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REFLEX PATHWAYS IN THE ABDOMINAL PREVERTEBRAL GANGLIA: EVIDENCE FOR A COLO-COLONIC INHIBITORY REFLEX

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SUMMARY

1. *In vitro* experiments were performed on preparations consisting of prevertebral ganglia attached to the entire colon of guinea-pigs. The colon was divided into an orad and a caudad segment and intraluminal pressure was recorded from the terminal end of each segment. Intracellular recordings were simultaneously obtained from neurones in the coeliac plexus.

2. The source of mechanosensory input from the colon paralleled the responses to mesenteric nerve stimulation. That is, section of the mesenteric nerve that contributed the strongest synaptic input to a neurone eliminated most of the mechanosensory input to that neurone.

3. The origin of the mechanosensory input to some neurones could be localized as coming from either the orad or caudad segment of the colon. In the coeliac ganglia 68% of the neurones tested responded primarily to orad distension and 37% to caudad distension. In the superior mesenteric ganglion 57% responded to orad distension and 43% to caudad distension.

4. Repetitive stimulation of the mesenteric nerve trunks arising from the prevertebral ganglia inhibited contractions differentially in the orad and caudad segments. The inferior coeliac nerves inhibited primarily the orad segments of colon and the lumbar colonic nerves inhibited primarily the caudad segments of colon. Stimulation of the superior coeliac nerves did not alter the motility of either segment.

5. When one of the colonic segments was distended, contractions in the other colonic segment were inhibited in 71% of the distensions. This inhibition operated in both directions: either orad inhibiting caudad or vice versa.

6. Cutting the intermesenteric nerve which communicates between the orad and caudad prevertebral ganglia eliminated the inhibitory reflex.

7. These experiments provide evidence for a colo-colonic inhibitory reflex mediated through pathways in the prevertebral ganglia.

INTRODUCTION

Many motor functions of the gastrointestinal tract are reflexly inhibited by stimulation of a distant portion of the tract. Bayliss & Starling (1899) observed that handling of a distal segment of the intestine produced an inhibition of the proximal intestine. This reflex was later referred to as the intestino-intestinal inhibitory reflex (Hermann & Morin, 1934).

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The pathway for this reflex has not been clearly elucidated. While it seems clear that bilateral section of the vagus nerves does not affect the intestino-intestinal inhibitory reflex (Youmans, 1944), the effects of sectioning the sympathetic pathways are controversial. The necessity for intact connexions with the central nervous system through sympathetic pathways has not been settled with certainty. Some investigators found that the inhibitory reflex was eliminated by either splanchnicotomy alone (Morin & Vial, 1934), by splanchnicotomy and lumbar sympathectomy (Freund & Sheehan, 1943; Youmans, Karstens & Auman, 1942), by dorsal rhizotomy (Chang & Hsu, 1942) or by spinal anaesthesia (Johansson & Langston, 1964). Others (Kuntz, 1940; Kuntz & Saccomanno, 1944; Semba, 1954) found that connexions with the central nervous system were not necessary because the reflex persisted as long as the gut was attached to the prevertebral ganglia even though these ganglia were decentralized. Under these conditions, treatment of the coeliac ganglia with nicotine abolished the reflex (Semba, 1954). These studies suggest the existence of reflex pathways between the prevertebral ganglia and the gastrointestinal tract.

Electrophysiologic experiments have shown that the classical post-ganglionic nerves which connect the abdominal ganglia with the gastrointestinal tract also contain nerve fibres which make synaptic connexions with neurones in the ganglia. These fibres are axons of neurones whose cell bodies lie in the periphery (McLennan & Pascoe, 1954; Job & Lundberg, 1952; Syromyatnikov & Skok, 1968). Intracellular recordings have been made from prevertebral ganglia (Crowcroft & Szurszewski, 1971; Crowcroft, Holman & Szurszewski, 1971; Weems & Szurszewski, 1977, 1978; Kreulen & Szurszewski, 1978*b*). These studies show that neurones in the inferior mesenteric ganglion, superior mesenteric ganglion, and coeliac ganglion of the guinea pig receive afferent input from the distal colon. The afferent input is from mechanoreceptors which are sensitive to distension of the colon (Crowcroft *et al.* 1971; Weems & Szurszewski, 1977).

Recently, we mapped the distribution of peripheral, synaptic input to cells in the coeliac plexus, coeliac and superior mesenteric ganglia, and found that the inferior coeliac nerves contain afferent fibres which synapse on neurones in the coeliac plexus and the inferior mesenteric ganglion (Kreulen & Szurszewski, 1978*b*). Further, we found that afferent fibres in the lumbar colonic nerves synapse on neurones in the inferior mesenteric ganglion and coeliac plexus. These results suggested to us that the orad and caudad colon may be in communication with each other through these extrinsic pathways and that motility patterns in the orad and caudad colon may be dependent on one another.

Thus the purpose of the present experiments was to determine if the nerve pathways which exist between the colon and the prevertebral ganglia function to mediate colo-colonic inhibitory reflexes initiated by distension. The experiments were performed on an *in vitro* preparation which precluded any interactions with the central nervous system or circulating hormones. Part of this study has been previously communicated (Kreulen & Szurszewski, 1978*c*).

METHODS

The results in this study were obtained from twenty-five preparations. Young, male guinea-pigs were stunned and bled. After the region around the abdominal aorta between the coeliac artery and the inferior mesenteric artery was exposed, the abdominal aorta and prevertebral ganglia attached to the gastrointestinal tract by vascular and nervous supply was removed from the animal. The stomach, small intestine and caecum were dissected away so that only the whole colon (18–25 cm) remained attached to the prevertebral ganglia and aorta by the neurovascular bundles. The coeliac ganglia and superior mesenteric ganglion and associated mesenteric and splanchnic nerve trunks were cleaned of surrounding fat and connective tissue.

Each preparation was placed in an organ bath consisting of two compartments separated by a 1 mm thick wall. The prevertebral ganglia were pinned down in one compartment and the colon laid loosely in the other. The mesentery containing the mesenteric arteries and nerves was draped over a separating partition and was covered with tissue paper kept moist by a drip of Krebs solution. Each compartment of the bath was perfused separately with Krebs solution of the following ionic composition (mM): Na^+, 137; K^+, 5·9; Ca^{2+}, 2·5; Mg^{2+}, 1·2; Cl^-, 134; HCO_3, 15·5; $H_2PO_4^-$, 1·2; glucose, 11·5. The solution was bubbled continuously with a 97% O_2, 3% CO_2 gas mixture (pH 7·4 ± 0·5). The solution was warmed before entry into the bath and the temperature in the bath was maintained at 35·5–37·5 °C as measured near the preparation with a thermistor probe.

Pressure measurement. Intraluminal colonic pressure was measured using a modification of the Trendelenberg technique. The system used is diagrammatically illustrated in Fig. 1. In each experiment, the colon was divided into an orad and a caudad segment. The orad segment was approximately 60% of the length of the colon and the caudad segment was approximately 40%. Catheters were tied into both the orad and caudad ends of each of the colonic segments. The catheters at the orad ends were connected to 12 ml. syringes filled with Krebs solution. The catheters at the caudad ends were connected to a Y-fitting one end which was connected to a Statham pressure transducer and the other to a cylindrical reservoir (inner diameter, 0·55 cm) and a 12 ml. syringe. The pressure in each segment was manipulated by adding or removing Krebs solution from either of the syringes thus altering the height of solution in the reservoir and hence the pressure in the colonic segment. This pressure measuring system also allowed the colon to change its intraluminal volume during contractile activity. Zero pressure was determined before inserting the catheter into the colonic lumen and was that pressure in the system with the end of the catheter in the bath at the level of the colon. The level of Krebs solution in the reservoir at zero pressure was marked on the reservoir. This served as a reference throughout each experiment. The general properties of a similar pressure recording system have been described previously (Szurszewski & Weems, 1976).

Nerve stimulation. Nerves to be stimulated were placed on bipolar plantinum electrodes which were isolated by stimulus isolation units. The voltage used for nerve stimulation was dependent upon the size of the nerve trunk and the amount of adherent connective tissue and adipose tissue. Thus, the specific voltage used for nerve stimulation was noted only in relation to the threshold intensity and the intensity required to produce a maximal response for a particular nerve trunk from a particular preparation. To rule out the possibility that the stimulating electrodes might be affecting the preparation non-specifically, a maximum stimulus was applied to the electrodes when they were in the bath but not touching any nerve trunks. In this case, there was no observable effect on either the electrical activity of ganglion cells or contractions of the colon.

Electrical recordings from ganglia were made with conventional intracellular micropipettes.

RESULTS

Mechanical activity of the colon

When the intraluminal pressure was monitored from the caudad end of the colonic segments, two patterns of activity were observed in both the orad and caudad segments. The occurrence of one pattern or the other depended upon the basal

intraluminal pressure. One pattern occurred at intraluminal pressures below 5–8 cm H_2O and consisted of pressure waves ranging in amplitude from 0·2 to 0·8 cm H_2O occurring at an irregular frequency. When observed visually, these contractions were not peristaltic and on the rare occasions when ring contractions occurred they were segmental and did not migrate along the colon. The other pattern occurred at intraluminal pressures above 5–8 cm H_2O and consisted of pressure waves ranging in amplitude from 2 to 10 cm H_2O occurring at a frequency of 1–2 per minute. These pressure waves were associated with peristaltic contractions which migrated aborally

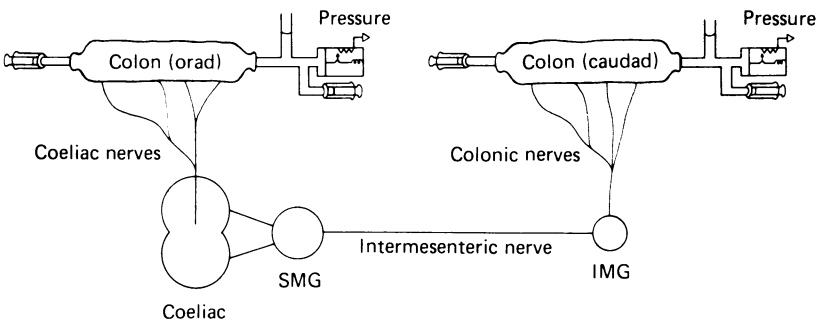

Fig. 1. Diagram of guinea-pig preparation showing the prevertebral ganglia at the bottom and the colon at the top with the mesenteric nerves joining them. The ganglia represented by circles are the right and left coeliac ganglia (coeliac); superior mesenteric ganglion (SMG); and the inferior mesenteric ganglion (IMG). The two major groups of mesenteric nerves are the coeliac nerves and the colonic nerves. The ganglia were pinned to the floor of an organ bath for intracellular recording.

and often resulted in sustained contraction over the terminal 3–5 cm of a segment. These two patterns have been described previously (Weems & Szurszewski, 1977) and are similar to the contractile patterns described for the guinea-pig small intestine by Trendelenberg (1917). In some preparations, the small pressure waves were superimposed on the larger pressure waves and in others the regular interval of the large waves was occasionally interrupted by a lower amplitude wave.

Organization of peripheral pathways

We wanted to determine if peripheral mechanosensory input to the neurones in the coeliac plexus showed any viscerotopic organization. This was suggested by our earlier experiments (Kreulen & Szurszewski, 1978*b*) where we found that more neurones in the coeliac ganglia received input from coeliac fibres than from intermesenteric nerves. Thus we first determined whether the patterns of responses to peripheral nerve stimulation paralleled patterns of natural mechanosensory input and secondly we determined if the mechanosensory input was localized to the orad or caudad segments of colon.

The distribution of natural input reflected the magnitude of the post-synaptic responses to electrical stimulation of the mesenteric nerve trunks. An example of this relationship is shown in Fig. 2. In panel a the responses to peripheral nerve stimulation are compared. In the left two panels, the near-threshold and maximum

response to inferior coeliac nerve stimulation are shown and in the right hand panel the maximum response to intermesenteric nerve stimulation is shown. This particular neurone received stronger synaptic input from the inferior coeliac nerve than from the intermesenteric nerve. Fig. 2*B* shows a recording of continuous synaptic to this neurone when attached to both segments of the colon. When the inferior coeliac nerve was cut, nearly all of the synaptic input to this neurone was abolished indicating that this neurone received nearly all of its mechanoreceptor synaptic input via this nerve.

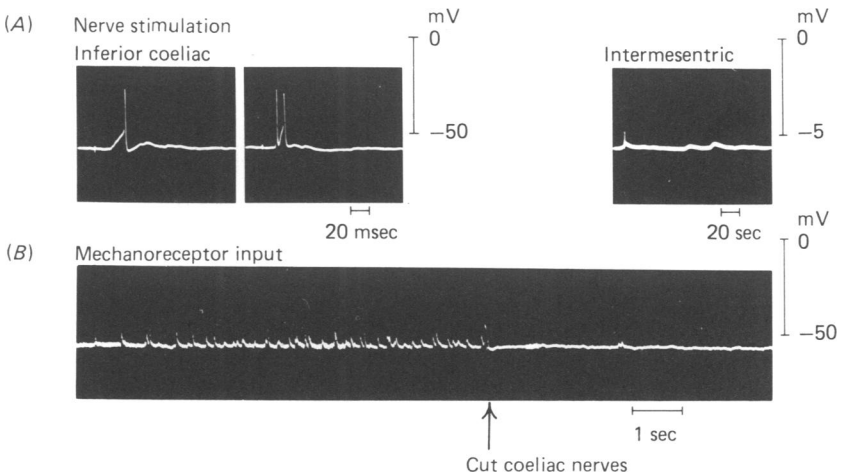

Fig. 2. Relationship between response to mesenteric nerve stimulation and origin of mechanoreceptor input to a neurone in the rostral coeliac ganglia. In *A*, the responses to stimulation of the inferior coeliac nerve at near threshold intensity (left panel) and at maximum stimulation intensity (right panel) are compared to the maximum response to stimulation of the inferior mesenteric nerve. In this same neurone most of the mechanoreceptor input was eliminated by section of the inferior coeliac nerve (*B*).

Localization of menchanosensory input

Evidence that the mechanosensory input to some coelaic plexus neurones was localized to either the orad or caudad region of the colon was obtained by separately distending the orad and caudad segments of the colon. In 68 % of the neurones tested (eleven of sixteen, eleven preparations) in the coeliac ganglia the synpatic input was increased when the orad colon was distended. Synaptic input was increased in 37 % when the caudad colon was distended. Two of these neurones responded to distension of both the orad and caudad segments. In the superior mesenteric ganglion 57 % responded to orad distension and 43 % to caudad. The response of a neurone in the coeliac ganglia to regional colonic distension is shown in Fig. 3. In Fig. 3*A* the pressure in the orad segment was raised momentarily from 0 to 7 cm H_2O and returned as rapidly as possible to zero pressure. As soon as the pressure in the orad segment began to rise, the frequency of synaptic potentials increased approximately 300 % and remained at this level throughout the distension. When the intraluminal pressure was reduced to zero, the frequency of e.p.s.p.s decreased immediately.

However, it was not possible to simultaneously reduce the intraluminal pressure throughout the whole segment of colon; emptying had to be accomplished by maintaining zero pressure at the terminal end of the segment. The result was that the synaptic input also gradually decreased back to the predistension level. In contrast to the response to distension of the orad segment, distension of the caudad colon produced no apparent change in the level of synaptic input to this neurone (Fig. 3*B*). Thus, this particular neurone received mechanosensory synaptic input to the orad segment of colon but not the caudad segment.

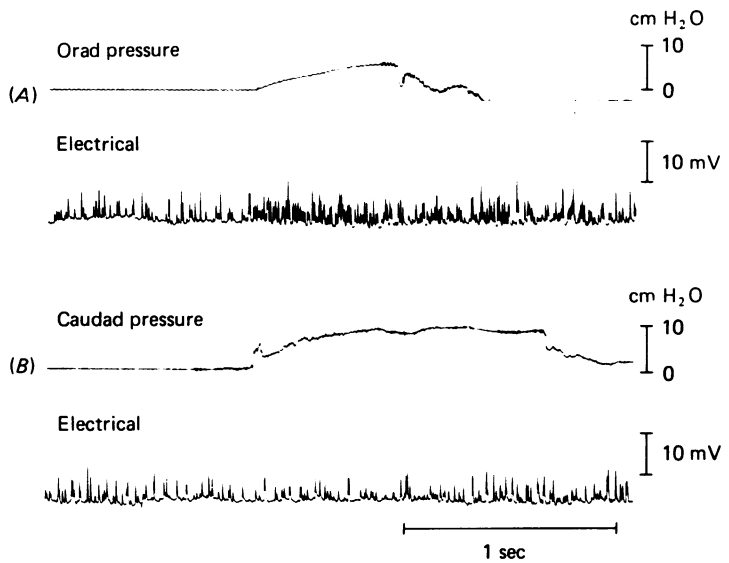

Fig. 3. Responses of a neurone in the coeliac ganglion to distension of orad and caudad segments of the colon. In each panel the upper trace is intraluminal pressure and the lower trace is the intracellular record from the same neurone. This neurone received increased synaptic input when the orad segment of colon was distended (*A*) but not when the caudad segment was distended (*B*).

Localized inhibition of colonic motility by stimulation of mesenteric nerve trunks

To determine if the post-ganglionic outflow to the colon innervated selected regions of the colon, we recorded changes in intraluminal pressure in the two colonic segments in response to repetitive stimulation of each of the mesenteric nerve trunks. Nerve stimulation produced decreases in basal intraluminal pressure and inhibited the propulsive contractions. The responses were qualitatively similar in all preparations An example is shown in Fig. 4. The top tracing in each panel shows the intraluminal pressure in the orad segment and the bottom tracing the intraluminal pressure in the caudad segment. Stimulation of the inferior coeliac nerve (Fig. 4*A*) caused a 38% decrease in the basal intraluminal pressure in the orad segment but resulted in no measurable decrease in the caudad segment. Furthermore, in the orad segment inferior coeliac nerve stimulation eliminated the pressure waves indicative of propulsive contractions. Stimulation of the superior coeliac nerve did not alter the pressure patterns in either of the colonic segments (Fig. 4*B*). Stimulating the inferior

mesenteric nerve produced decreases in both the orad and caudad pressures (Fig. 4*C*) but there was a greater inhibition of the basal intraluminal pressure in the orad segment (30%) than in the caudad segment (19%). In spite of the greater decrease in the basal pressure in the orad segment, inferior mesenteric nerve stimulation had a more profound inhibitory effect on contractions in the caudad segment. Stimulation of the lumbar colonic nerve had a greater inhibitory effect on the caudad segment than on the orad segment (Fig. 2*D*). This was manifested by a greater

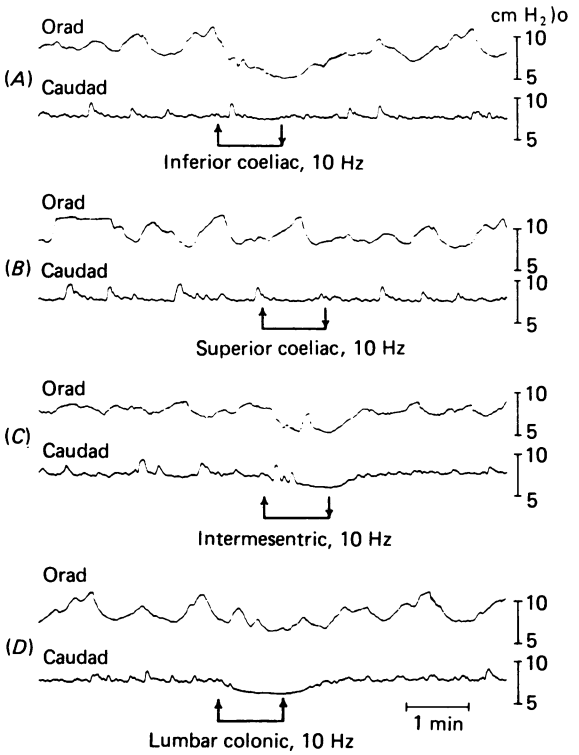

Fig. 4. Pressure changes in an orad and a caudad segment of colon in response to stimulation of mesenteric nerve trunks. In each panel the upper trace is intraluminal pressure in the orad segment and the lower trace is the pressure in the caudad segment. Period of nerve stimulation is indicated by the line between the arrows.

decrease in the basal intraluminal pressure (orad: 15% decrease; caudad: 22% decrease) and especially by an inhibition of all contractions in the caudad segment. Thus inferior coeliac nerve stimulation affected almost exclusively the pressure in the orad segments but the inferior mesenteric nerve and the lower coeliac nerve stimulation inhibited *both* orad and caudad segments.

Inhibitory reflexes between segments of colon: evidence for colo-colonic inhibitory reflexes

Since the above data suggest that pathways exist for a peripheral reflex between the colon and the prevertebral ganglia, we wanted to determine if reflex inhibition between the orad and caudad colonic segments can occur when the only pathway of communication between them is through the prevertebral ganglia. In seven

preparations we tested the response of one colonic segment to distension of the other segment in forty separate distensions. In ten (71 %) of these distensions, contractions in the non-distended segment were inhibited after rapidly distending the other segment. Segments remained inhibited for as long as the other segment was distended (maximum time 7·5 min). From 28 to 60 sec after rapid release of distension contractions in the non-distended segment returned to predistension levels. Fig. 5

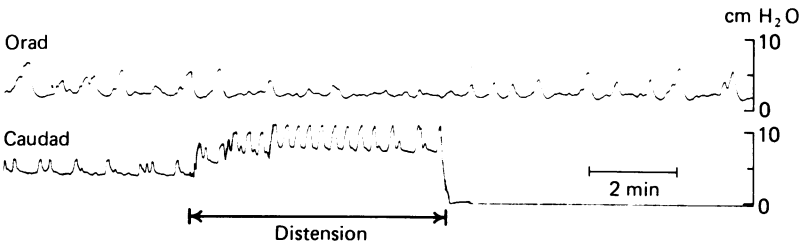

Fig. 5. Effect of distension of caudad segment of colon on contractions in orad segment. Distension was ended by reducing the intraluminal pressure in the caudad segment to zero.

shows an experiment in which a caudad segment of colon was distended and contractions in the orad segment (top trace) were inhibited. Prior to distension, both segments were at the same basal intraluminal pressure. Ninety seconds after distension of the caudad segment the amplitude of contraction in the orad segment was reduced and after 2·3 min the contractions were virtually abolished. The pressure in the caudad segment at the peak of contractions did not exceed 10 cm H_2O. The distension was maintained for 5·7 min. Twenty seconds after release of the distension in the caudad segment, the amplitude of contractions began to increase in the orad segment. After 3 min, the amplitude of contractions in the orad segment returned to control amplitude.

This inhibitory reflex also operated in the opposite direction. Distension of orad segments inhibited contractions in caudad segments. An example is shown in Fig. 6. In both panels the top trace is a recording of intraluminal pressure in the orad colonic segment and the bottom trace is a recording of intraluminal pressure in the caudad segment. Before distension, basal pressure in the orad segment was zero and basal pressure in the caudad segment was 3 cm H_2O. One-hundred seconds after the pressure in the orad segment was increased (Fig. 6*A*) to a basal pressure of 5 cm H_2O and a peak pressure of 10 cm H_2O, contractions in the caudad segment were inhibited and remained so until 40 sec after the pressure in the orad segment was returned to zero, at which point the amplitude of contractions returned to their pre-distension level.

Effect of sectioning intermesenteric nerve on colo-colonic reflex

The only apparent pathway of communication between the two segments of colon was through the prevertebral ganglia. To confirm that the colo-colonic inhibitory reflexes were mediated through pathways in the ganglia, the inferior mesenteric nerve was sectioned in three preparations after the inhibitory reflex was produced. Similar

results were obtained in these preparations. Fig. 6*B* shows a second distension of the orad segment after the contractions in the caudad segment had returned from their inhibited state (Fig. 6*A*). Again, contractions in the caudad segment were inhibited so that by 70 sec after beginning distension, the contractions were abolished. The caudad segment remained inhibited as long as the inferior mesenteric nerve remained intact. When this nerve was cut, the amplitude of contractions in the caudad segment increased and returned to predistension amplitude over the next 4 min. This recovery occurred even though the orad segment remained distended. Release of the distension of the orad segment (not shown) did not result in any increase in basal intraluminal pressure or further increases in contraction amplitude in the caudad segment.

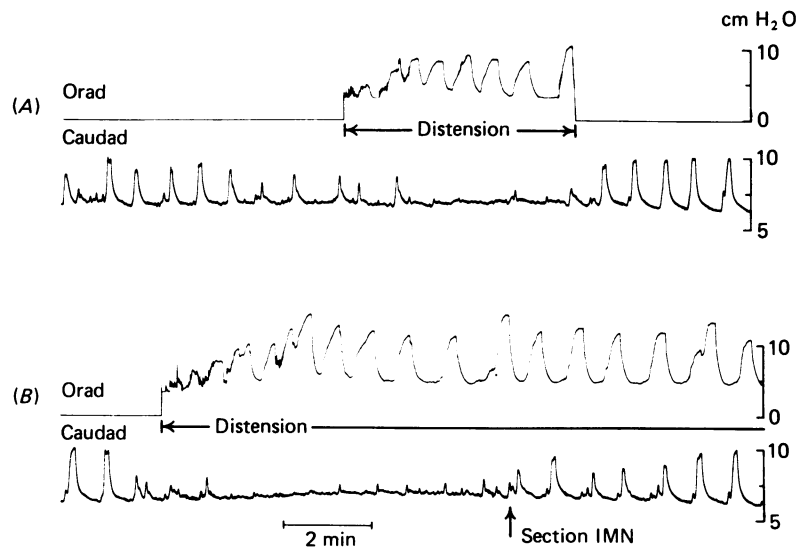

Fig. 6. Effect of distension of orad segment of colon on contractions in caudad segment and the effect of sectioning intermesenteric nerve (IMN). The intraluminal pressure was zero before and after distension. The trace in *B* begins 2 min after the end of panel *A*.

DISCUSSION

In this study we have shown that (1) mechanoreceptor input from the colon to neurones in the coeliac plexus is organized so that approximately 65% of the neurones tested received synaptic input from orad regions of the colon whereas 39% of the neurones tested received synaptic input from caudad regions of the colon; (2) the post-ganglionic outflow to the colon is similarly organized in an orad-caudad manner; and (3) inhibitory reflexes between orad and caudad regions of the colon are mediated through the prevertebral ganglia *in vitro*.

This is the first demonstration that neurones in the coeliac plexus receive sensory input primarily from specific regions of the colon. In a previous report, Crowcroft *et al.* (1971) concluded that mechanosensory synaptic input to inferior mesenteric ganglion neurones came from all regions of the distal colon since progressive reduction

in the number of mesenteric nerve fibres between this ganglion and distal colon resulted in progressive decreases in the amount of excitatory synaptic input. However, their preparations included a 11–12 cm segment of only the distal colon which would be primarily the aborad region of the colon as studied in the present study. We did not determine the size of the 'receptive fields' of individual neurones but it was apparent while recording in some neurones that distension of the first 1 or 2 cm of a segment maximally increased the mechanosensory input whereas distension of the remainder of the segment did not increase synaptic input. It may be then that while a neurone receives input from a large area of the colon, or perhaps even the whole colon, the majority of the fibres synapsing on it come from a limited region of the colon.

The neuronal pathways of the peripheral colo-colonic reflex arc have been demonstrated in this study by observing changes in synaptic input in response to distension of the orad or caudad regions of the colon. The arrangement of pathways between the prevertebral ganglia and the colon suggests how information travels through the ganglia between different regions of the colon. The orad region of the colon receives inhibitory post-ganglionic outflow primarily from the inferior coeliac nerve which contains axons of neurones located in the coeliac ganglia (Kreulen & Szurszewski, 1978*b*). In turn, most of the neurones in these ganglia receive mechanosensory input primarily from the orad region of the colon. The caudad region of the colon receives post-ganglionic outflow primarily from the lumbar colonic nerves. This nerve trunk contains axons of cell bodies located in the inferior mesenteric ganglion and, to a lesser extent, the superior mesenteric ganglion (Kreulen & Szurszewski, 1978*a*). The neurones in these ganglia receive mechanosensory input primarily from the caudad regions of the colon. However, some neurones in the superior mesenteric ganglion received synaptic input from both orad and caudad fibres. Thus some neurones which project to the orad colon receive synaptic input from fibres coming from the caudad colon and some neurones which project to the caudad colon receive mechanosensory input from fibres from the orad colon. This explains why distension of the caudad colon increases synaptic input to and thereby the post-ganglionic outflow from neurones that project to the orad colon. In a similar manner, distension of the orad colon increases the firing of neurones that project to the caudad colon.

Intestino-intestinal inhibitory reflexes were first reported by Bayliss & Starling (1899) who found that pinching or distension of one segment of bowel inhibited contractions in the rest of the gastrointestinal tract. Since then many investigators have demonstrated such reflexes in intact animals (See Youmans, 1968). Controversy has centred around the role of the prevertebral ganglia in mediating these reflexes. Some investigators (Kuntz & Van Buskirk, 1941; Kuntz & Saccamono, 1944; Semba, 1954) found that the reflex persisted after decentralization of the prevertebral ganglia or treatment of the ganglia with nicotine while others found that the reflex was eliminated by severing connexions with the spinal cord or by decentralization and removal of the abdominal sympathetic chains (Youmans, *et al.* 1942; Freund & Sheehan, 1943; Morin & Vial, 1934). Two questions are central to the disagreement: (1) were all connections with the central nervous system transected when reflexes were found after decentralization of the ganglia, and (2) were the intraluminal pressures used during distension unphysiologically high and therefore injurious to the intestine?

In the present study the first question has been obviated by the use of an *in vitro* preparation. The only connexion between the two segments of colon was through the prevertebral ganglia. As to the second question, intraluminal pressure never exceeded 12 cm H_2O at the peak of the contractions during distension. Furthermore, the propulsive contractions elicited by distension is an indication that the colonic segment was not injured and that the intrinsic nerve plexuses were intact. At any rate, it is not probable that the mechanoreceptors which project to the prevertebral ganglia have low sensitivity and therefore would require extremely high distension pressures to be activated (Kuntz & Saccomanno, 1944) because afferent synaptic input occurred below pressures of 5 cm H_2O. Furthermore, the amount of mechanoreceptor input to inferior mesenteric ganglion neurones reached a maximum between 8 and 20 cm H_2O; further increases in pressure did not alter synaptic input (Weems & Szurszewski, 1977).

However, since the central nervous system is a source of excitatory synaptic input to prevertebral ganglion neurones, it is probable that after decentralization of the ganglia it would be necessary to distend a longer portion of the intestine to recruit more mechanoreceptors. Recruitment of a larger number of mechanoreceptors would more likely bring the post-ganglionic neurones to the same level of activity reached when both peripheral and central synaptic inputs converged on these neurones.

Demonstration that a colo-colonic reflex was mediated through pathways in the prevertebral ganglia raises the possibility that other viscero-visceral reflexes could be mediated by a peripheral reflex arc. Kuntz & Van Burkirk (1941) and Warkentin, Huston, Preston & Ivy (1943) found that distension of ileum and colon inhibited bile flow in cats which the coeliac ganglia had been decentralised. Application of a nicotine solution to the ganglia abolished the reflex (Kunts & Van Buskirk, 1941). Likewise Schapiro & Woodward (1959) found that the inhibition of gastric contractions produced by instilling acid into the duodenum was unchanged following decentralizing of the coeliac ganglia but was eliminated by coeliac ganglionectomy. Since the mesenteric nerve fibres which synapse with neurones in the prevertebral ganglia innervate all of the abdominal organs, it is possible that pathways exist which could mediate reflexes between all parts of the gastrointestinal system.

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