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SPINAL CORD INFLUENCES ON THE COLONIC MYOELECTRICAL ACTIVITY OF FED AND FASTED RATS

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SUMMARY

1. The myoelectrical activity of the large intestine of fed and fasted rats was recorded with chronically implanted nichrome wire electrodes after destruction of the spinal cord, after spinal cord transection, and after spinal anaesthesia.

2. After spinal cord ablation, the cyclical organization of the colonic electrical spiking activity, as well as the gastrocolic reflex and accompanying postprandial enhancement of the cyclical pattern of activity, persisted on the proximal and distal colon. On the transverse colon, however, the spiking activity was considerably increased. This latter effect obliterated the gastrocolic response due to feeding but not the subsequent postprandial enhancement of the cyclical pattern of activity.

3. After spinal cord transection, the level of spiking activity also increased on both the transverse and distal colon, but no major changes in cyclical activity or in postprandial responses were recorded.

4. Spinal anaesthesia produced by intrathecal lidocaine increased the motility of the transverse colon to a level which masked the gastrocolic reflex.

5. These results suggest a prevertebral ganglia and/or a local control mechanism for the cyclical organization of the spiking activity of the colon. The central control mechanisms involve mostly spinal inhibitory influences on the transverse colon and supraspinal inhibitory influences on the distal colon.

INTRODUCTION

The role of the spinal cord in the control of colonic motility has been frequently investigated not only as a pathway between the brain and colon, but also as a lower control centre in itself. A tonic inhibitory outflow to the colon via lumbar sympathetic pathways has been demonstrated in the dog (Learmonth & Markowitz, 1930) and cat (Garry, 1933). This inhibitory activity seems to be generated within the spinal cord (Hulten, 1969; De Groat & Krier, 1979) which agrees with the observation that patients with thoracic spinal cord injury possess an increased basal colonic motility (Connell, Frankel & Guttmann, 1963).

Recently it has been demonstrated that the basal spike activity of the human distal colon receives its tonic inhibitory influence from the central nervous system, above

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the C5 spinal level. This finding is in accordance with the experiments of spinal cord section in cats showing supraspinal influences on colonic motility (Gardette & Gonella, 1974). In addition, no gastrocolic reflex could be demonstrated in persons with spinal cord injury, suggesting that this reflex was mediated through the spinal cord (Aaronson, Freed & Burakoff, 1985). Since studies in humans have been limited to the distal part of the colon, our understanding of the spinal and supraspinal influences on motility of other parts of the colon remains very incomplete.

In a previous study, the motor profile of the whole colon in conscious fasted and fed rats was characterized by a cyclical pattern of activity. Following a meal this motor profile was disrupted by an immediate period of hyperactivity lasting 40–60 min and corresponding to the gastrocolic reflex (Ferré & Ruckebusch, 1985). In addition, a strong colonic motor response was induced by the intrathecal administration of opioids at systemically inactive doses, suggesting the importance of the spinal cord in the control of the colonic motility in the rat (Ruckebusch, Ferré & Du, 1984).

The aim of the present study was to determine the influence of the spinal cord on the motility of the different parts of the colon in conscious rats in the fasted and fed states. The role of spinal sympathetic outflow was determined by destroying the thoracolumbar cord (ablation) or by its blockade with local anaesthesia. The influences on motility generated at a spinal or a supraspinal level were examined by comparing the effects of cord transection with those of cord ablation on the colonic motor activity.

METHODS

Animal preparation

Twenty male Wistar rats weighing 300–400 g housed singly in wire-bottomed cages and fed lab rat chow were used in these experiments. Under halothane anaesthesia, pairs of insulated nichrome electrodes were implanted under aseptic conditions on the colon at 2, 5 and 10 cm from the ileocaecal sphincter, corresponding to the ascending, transverse and descending colon, respectively (Ferré & Ruckebusch, 1985). Electrodes were also implanted in the masseter muscle for detecting the distribution and duration of the periods of chewing movements. In a group of four rats, the spinal subarachnoid space at the level of the last lumbar vertebra was catheterized. The technique described by Yaksh & Rudy (1976) was modified in order to reduce the motor impairments of the subject, i.e. paralysis with reactions to handling, by the use of a catheter with a total volume of 10 μl. The catheter was inserted at the ninth thoracic vertebra level and pushed until the first lumbar vertebra so that the intrathecal part of the catheter did not exceed a length of 2·2–2·5 cm. At the point of entrance it was fixed to the adjacent muscles by a surgical wire. Its free extremity was passed subcutaneously to exit in the neck region and was fixed to the nearby muscles.

Electromyographic recordings

Recordings were started 5 days after surgery and the electrical activity was registered 24 h day^{-1} with an electroencephalograph (e.e.g.) (Reega Mini 8, Alvar, Paris), using a paper speed of 2·4 cm min^{-1}. The amount of electrical activity was amplified by the e.e.g. machine, summed at 15 s periods and automatically plotted 24 h day^{-1} on the 'y' axis of a potentiometric record with a paper speed of 6 cm h^{-1} (Latour, 1973). In addition, a motility index for a 20 min period was assessed by computerization of all the values at 15 s intervals, using an Apple II computer with an eight-channel Analog–Digital converter and an X–Y plotter for the off-line display of data as previously described (Latour & Ferré, 1984). The e.m.g. (electromyogram) value of each 20 min period varied from 0·5 to 3×10^3 arbitrary units, according to the level of spiking activity.

Experimental procedure

Each rat served as its own control. Rats were fed *ad libitum* during the first 2 days, but on the third day were fasted, and 24 h later given a lab chow meal for 2 h. The amount of food ingested per day or during the test meal, was measured. After 8 days of control recordings, rats were anaesthetized with halothane and spinal cord ablation of the fifth lumbar (L5) to the tenth thoracic (T10) vertebra was obtained in one group of eight rats by manipulating a 10 cm hypodermic trocar introduced at the level of L5 and inserted as far as T10. In another group of eight rats, the spinal cord was sectioned between T9 and T10 again under halothane anaesthesia. The rats were reconnected for electromyography and fed *ad libitum* for 6 days. Food was then withheld for 22 h at the end of which a lab chow meal was given for 2 h.

TABLE 1. Comparative effects on colonic motility of spinal cord ablation and spinal cord transection in two groups of four rats fed *ad libitum*. Values are mean ± S.D. for 48 h recordings

	Control	3–6 days after ablation	Control	3–6 days after transection
Proximal colon				
Motility index	2891·3 ±276·5	2721·5 ±305·4	2350·1 ±271·2	2307·2 ±221·3
Frequency of spike bursts	1·22 ±0·11	1·16 ±0·15	1·36 ±0·15	1·35 ±0·09
Transverse colon				
Motility index	1540·7 ±369·8	2737·7* ±444·7	1407·0 ±217·1	1898·5* ±291·9
Frequency of spike bursts	0·96 ±0·19	1·36* ±0·22	1·09 ±0·19	1·19 ±0·20
Distal colon				
Motility index	1027·2 ±229·3	1207·0 ±209·4	1130·3 ±258·9	1295·3 ±387·5
Frequency of spike bursts	0·66 ±0·15	0·75 ±0·11	0·75 ±0·15	0·83 ±0·20

* Significantly different from control values at $P < 0·05$.

Care was taken to empty the urinary bladder twice a day and only results obtained from animals with a normal ingestive behaviour of at least 20 g day^{-1} after surgery were taken into account. In addition, handling was apparently painless and without reactions in rats which exhibited normal ingestive behaviour 2 days after spinal cord section or ablation; therefore only animals which exhibited paralysis of the hind legs were used for recordings.

The group of four rats with intrathecal catheters was used in two series of experiments. First, the rats were fed *ad libitum*, and 20 μl of lidocaine (10%, Xylocaine N.D.) was administered intrathecally after 2 h of control recording. In the second series, the rats were fasted for 24 h and 20 μl of lidocaine were injected 20 min before giving a lab chow meal. Control experiments were performed with intrathecal injection of 20 μl of saline in both series.

Post mortem examination was performed within 2–3 days, after the last recording session under pentobarbitone anaesthesia. The extent and the level of lesions were examined after laminectomy under microscopic magnification.

Statistical analysis of the results was performed using a paired Student's t test and covariance analysis.

RESULTS

Effects of spinal cord ablation vs. transection

Colonic myoelectrical activity was restored 16–18 h after spinal cord ablation and 8–9 h after transection. The motility index was unchanged in the proximal and distal

colon at 2 and 10 cm from the ileocaecal junction (Table 1) and normal alimentary behaviour was found in the two groups of rats fed *ad libitum*. The quantity of lab chow ingested per day (25·8 ± 3·6 g) in nine to eleven meals was unchanged compared to controls (26·3 ± 3·2 g; mean ± S.D. for twenty animals) and the feeding pattern was similar before and after surgery with maximum consumption during the night

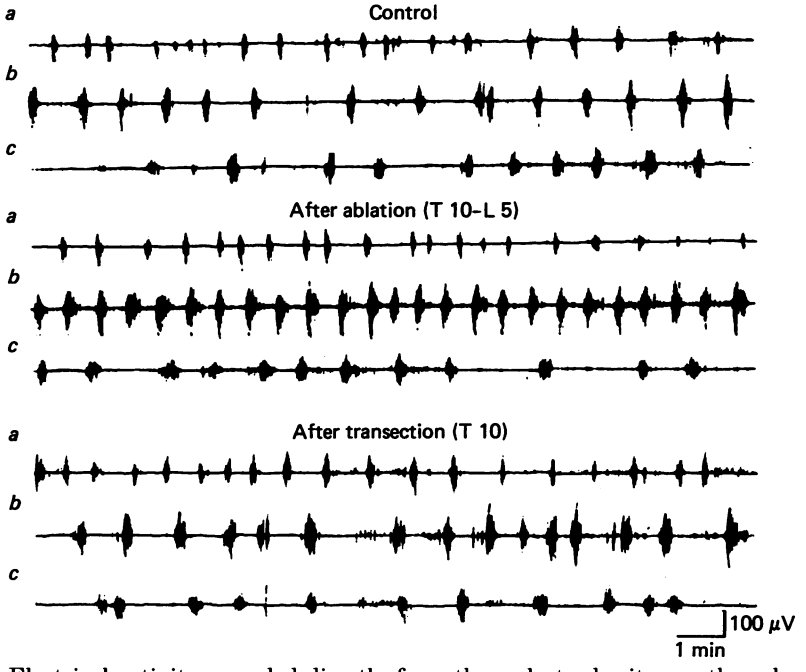

Fig. 1. Electrical activity recorded directly from three electrode sites on the colon at 2 (*a*), 5 (*b*) and 10 cm (*c*) from the ileocaecal junction in the rat. After ablation of the spinal cord, the frequency and duration of spike bursts increased greatly on the middle part of the colon (*b*), whereas after spinal cord transection, only the duration of the spike bursts was slightly increased.

between 21.00 and 02.00 h in the four rats of each group. Furthermore, the spike burst frequency in the proximal and distal colon were not significantly different (Table 1) before and after spinal cord ablation or transection, suggesting that no significant effects resulting from blood pressure changes were experienced. In contrast, the myoelectrical activity of the transverse colon, which consisted of spike bursts (6–14 s in duration, 150–250 μV in amplitude) at a frequency of 0·96 min^{-1}, was strongly modified (Fig. 1).

After spinal cord ablation, the motility index of the transverse colon was increased by 78% as a result of a higher spike burst frequency (about 42%) and duration (26·5%) without nycthemeral variation (see Fig. 2). The velocity of propagation was slightly decreased (3·85 ± 0·25 *vs.* 4·75 ± 0·39 mm s^{-1}, $P > 0·05$). The periods of high spiking activity at intervals of 27–29 min (see Fig. 2) persisted at a similar frequency, so that the increased activity was related to a shorter duration (17%) of the periods of quiescence.

After spinal cord transection, the motility index of the transverse colon was also increased but only by 35% and mostly as a result of an increased duration of spike bursts (24·7%). The velocity of propagation of spike bursts was decreased (4·36±0·54 *vs.* 4·99±0·56 mm s^{-1}, $P > 0·05$). The frequency of the spike bursts and the cyclical organization of the spiking activity were similar to the control rats fed *ad libitum*.

TABLE 2. Electrical spiking activity of the transverse and distal colon in response to feeding in the rat after spinal cord ablation and transection. Values are mean ± S.D. for 3 h recordings in groups of four rats

	Control		Ablation		Transection	
	Fasted	Fed	Fasted	Fed	Fasted	Fed
Transverse colon						
Motility index	1283·9 ±257·8	1934·8 ±319*	2616·9 ±360·8	2831·5 ±334·7	1461·6 ±246·8	2165·3 ±287·5*
Frequency of spike bursts	0·80 ±0·22	1·22 ±0·20*	1·30 ±0·15	1·38 ±0·12	0·92 ±0·16	1·30 ±0·14*
Distal colon						
Motility index	860·8 ±161·2	1307·6 ±250·4*	1072·5 ±131·6	1385·4 ±211·6*	1159·0 ±151·5	1487·8 ±205·8*
Frequency of spike bursts	0·55 ±0·10	0·83 ±0·16*	0·67 ±0·11	0·86 ±0·13*	0·74 ±0·10	0·93 ±0·12*

* Significantly different from the fasted state values at $P < 0·05$.

Colonic responses to feeding

The quantity of lab chow ingested during a test meal (6·8±1·5 g, $n = 20$ meals) and the spiking activity of the proximal colon were unchanged after either spinal cord ablation or transection. After spinal cord ablation, the motility index of the transverse colon increased by 109·8%, i.e. to twice the values of the control fasted rats (Table 2). The frequency and duration of spike bursts were increased by 62·5 and 26·5% respectively without changes in their amplitude or percentage of propagation. Fig. 2 shows that no further increase in the spike burst frequency occurred at feeding. After spinal cord transection, the motility index of the transverse colon was almost identical to that of the control rats during fasting and feeding (Fig. 2, Table 2).

The changes induced in the motility of the distal colon during fasting and feeding were similar after spinal cord ablation or transection. The motility index during fasting was 25–35% higher than in control rats as a result of a higher frequency of spike bursts (Table 2). The motor responses after a meal, which persisted for both the primary response and the secondary phase of enhancement of the cyclical activity, represented an increase in spiking activity of 29 *vs.* 50% in control rats.

Effects of spinal anaesthesia

Following the intrathecal injection of lidocaine, the colonic motility was enhanced within 3 min for about 40 min. The enhancement of spiking activity was markedly greater on the transverse colon than on the distal colon, with no significant changes on the proximal colon (Fig. 3).

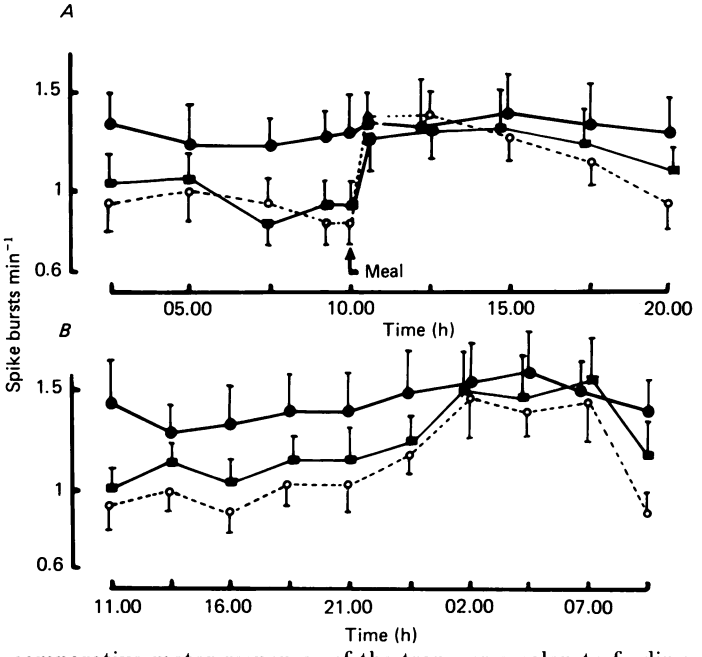

Fig. 2. *A*, comparative motor responses of the transverse colon to feeding expressed in terms of spike burst frequency after a 22 h fast in the normal rat (○) and after ablation (●) of the spinal cord. *B*, colonic motility of rats fed *ad libitum* (○) and after ablation (●) of the spinal cord. Note that after spinal cord ablation the increased frequency of spike bursts during feeding (*A*) and the circadian variations in spike bursts frequency in the rat fed *ad libitum* (*B*) disappeared. In *A* and *B* the responses of rats after spinal cord section are indicated as squares (■).

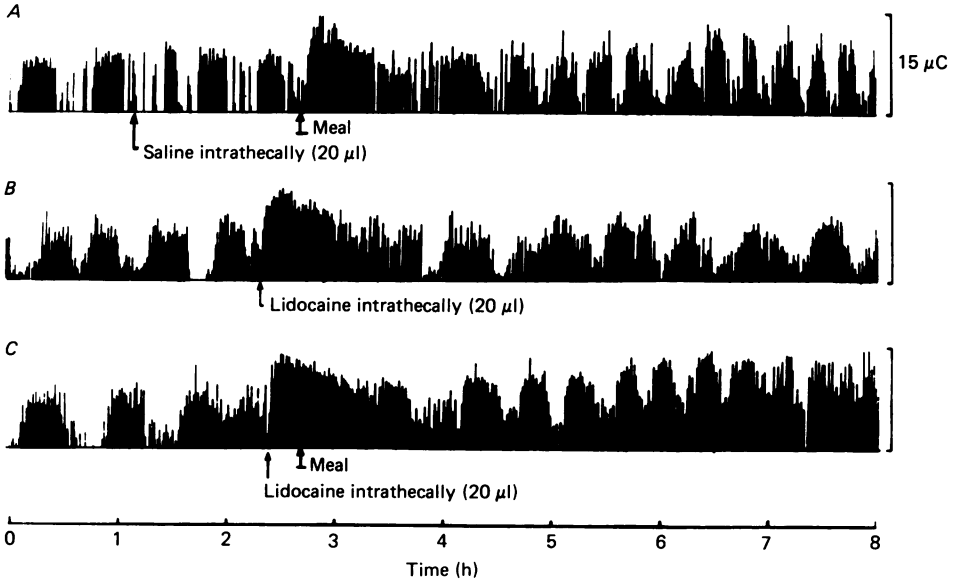

Fig. 3. Effects of spinal anaesthesia on the transverse colonic myoelectrical spiking activity, summed at 20 s intervals, in a rat receiving 6 g lab chow 22 h after a previous meal. *A*, absence of effects of saline on the increased activity during feeding and the postprandial enhancement of the cyclical pattern of activity. *B*, increased colonic motility during 40 min following the intrathecal administration of lidocaine. *C*, persistence of the postprandial response to feeding under spinal anaesthesia.

In the transverse colon, the motility index of fasted rats was increased by 70% as a result of an increased frequency of spike bursts (63·1%) without changes in duration (Table 3). The velocity of propagation of the spike bursts decreased from $5{\cdot}16 \pm 0{\cdot}35$ to $4{\cdot}20 \pm 0{\cdot}34$ mm s^{-1}. During spinal anaesthesia, the primary colonic postprandial response was masked by the already high level of spiking activity. However, the long-lasting enhancement of the cyclic activity was present (Fig. 4*B* and *C*). The motility of the distal colon during spinal anaesthesia was moderately increased (21·3%) in fasted rats as a result of an increased frequency in the spike bursts (15·4%). The two phases of the postprandial response were present on the distal colon.

TABLE 3. Effects of spinal anaesthesia on the increased electrical spiking activity of the transverse and distal colon 20 min after a meal in the rat. Results show mean ± S.D.; $n = 4$ rats

	Saline		Lidocaine	
	Fasted	Fed*	Fasted	Fed*
Transverse colon				
Motility index	1609·4 ±410·7	2490·8 ±250·3‡	2548·6 ±234·1†	2705·0 ±246·5
Frequency of spike bursts	0·84 ±0·21	1·31 ±0·13‡	1·28 ±0·12†	1·33 ±0·12
Distal colon				
Motility index	1279·5 ±205·1	1969·2 ±296·3‡	1551·6 ±191·4	2201·7 ±287·5
Frequency of spike bursts	0·52 ±0·09	0·80 ±0·12‡	0·60 ±0·08	0·84 ±0·10

* 20 min after the beginning of the meal.
† Significantly different from saline, $P < 0{\cdot}05$.
‡ Significantly different from the fasted state, $P < 0{\cdot}01$.

DISCUSSION

The assessment of the extrinsic nervous control of the motor activity of the colon is hampered by the coexistence of inhibitory supraspinal influences (Connell *et al.* 1963; Aaronson *et al.* 1985) and also an inhibitory lumbar sympathetic nerve supply (Learmonth & Markowitz, 1930; Rosta, 1973). In addition, the effects may involve only one facet of the over-all pattern of activity. For example, in man, the cyclical motor events of the small intestine persisted after spinal injury at T1 even though there was a marked decrease in antroduodenal co-ordination (Fealey, Szurszewski, Merritt & DiMagno, 1984).

The present study demonstrates unequivocally that different parts of the rat colon are controlled by spinal and supraspinal influences. (1) The lumbar sympathetic outflow exerts a tonic inhibitory influence upon the motility of the rat transverse colon, since spinal cord ablation significantly increased the motility index by 78% in rats fed *ad libitum* and by 103% in fasted rats. Since this tonic inhibitory influence was still present after spinal cord transection, it would appear that the effect, from lumbar sympathetic outflow, is devoid of supraspinal influences. (2) The distal colon receives supraspinal inhibitory influences since the enhancement of its motility

brought about by spinal ablation is also found after spinal transection. (3) The motor activity of the proximal colon was affected neither by ablation nor by transection, indicating that this part of the colon is not under the control of spinal or supraspinal influences.

The frequency of spike bursts of the transverse colon increased considerably (41–62 %) after the ablation but not significantly (9–15 %) after sectioning of the spinal cord (Tables 1 and 2). However, the duration of spike bursts increased to the same extent, and their velocity of propagation decreased after both ablation and transection. This suggests that frequency of contractions of transverse colon motility is essentially affected by spinal influences, while the duration and the velocity of propagation of spike bursts are mostly affected by supraspinal influences. This differential control does not hold for the distal colon since the frequency of spike bursts increased both after ablation and sectioning of the spinal cord. The effects of spinal anaesthesia further support the existence of these differences in the spinal and supraspinal control of the transverse and distal colon.

Another aspect of colonic motility still poorly understood is the cyclical organization of its activity, including its enhancement as a secondary response to feeding (Ferré & Ruckebusch, 1985). The persistence of the cyclical organization of the colonic activity in the fasted state and of the secondary phase in the response to feeding after ablation or section of the spinal cord demonstrate that colonic cyclical organization is not initiated by lumbar spinal or supraspinal influences. This indicates the involvement of the local enteric nervous system and/or the prevertebral ganglionic system in the cyclical organization of colonic motility. Furthermore, the absence of nycthemeral variations in colonic motility after ablation of the spinal cord might be simply a result of the permanent high motility level subsequent to the removal of tonic inhibitory spinal influences.

The striking feature in the rat was the disappearance of the immediate colonic motor response to a meal, i.e. the gastrocolic reflex, after ablation of the spinal cord, but it persisted after spinal section on the transverse colon and after spinal ablation on the distal colon. The colonic hyperactivity corresponding to the gastrocolic reflex, first described in man by Hertz & Newton (1913) and in dogs by von Lehmann (1913), is through a spinal pathway, at least in the dog (Tansy, Kendall & Murphy, 1972), rather than a vagal-mediated reflex, as suggested by Galopeaux & Templeton (1937).

Recent studies such as those of Hulten (1969), De Groat & Krier (1979), as well as the observations of Gregory (1950) on Thiry–Vella jejunal loops, indicate that a disinhibition of the spinal sympathetic pathways could be involved. Such a concept is highly relevant for the postprandial enhancement of motility of the transverse colon as we observed after spinal section (Fig. 2). The increase in activity of the transverse colon mimicked by spinal anaesthesia or epidural analgesia in man (Lisander & Stenqvist, 1985), and also found in rats after intrathecal administration of lidocaine, is in agreement with such a hypothesis.

In contrast, the persistence of the gastrocolic reflex after ablation of the spinal cord in the distal colon can be related to the influence of a sympathetic prevertebrate ganglionic pathway. This system continues to exert strong inhibitory influences upon the colonic motility as shown by De Groat & Krier (1979) in the cat and by Kreulen & Szurszewski (1979) in the guinea-pig.

Our experiments also clearly emphasize the differential effects of spinal ablation *vs.* chronic spinal transection on the colonic motility. A possible causal relationship of such differential effects could be changes in the concentration of spinal neurotransmitters, e.g. catecholamines (Commissiong, 1985) or spinal neuromodulators, e.g. substance P. In contrast to serotonin (Hadjiconstantinou, Panula, Lackzovic & Neff, 1984), substance P located mainly in spinal afferent neurones (Sharkey, Williams & Dockray, 1984) was significantly decreased after spinal injury in the rat (Faden, Jacobs & Helke, 1985). On the other hand, the intrathecal administration of substance P inhibited the motility of transverse colon (Bardon, Ferré & Ruckebusch, 1985). This effect, contrary to the increased motility observed after spinal ablation, is in accordance with the hypothesis that neuropeptides are the possible modulators in the spinal inhibitory control of colonic motility.

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