

PCOL 2022 LAB REPORT 2

Aim:

- To demonstrate that distension-induced peristalsis (propagating rhythmic contractile activity) is due to activation of the enteric neuronal network i.e. neurogenic (meaning neuronal in origin)
- To investigate the role of L-type Ca^{2+} channels in distension-induced contractile activity.

Hypothesis:

Title:

Introduction:

Methods:

Result

On observation of each contractile response, all 4 drugs produced inhibitory effects on peristalsis. However, nicardipine is relatively more effective compared to others i.e. lignocaine, atropine and hexamethonium.

There was significant reduction in distension-induced peristalsis with the addition of lignocaine and elevation during the recovery period (Figure 2a). Average contractile response for the control

group was 22.68 a.u. and 6.01 a.u. after lignocaine added, reduced 74% by the presence of Lignocaine. During the recovery period, the peristalsis returns to 84% of the control value.

Similar trends are also observed for atropine and hexamethonium addition. Atropine significantly reduced peristalsis by 69% (CON = 12.4 a.u., ATR = 2.4 a.u.). 54% recovery for atropine (Figure 2b). Hexamethonium significantly reduced the peristalsis by 76% (CON = 21.1 a.u., HEX = 6.2 a.u.). 79% recovery for hexamethonium (Figure 2c).

Nicardipine addition demonstrated highest inhibitory effect on peristalsis, as it completely blocks out all contractile activity of the tissues (NIC = 0 a.u.). No recovery detected after nicardipine added (Figure 2d).

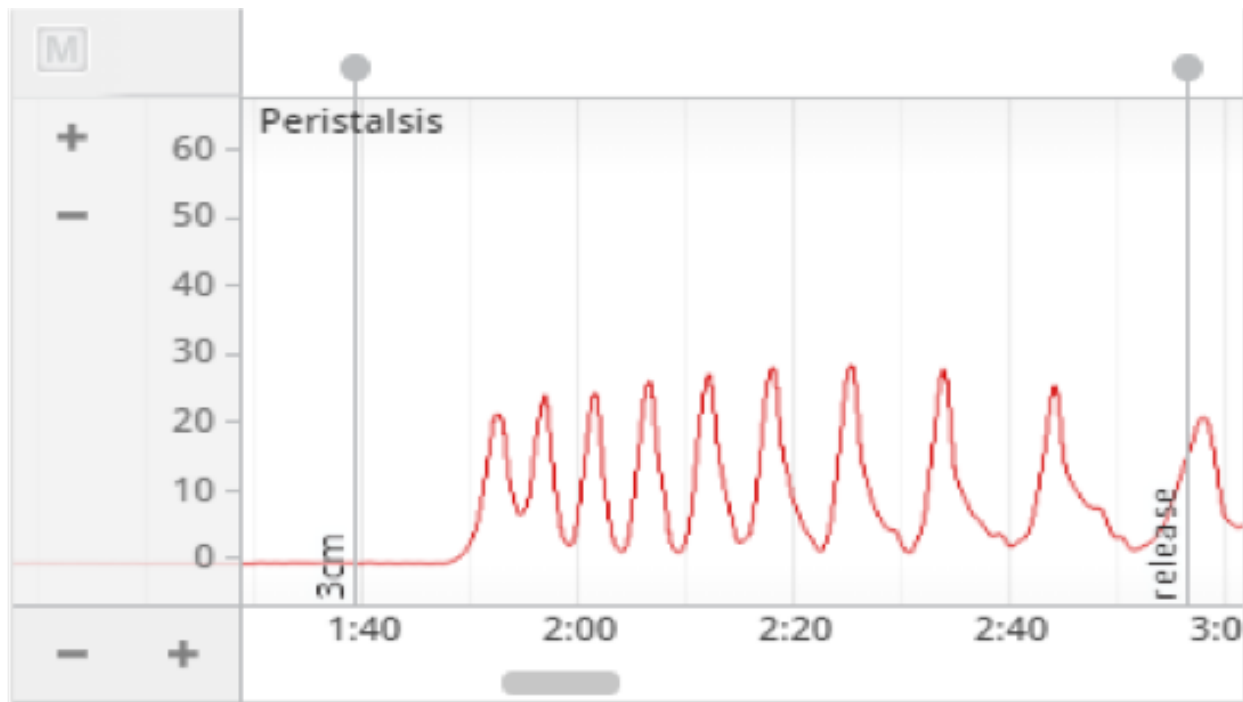


Figure 1. Standard curve showing the contractile response generated by 3 cm hydrostatic pressure in guinea-pig intestine.

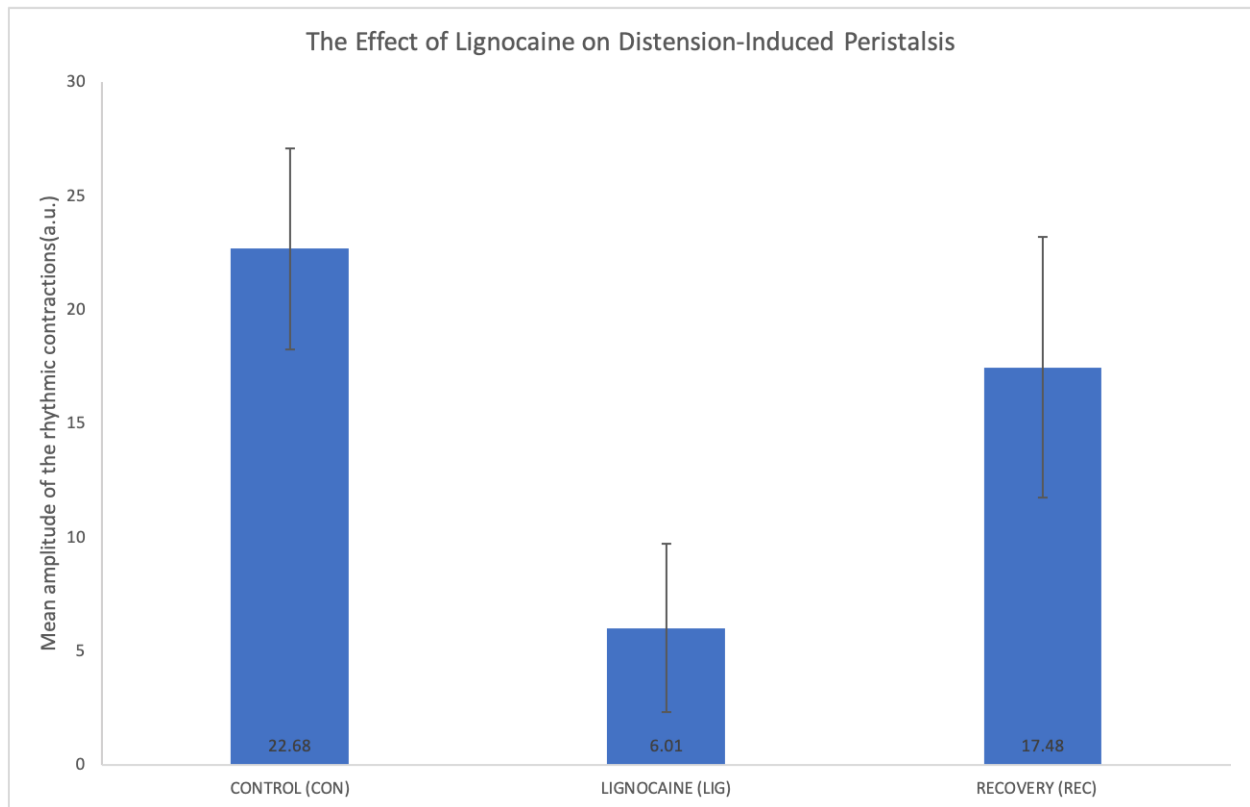


Figure 2a. Mean distension-induced peristalsis (a.u.) ($\pm$ SEM) generated by standard pressure (1 to 4 H₂O cm pressure) in the guinea-pig intestine during control, lignocaine addition (1×10^{-2} M) and recovery period. (N=5)

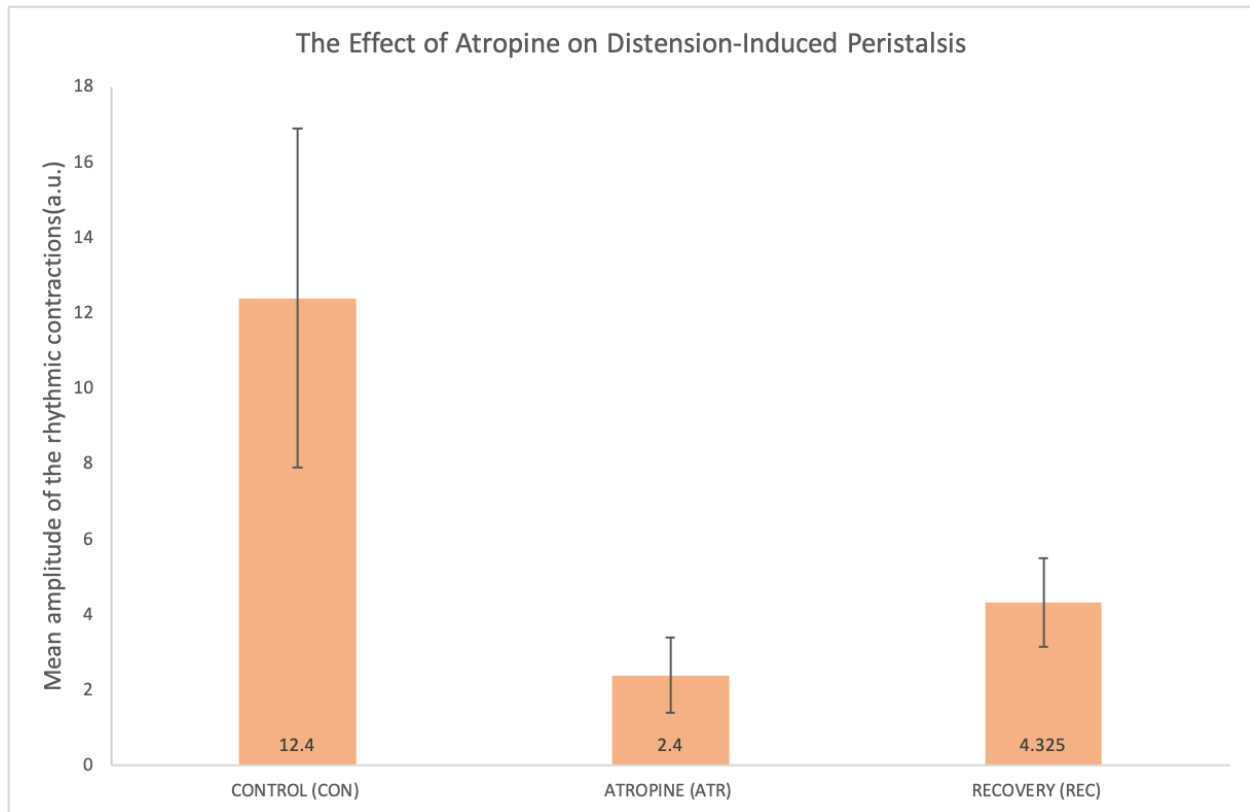


Figure 2b. Mean distension-induced peristalsis (a.u.) ($\pm$ SEM) generated by standard pressure (1 to 4 H₂O cm pressure) in the guinea-pig intestine during control, atropine addition and recovery period. (N=4)

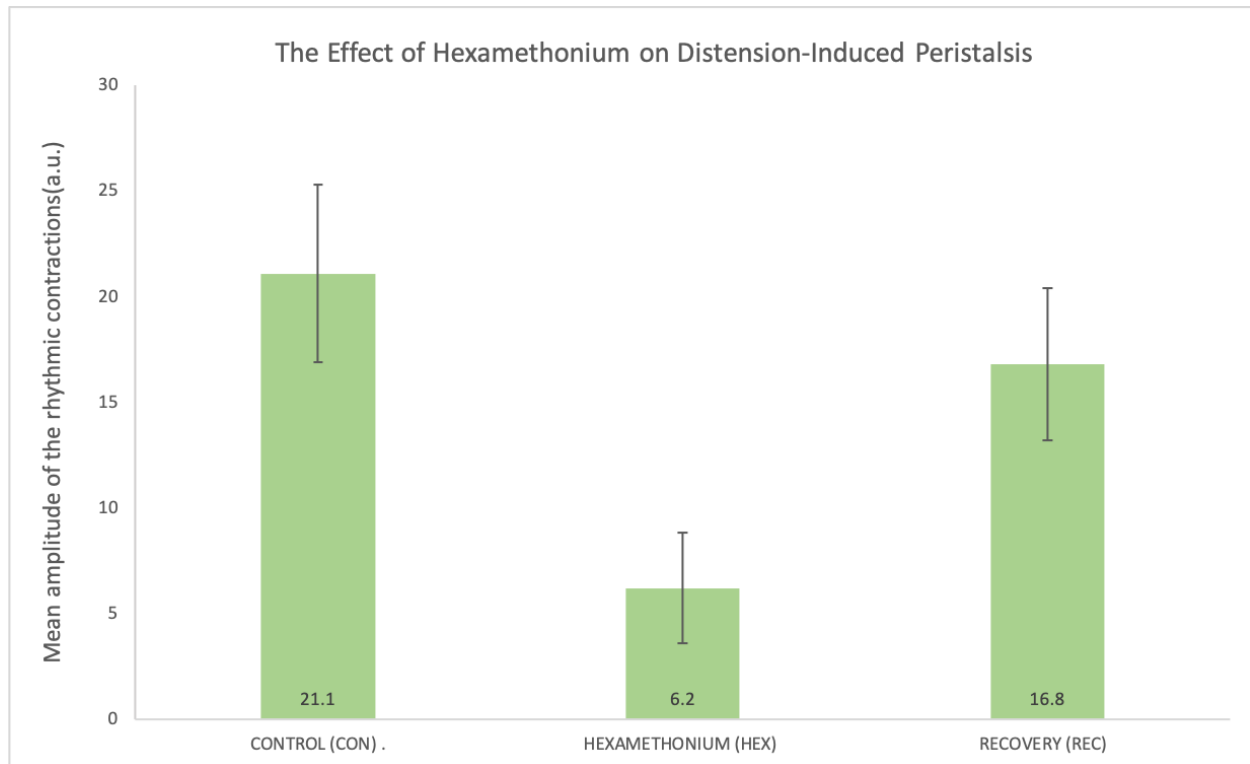


Figure 2c. Mean distension-induced peristalsis (a.u.) ($\pm$ SEM) generated by standard pressure (1 to 4 H₂O cm pressure) in the guinea-pig intestine during control, hexamethonium addition and recovery period. (N=5)

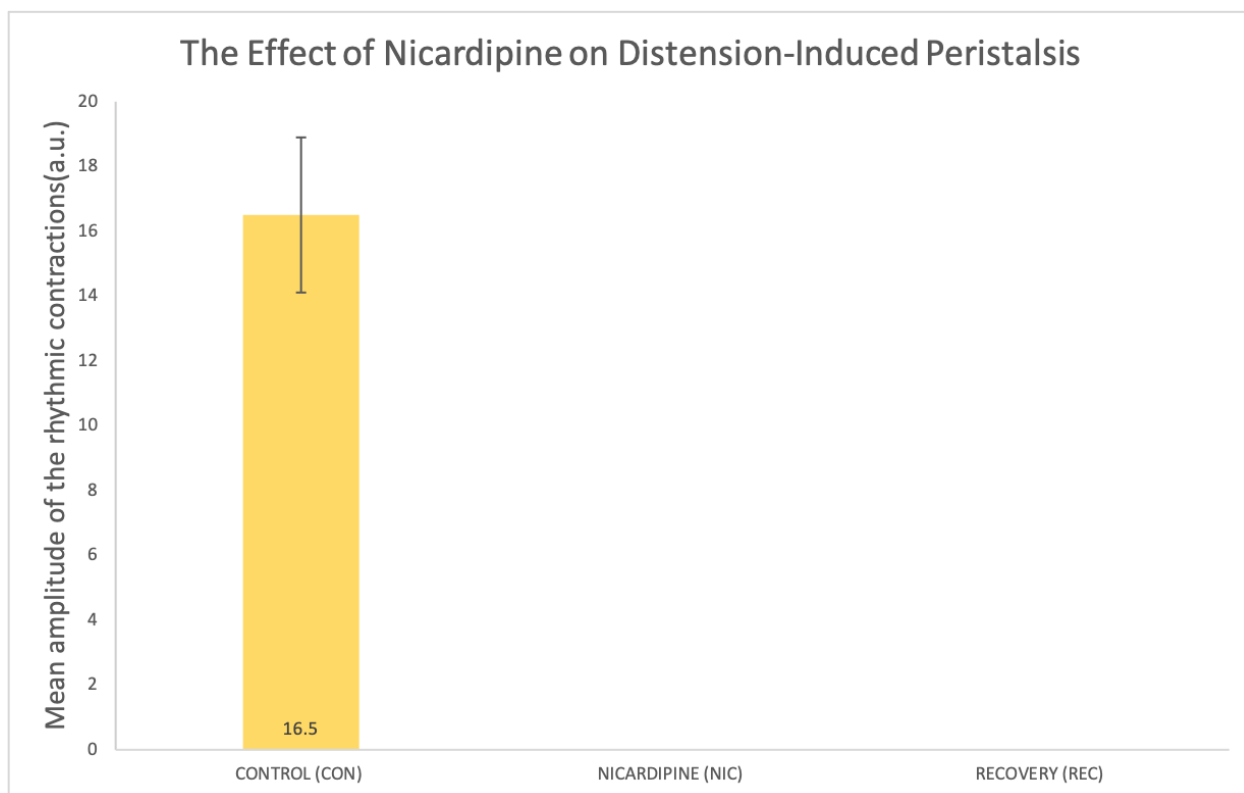


Figure 2d. Mean distension-induced peristalsis (a.u.) ($\pm$ range) generated by standard pressure (1 to 4 H₂O cm pressure) in the guinea-pig intestine during control, nicardipine addition and recovery period. (N=2)

Table 1. Contractile response as percentage with the presence of 4 drugs and the associated recovery rate

	LIG/ CON (%)	REC/ CON (%)	ATR/ CON (%)	REC/ CON (%)	HEX/ CON (%)	REC/ CON (%)	NIC/ CON (%)	REC/ CON (%)
Me an	26.75	83.68	31.34	53.78	23.70	79.28	0	0
SE M	± 11.81	± 21.16	± 16.69	± 18.56	± 10.46	± 9.31	± 0	± 0

Discussion: Ivy and Sherry

- Summarisation and explanation of results
- Limitations
- Conclusion

Peristalsis is a type of smooth muscle contractile activity which is typically rhythmic, propagates and is directional. The experimental results show the effect of four drugs: lignocaine, atropine, hexamethonium and nicardipine on distention induced peristalsis

Lignocaine blocks voltage gated sodium channels, which selectively inhibits nerve action potential conduction. Rhythmic contractile motion of peristalsis was reduced significantly in the presence of lignocaine with an average of 74% reduction. This suggests that distention-induced peristalsis is linked to the activation of nerve action potentials and is neurogenic. Inhibition is non-covalent as recovery is observed.

Atropine is a muscarinic ACh receptor antagonist, which blocks communication between enteric motor neurons and GI smooth muscle cells.

Hexamethonium is a nicotinic ACh receptor antagonist, which inhibits communication between enteric interneurons

Nicardipine is an L-type Ca^{2+} channel blocker, which inhibits the influx of Ca^{2+} into all types of smooth muscle cells. Nicardipine had the most inhibitory effect, completely blocking out all inhibitory activity and having no recovery. This shows that the influx of Ca^{2+} into smooth muscle cells is crucial in peristalsis and that nicardipine is a very selective antagonist for the Ca^{2+} channel. No recovery signifies irreversible binding

Limitations: high variance between results, not enough data, in vitro not in vivo

Conclusion:

Reference: (~5 reference)