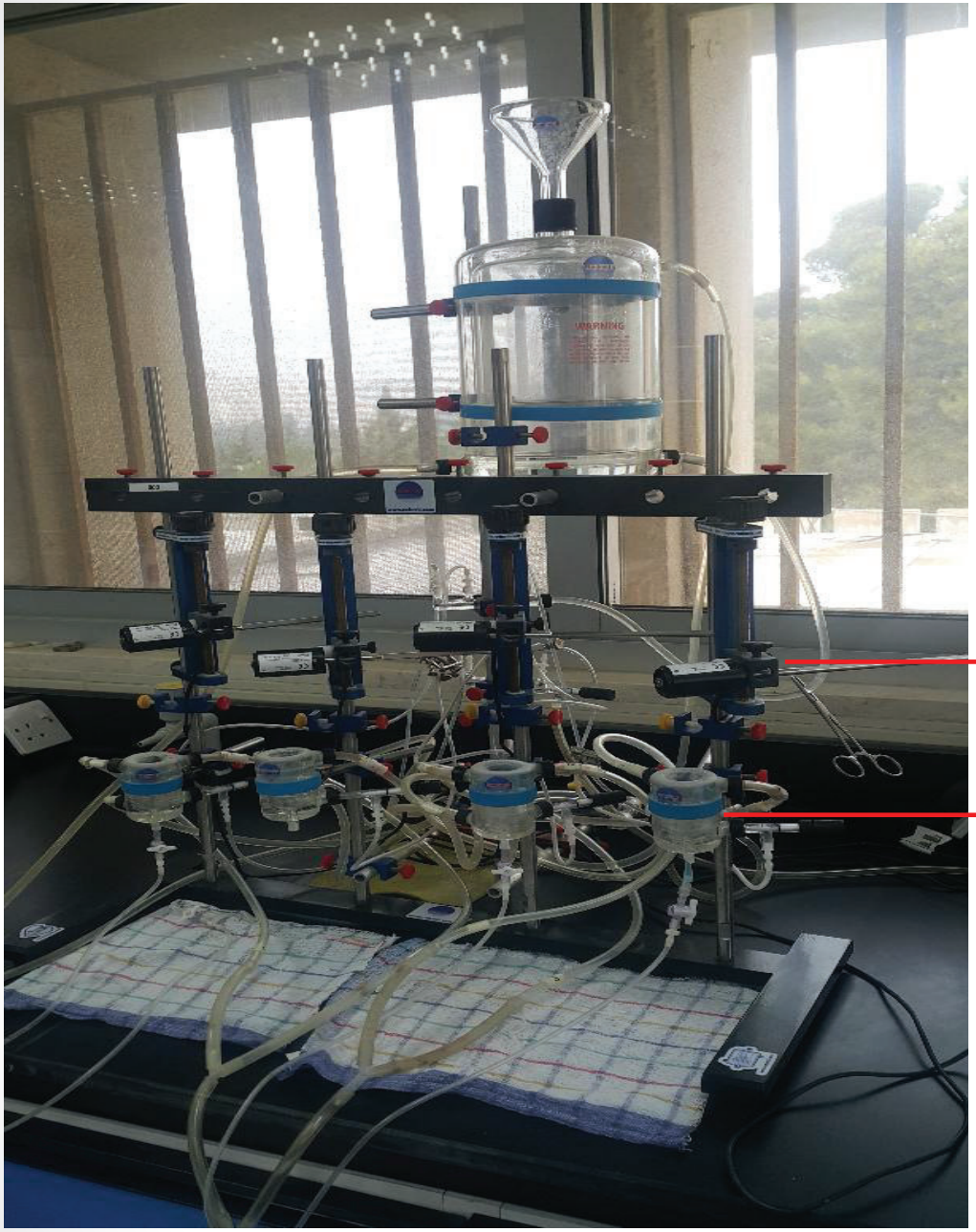
Method

- 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°C) 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.

An organ bath is a solution that contains certain nutrients and salts, and it's used to maintain the viability of the tissue for some time, it's also usually oxygenated and heated to the perfect temperature for the same underlined reason we just mentioned.

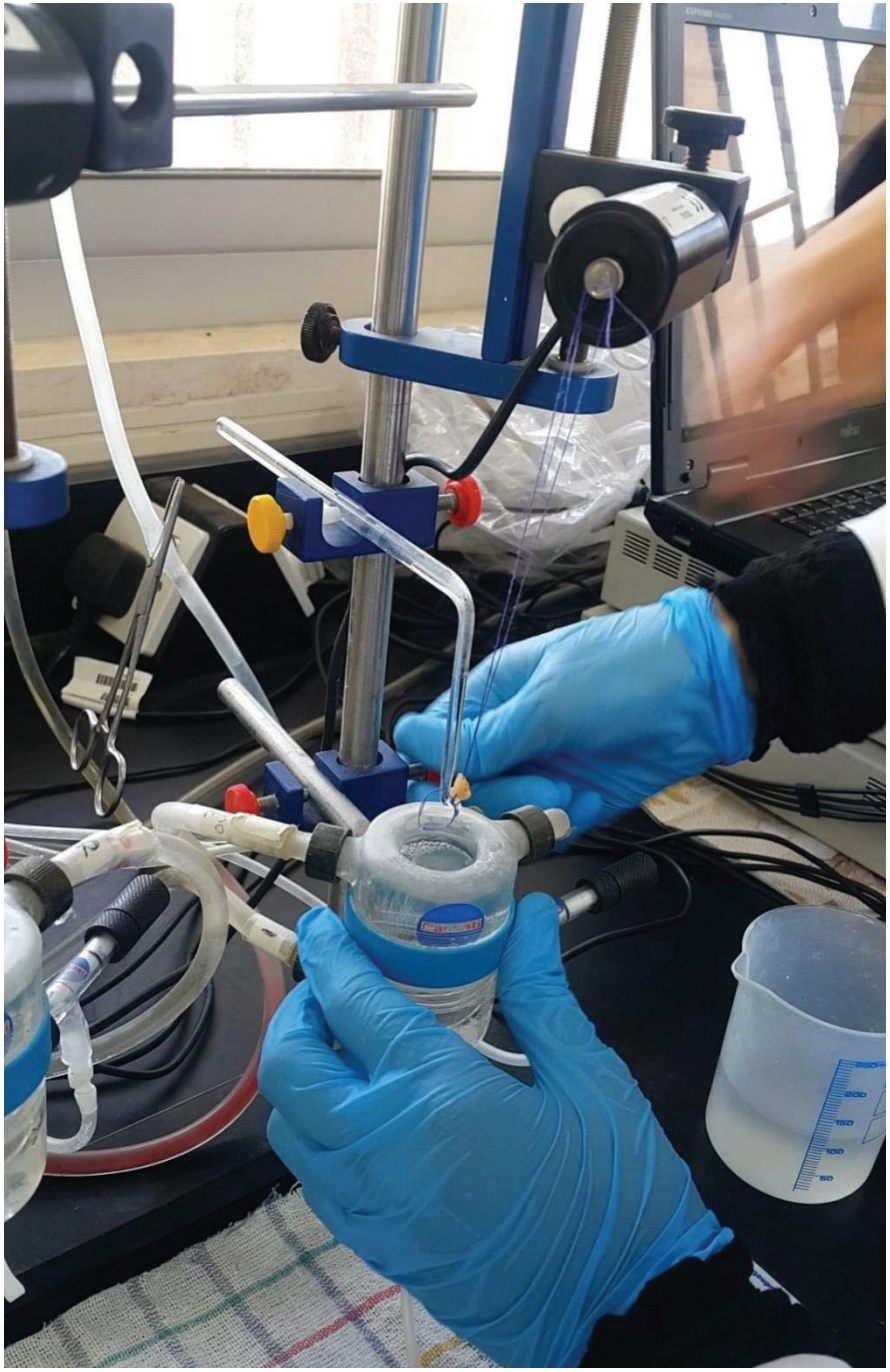
- After hanging the tissue it is allowed to rest for 15-20 minutes to allow the muscle to recover normal function after being handled.
- The tension created by the small intestinal segment is recorded.
- Then Acetylcholine is added to the organ bath.
- Finally Atropine is added to the organ bath.


This plate has pieces of small intestines taken from a rat!
glass hook
That's the organ bath

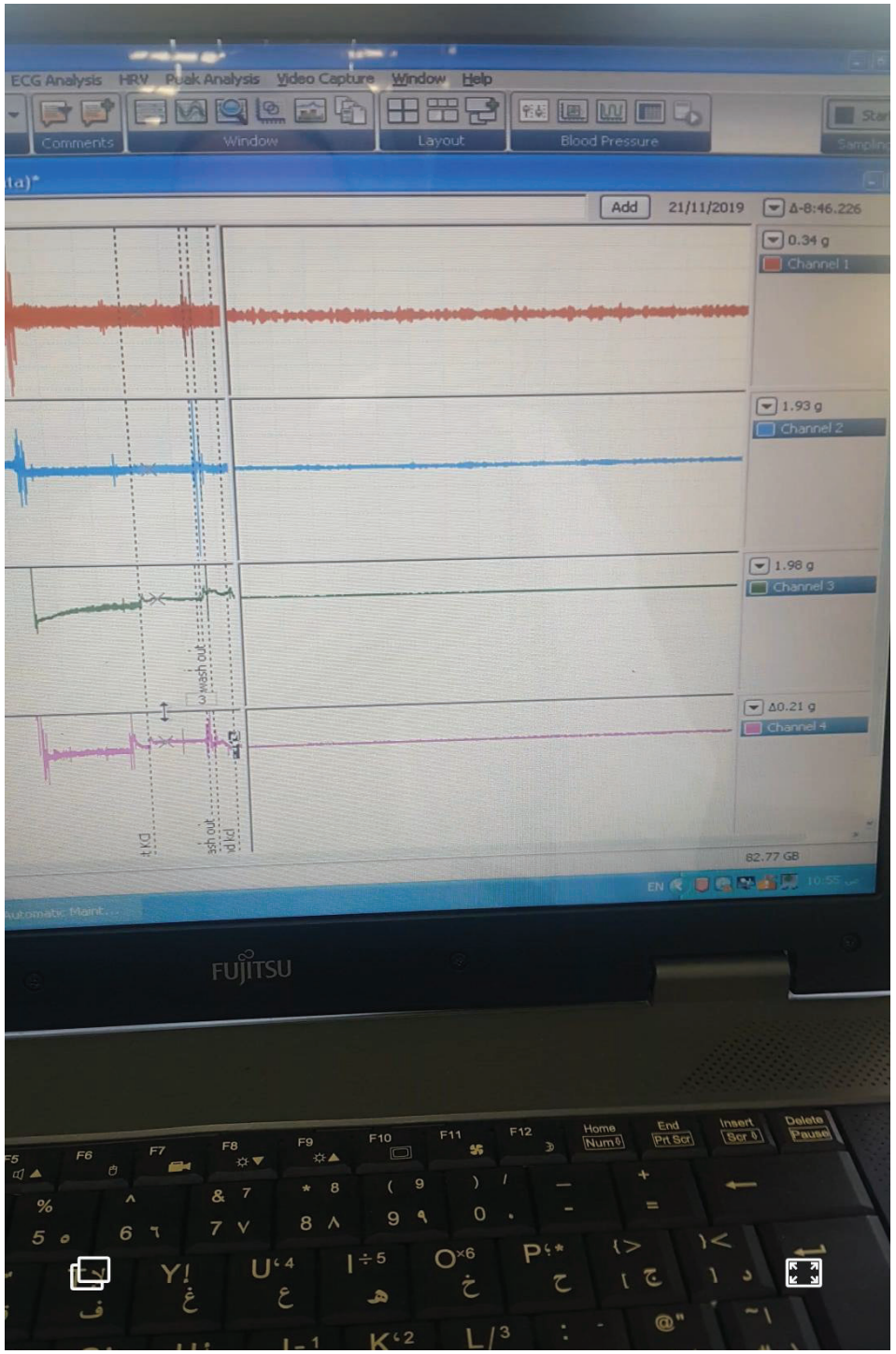


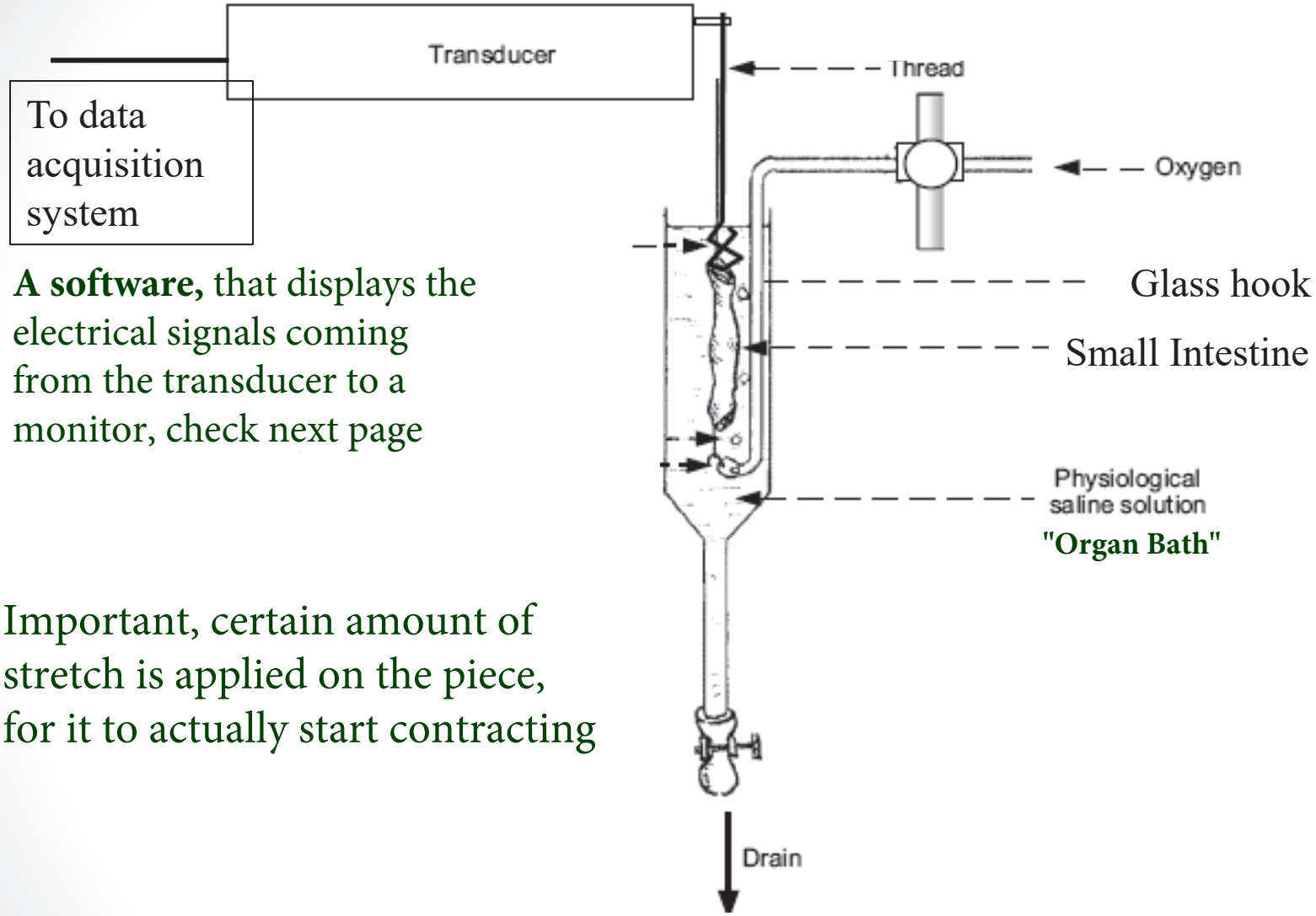
Tension transducer

Organ bath



That's the software displaying the output from the transducer





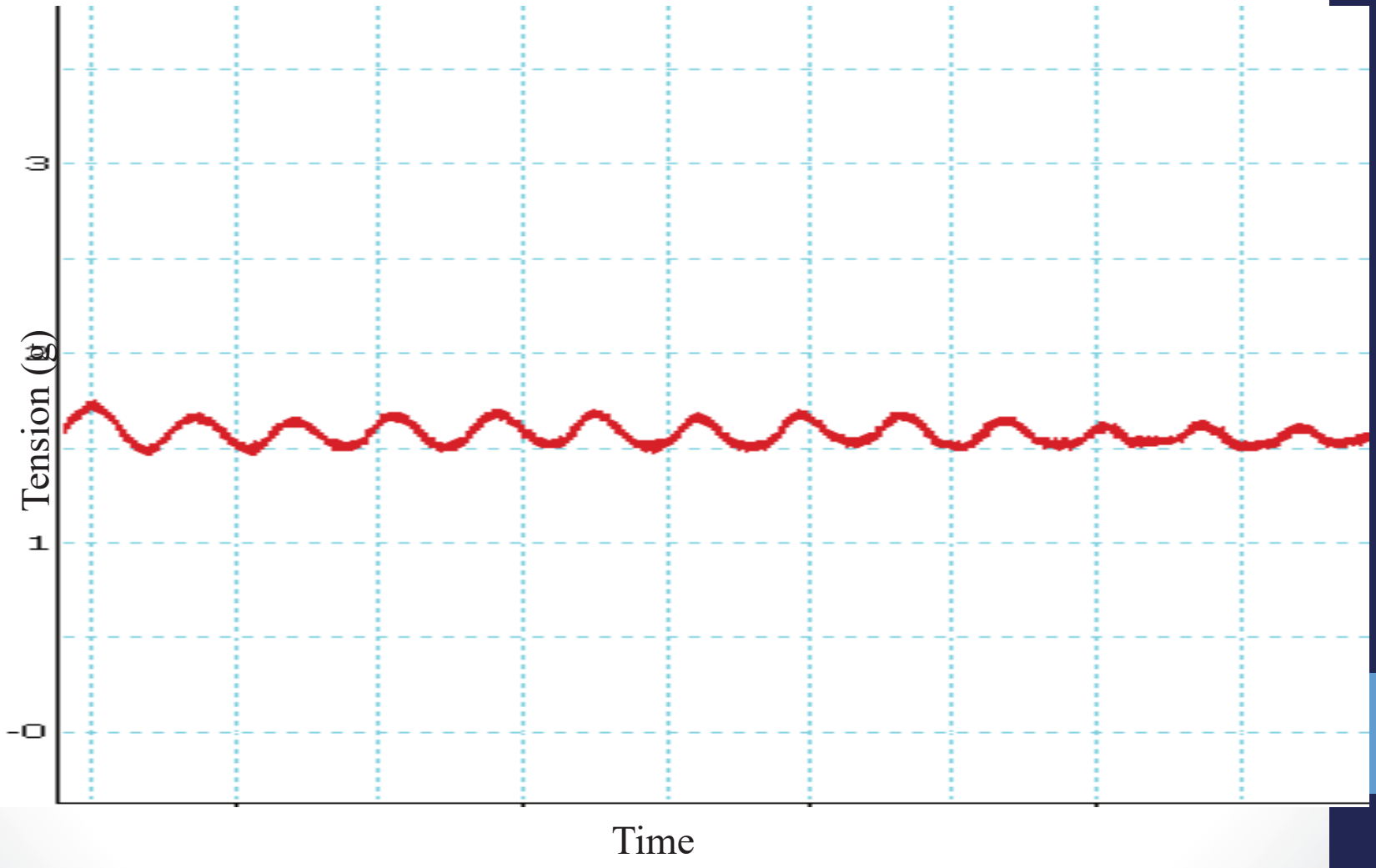
A software, that displays the electrical signals coming from the transducer to a monitor, check next page

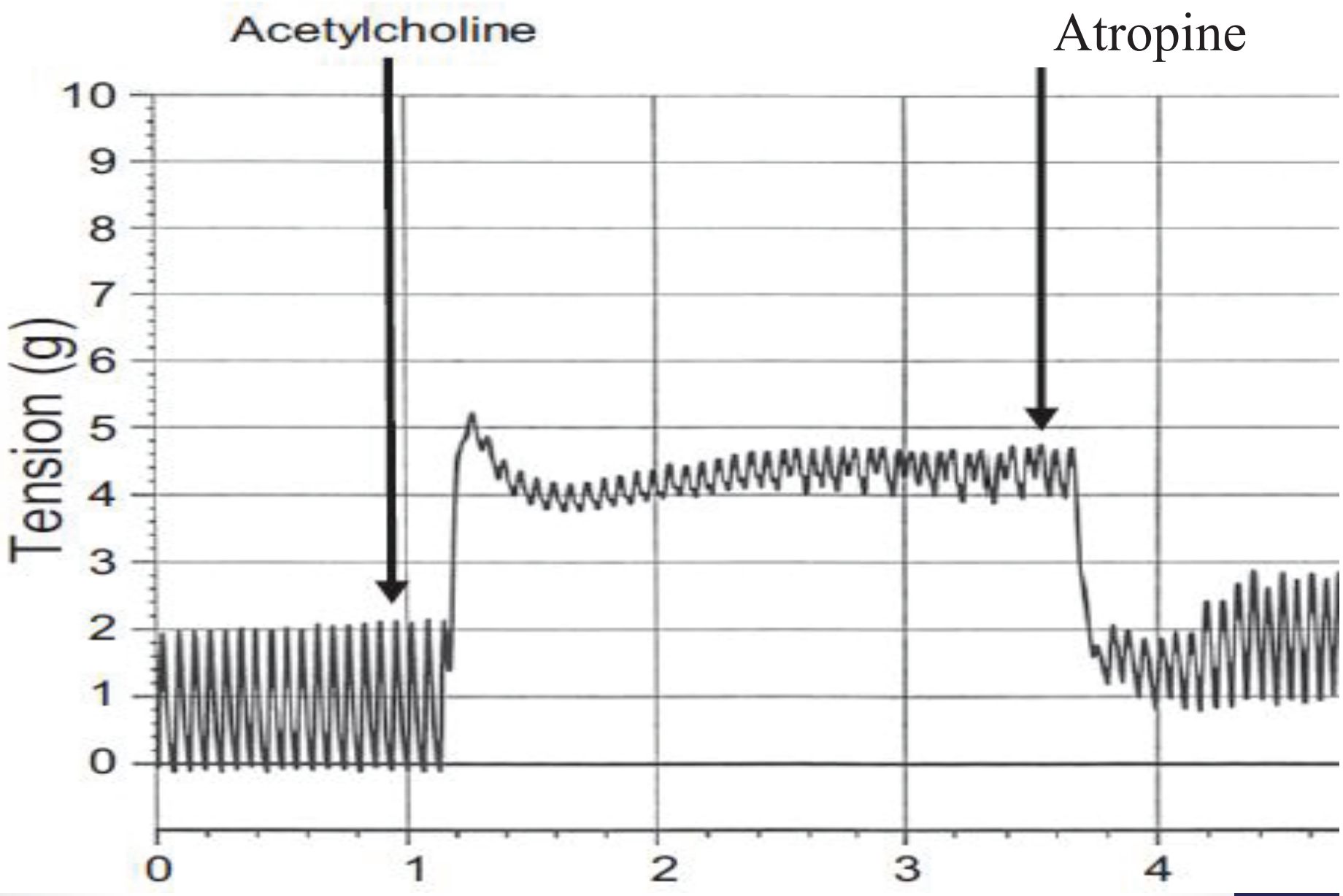
Important, certain amount of stretch is applied on the piece, for it to actually start contracting

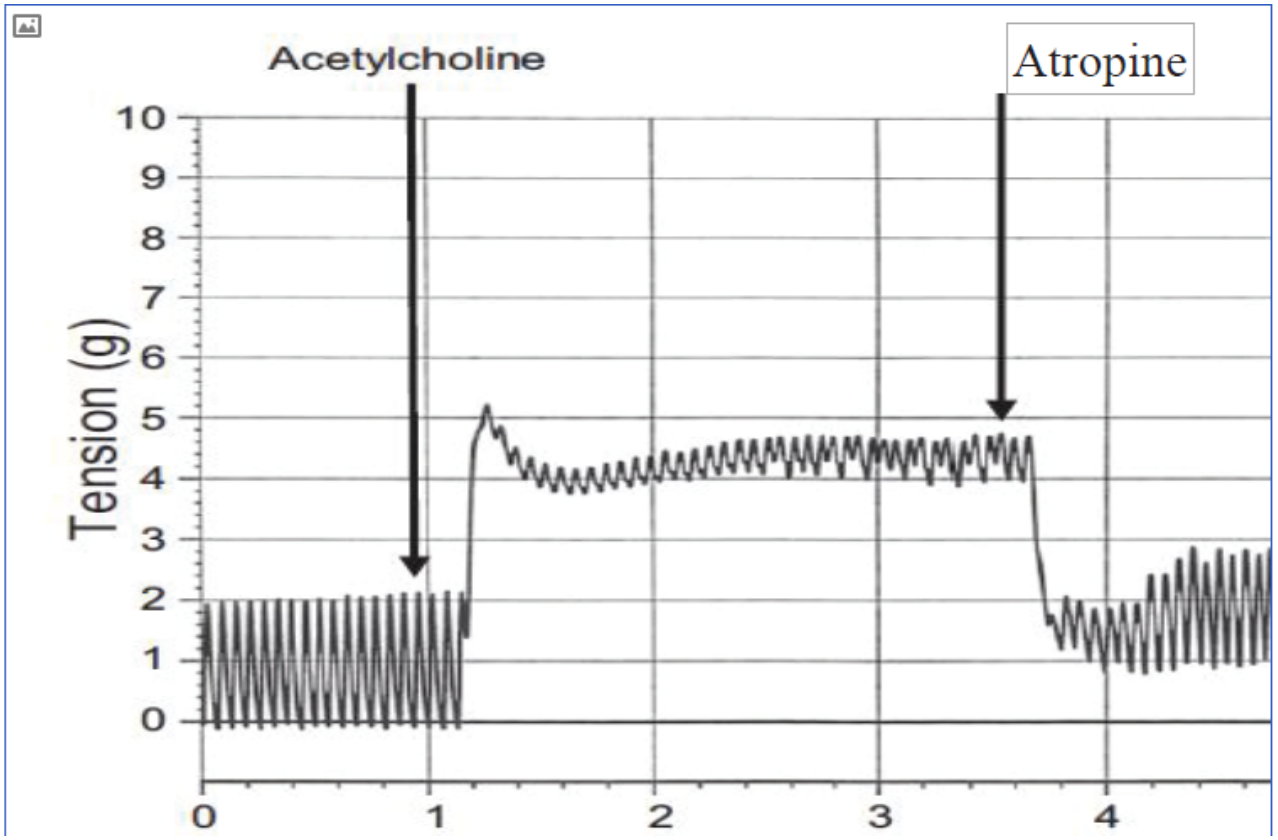
Arrangement of the organ bath, tissue, and pressure transducer.

Results

As you can see, in this experiment, what we are measuring is the **tension** (The contractile force from that piece of intestine)




Acetylcholine
Atropine
Tension (g)
10
9
8
7
6
5
4
3
2
1
0
0
1
2
3
4



Note: The doctor said that the only goal of this experiment is to observe the general effect of Ach and Atropine on the tension generated by this piece of small intestine, so don't focus on the small details like why the contractile waves are actually narrower after adding Ach, or why the tension didn't go back to the same point it was at before adding Ach, just focus that Ach effect is excitatory, meaning that the tension will increase after it's addition, and of-course adding a competitive antagonist of Ach will have the reverse effect; decreasing the tension.

What do you expect would happen if we added epinephrine/nor epinephrine instead of Atropine?
We would have exactly the same effect as if we added atropine, yet on the rat's experiment we noticed that it doesn't respond to epinephrine or nor epinephrine as much (less sensitive), but if we took a piece from another species like a rabbit as an example it would have exactly the same response!

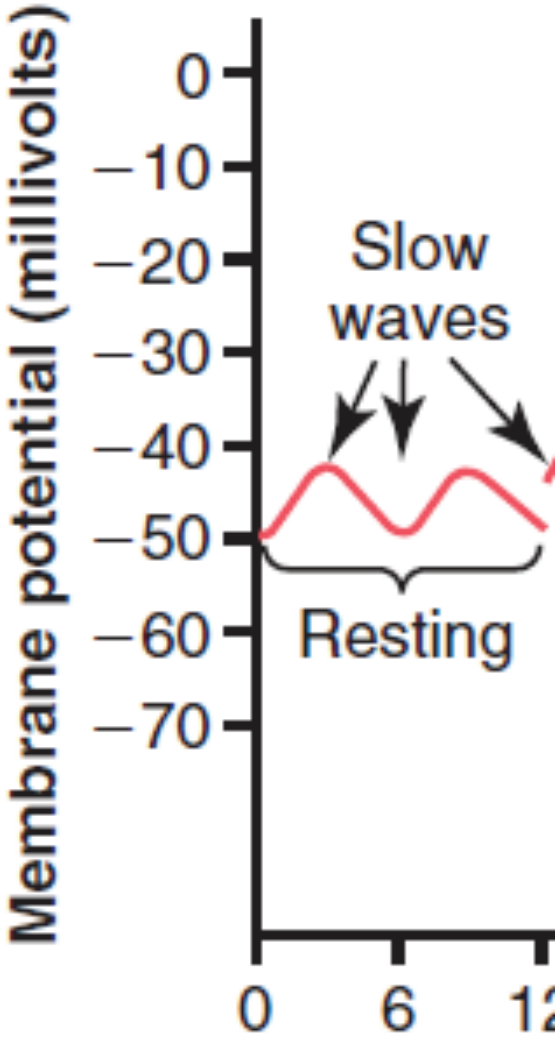
- Ach is the major excitatory neurotransmitter in the small intestine
- Secreted by enteric neurons and parasympathetic neurons
- Acetylcholine promotes increased contractile force
  - The increase in contractile force is due to an increase in the number spikes not in the frequency of slow waves.

- 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.

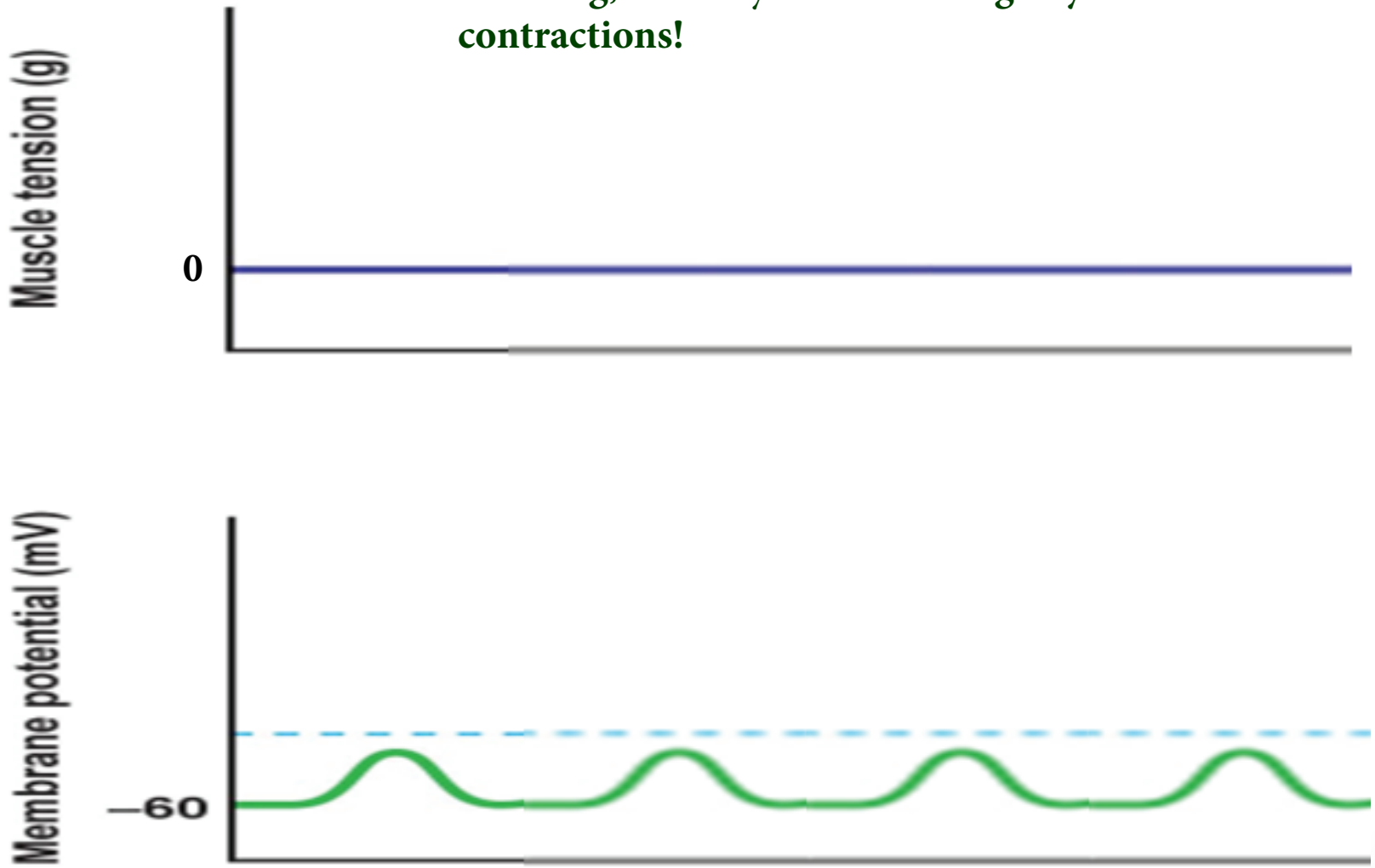
Discussion

- Most gastrointestinal contractions occur rhythmically.
- Phasic (Rhythmical)contractions: periodic contractions and relaxations.
- Smooth muscle 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.

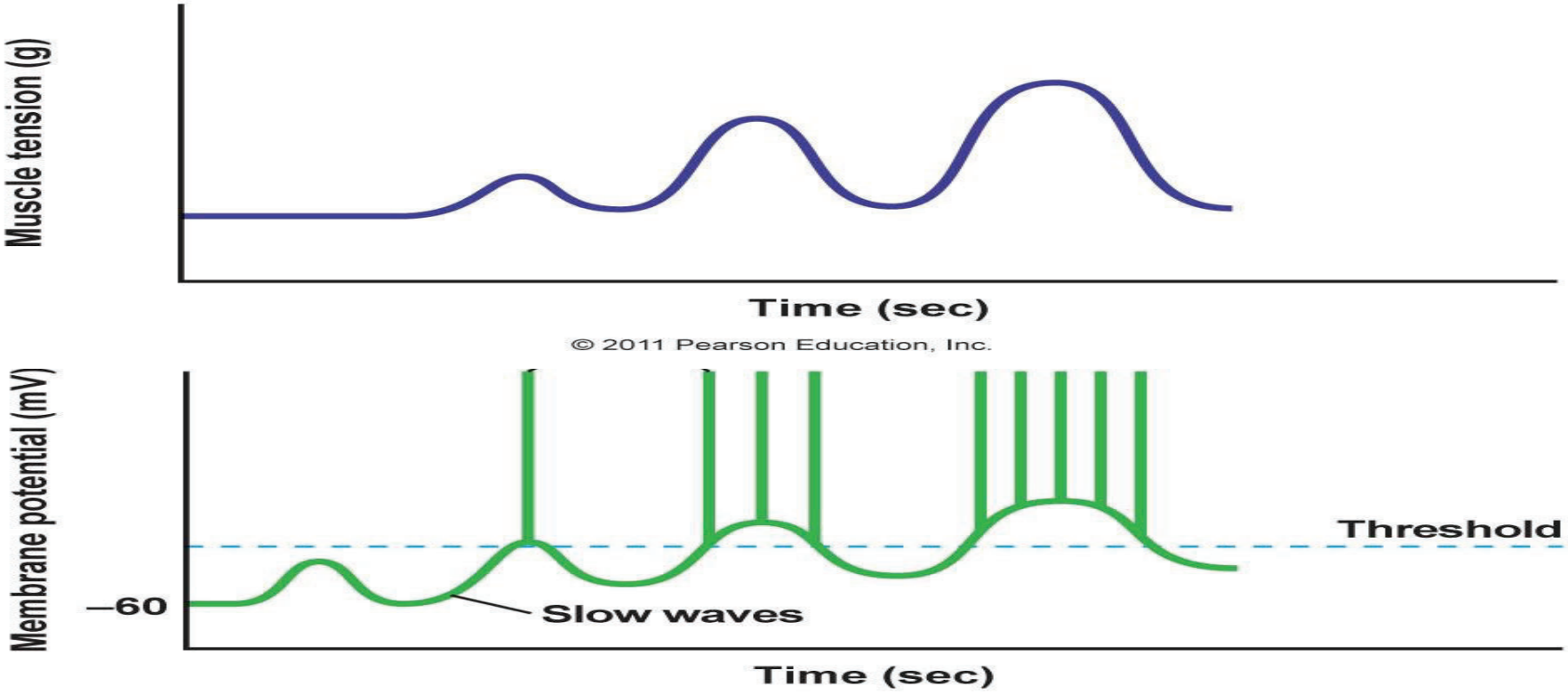


As you can see, slow waves are **always occuring**, and they're **not causing any contractions!**



- 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.
  - They are true action potentials
  - Stimulated by stretch, acetylcholine and some GI hormones

like **Motilin**

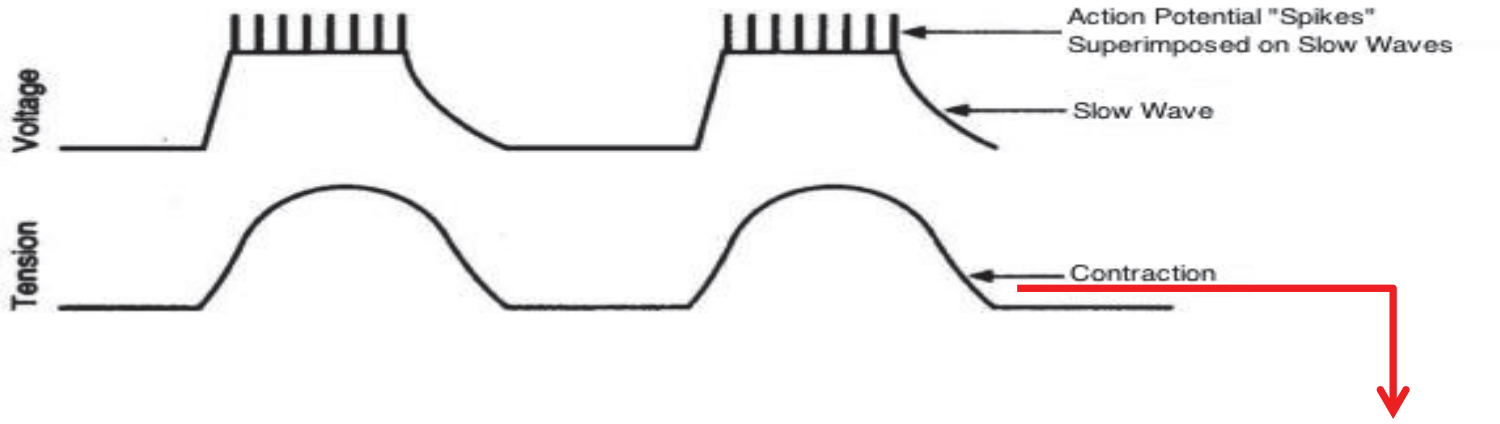


Increasing stretch, will increase the number of spike potentials, thus the force of contraction increases, slow waves are ALWAYS FIXED!,

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

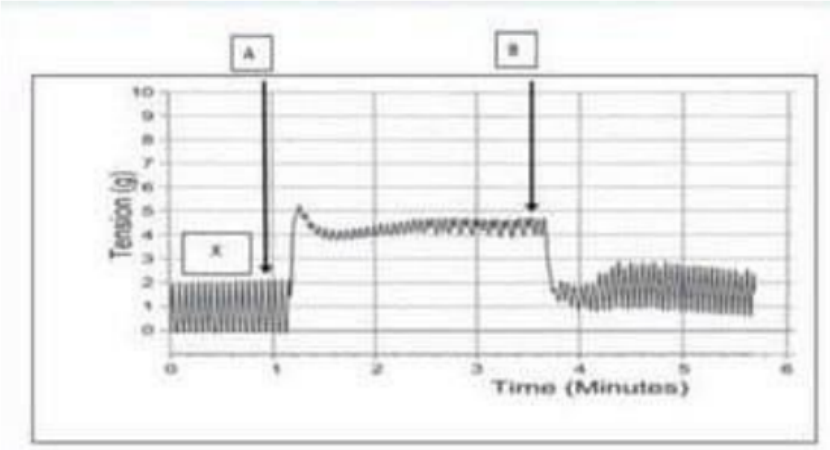
- **Slow waves** determine the rate of contraction.
- **No. of spike potential** determine the strength of the contraction

Notice what's on the Y axis



This is what we measured

2-The graph below represents the rat's small intestinal motility as shown in the physiology lab, before and after adding substance A and substance B. All the following sentences are true, EXCEPT.



a. Substance A mimics the effect of acetylcholine.

b. The effects of substance A could be mediated via muscarinic receptors.

c. The part of the graph labelled as X represents slow waves.

d. Substance A has increased tonic contraction.

e. Substance B could be a competitive antagonist for substance A.

Answer is C