Acta Medica Okayama

Volume 38, *Issue* 1 1984 *Article* 3

FEBRUARY 1984

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Abstract

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KEYWORDS: intestine, rectal motility, enteric nerves, automic nerves, defecation

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*PMID: 6702482 [PubMed - indexed for MEDLINE]

Acta Med. Okayama **38,** (1), 21-27 (1984)

MECHANISM OF RECTAL CONTRACTION MEDIATED BY SYMPATHETIC EFFERENTS FROM RECTOANAL PELVIC AFFERENTS IN GUINEA PIGS

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Received September 19, 1983

Abstract. In guinea pigs whose pelvic nerves were bilaterally sectioned, afferent stimulation of rectoanal branches of the pelvic nerve (PAS) could produce an intense contraction in the rectum similar to propulsive contractions elicited during defecation. The mechanism of this reflex was analyzed. Rectal contraction by PAS was abolished after transecting the spinal cord at T13 or sectioning the lumbar splanchnic nerves (LSN) or lumbar colonic nerves (LCN), but was unaffected by severing the intermesenteric and hypogastric nerves. Rectal contraction induced by PAS was abolished peripherally by atropine, guanethidine or yohimbine, while propranolol had no affect. Yohimbine antagonized the inhibitory effect of LSN or LCN stimulation on atropine-sensitive rectal contractions. It may, therefore, be concluded that PAS blocks the inhibition, by LCN efferents acting through α-adrenoreceptors, of cholinergic neurons in the myenteric plexus, thus facilitating recto-rectal propulsive contractions initiated by the defecation reflex.

Key words : intestine, rectal motility, enteric nerves, autonomic nerves, defecation.

It has been demonstrated that during defecation lumbar colonic efferent discharges induced by the lumbar spinal reflex are restrained by discharges descending from the supraspinal sympatho-inhibitory center, possibly activated by pelvic afferent inputs arising from the rectal wall, resulting in an initiation of propulsive contractions (1). Therefore, if rectal pelvic afferents were selectively activated in pelvicotomized animals, lumbar colonic efferent discharges would be expected to be reduced, thereby resulting in rectal contraction.

In the present experiments, the effect of the pelvic-colonic reflex on the rectal motility was examined. Intense phasic contraction was obtained by afferent stimulation of rectoanal branches of the pelvic nerve in guinea pigs. The mechanisms involved in this response were studied.

METHODS

Thirty-six guinea pigs weighing between 370 to 700 g were used. Animals were anesthetized with urethane (1.0 mg/kg, i.p.) and immobilized with gallamine (0.1 mg/kg, i.v.) for 30-60 min. Artificial ventilation was carried out through a tracheal cannula. Body temperature was kept at 38 ± 1 ℃ with a heating pad.

Rectal motility was recorded with a 1 ml balloon inserted 4 cm into the anus. Rectoanal and vesical branches of pelvic nerves were bilaterally sectioned and both branches and the pelvic plexus including posterior pelvic ganglia were carefully separated from the surrounding tissue. Peripheral innervation of the pelvic nerve was shown to be completely severed since the rectum did not respond to peripheral stimulation of the pelvic nerve central to the pelvic plexus. Afferent stimulation of the unilateral rectoanal branches of the pelvic nerve (PAS) and efferent stimulation of contralateral rectoanal branches of the pelvic nerve (PES) were carried out with bipolar platinum electrode.

Sympathetic nerves, *i.e.*, the lumbar splanchnic (LSN), intermesenteric, hypogastric and lumbar colonic (LCN) nerves were sectioned near the inferior mesenteric ganglia in same experiments. LSN and LCN were stimulated peripherally.

The receptor antagonists and neuron blockers used were atropine sulphate (Sigma), atropine methylbromide (Sigma), guanethidine sulphate (Tokyo-Kasei), yohimbine hydrochloride (Sigma), phentolamine hydrochloride (Sigma), propranolol hydrochloride (Sigma) and 4-7-exomethylene-hexahydroindoline-ethyl-guanidine hemisulphate (No. 865-123, Eisai).

RESULTS

Effect of PAS on the rectal motility. PAS produced an intense phasic contraction in 3 animals whose contralateral pelvic nerves were kept intact. This contraction was abolished after spinalization at the T13 segment but returned immediately after sympathetic denervation to the rectum by pithing the spinal cord between the L1 and L4 segments (Fig. 1) or by sectioning LSN. This suggests that PAS

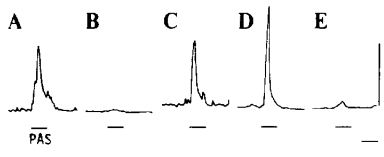

Fig. 1. Effect of afferent stimulation of unilateral rectoanal branches of the pelvic nerve (PAS) on the rectal motility in the guinea pig. A : control, B and C : after transection at T13 and pithing of the spinal cord between L1 and L4, D and E : after intravenous injection of guanethidine (3 mg/kg) and atropine sulphate (1 mg/kg). The vertical bar indicates 50 cmH_2O, and the horizontal bar 1 min.

as well as rectal distension causes an excitation of pelvic efferents through sacral cord and an inhibition of lumbar sympathetic efferents through the supraspinal sympatho-inhibitory center as proposed in our previous paper (1). The following experiments were carried out in the animals, whose pelvic nerves were sectioned bilaterally.

PAS (frequency : 20 Hz, duration : 1-2 msec, intensity : 5-12 V) for 30 to 60 sec at 20 min interval elicited intense contractions of the rectum in 34 out of 37 animals. In the remaining 3 animals, there was a slight and transient relaxation folowed by an intense phasic contraction. The latency, amplitude and duration of the contractile response determined from 57 trials in 14 animals were 29.3 ± 9.6 sec ranging from 12 to 54 sec, 58.9 ± 26.5 cmH_2O (12-77 cmH_2O) and 63.7 ± 39.1 sec (24-180 sec), respectively.

In four guinea pigs, the phasic contraction of the rectum caused by PAS was

abolished after transection of the spinal cord at the T13 segment, and did not recover after sectioning LSN and the intermesenteric nerve (Fig. 2). The contractile response evoked by PAS in the rectum did not change after sectioning the hypogastric nerves, but it was abolished after sectioning LCN in 3 animals (Fig. 3). Sectioning LSN and the intermesenteric nerve also abolished the rectal

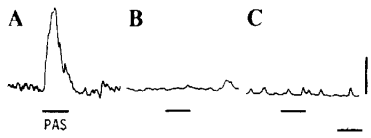

Fig. 2. Effect of PAS on rectal motility in guinea pig whose pelvic nerves were bilaterally sectioned peripheral to the pelvic plexuses. A : control, B : after transection at T13, C: after section of the lumbar splanchnic and intermesenteric nerves. Indicator bars as in Fig. 1.

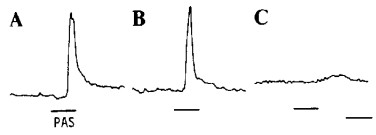

Fig. 3. Effect of PAS on rectal motility (pelvic nerves sectioned). The PAS-induced contraction (A) was not affected by section of bilateral hypogastric nerves (B), but was abolished by section of the lumbar colonic nerve (C). Indicator bars as in Fig. 1.

contraction evoked by PAS. These results indicate that PAS can supraspinally produce contractions in the rectum by reducing LCN activities. There was no correlation between the response and the change in systemic blood pressure observed after spinalization.

Effect of autonomic drugs on the rectal contraction evoked by PAS. To study the mechanism of the reflex rectal contraction induced by PAS through LCN efferents, the effect of the adrenergic neuron blockers, guanethidine and No. 865-123, the α-adrenoreceptor antagonist, yohimbine, the β-adrenoreceptor antagonist, propranolol, and the cholinergic-muscarinic receptor antagonists, atropine sulphate and atopine methylbromide, were examined.

Guanethidine (3-5 mg/kg, i.v.) abolished the contractile response of the rectum in all 5 animals tested (Fig. 4). PAS and PES produced intense contraction in the rectum. The contraction by PAS was abolished after administration of guanethidine (3 mg/kg, i.v.), while the contraction due to PES was resistant to guanethidine. The pelvic-pelvic reflex contraction of the rectum mediated via the

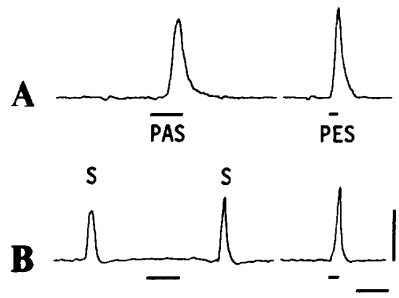

Fig. 4. Effect of PAS and efferent stimulation of rectoanal branches of the pelvic nerve (PES) on rectal motility (pelvic nerves sectioned), A : control, B : 140 min after guanethidine (3 mg/kg), s : periodical phasic contractions which occurred spontaneously after guanethidine. Indicator bars as in Fig. 1.

sacral cord was also resistant to guanethidine (Fig. 1). The rectal contaction produced by PAS was abolished by the adrenergic neuron blocking effect of guanethidine but not by the nonspecific anesthetic effect of this drug. No. 865-123 (3 mg/kg, i.v.) eliminated rectal contractions induced by PAS.

Within about 5 min of an intravenous administration of yohimbine (0.2-1.0 mg/kg, i.v.) the rectal contraction induced by PES was reduced, and the contraction induced by PAS was abolished (3 animals). After the recovery of the contractile response to PES, PAS-induced contractions were still inhibited (Fig. 5). The small contraction observed just after the on-set of PAS was not examined further.

Propranolol (1-2 mg/kg, i.v.) failed to antagonize the contractile response of the rectum to PAS and PES in 5 guinea pigs.

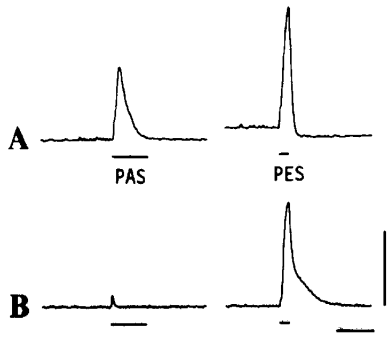

Fig. 5. Effect of yohimbine on PAS- and PES-induced rectal contraction (pelvic nerves sectioned). A : control, B : 22 min after yohimbine (0.2 mg/kg). Indicator bars as in Fig. 1.

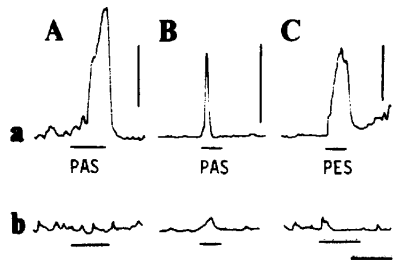

Fig. 6. Effect of atropine on PAS- and PES-induced rectal contraction. a : control, b : after atropine sulphate (0.1 mg/kg). Pelvic-colonic reflex contraction mediated by the supraspinal sympatho-inhibitory center (A), pelvic-pelvic reflex contraction mediated by the sacral parasympathetic center (B) and PES-induced contraction (C) were abolished after atropine, Indicator bars as in Fig. 1.

As shown in Fig. 6, atropine sulphate (0.1-1 mg/kg, i.v.) abolished contractions due to the pelvic-colonic reflex via the supraspinal sympatho-inhibitory center (A) and reflex contractions through pelvic afferents via the sacral parasympathetic center by PAS (B). The contraction induced by PES was also abolished by atropine sulphate (C). Contractions due to the pelvic-colonic reflex after recovering from their inhibition by atropine were abolished by guanethidine (3 mg/kg, i.v.). Atropine methylbromide (0.5-2 mg/kg, i.v.) abolished the pelvic-colonic reflex contractions induced by PAS as well as those induced by PES.

Effect of stimulation of sympathetic nerves on the rectal motility. In 5 animals, stim-

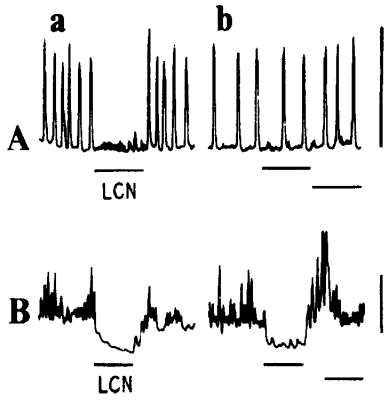

Fig. 7. Effect of yohimbine (0.5 mg/kg) on inhibition of rectal motility induced by colonic efferent stimulation. a : control, b : 25 min after yohimbine. Calibrations : 50 cmH_2O and 5 min in A, and 10 cmH_2O and 1 min in B.

ulation of LSN or LCN efferents reduced rectal tone and eliminated spontaneous intense phasic rectal contractions which were diminished by atropine (0.1 mg/kg, i.v.) but did not produce excitation of rectal motility. After yohimbine (0.2-1 mg/kg, i.v.) was injected, the inhibiting effect of LCN stimulation on spontaneous phasic contractions was abolished in 3 animals, while reduction of the rectal tone produced by LCN stimulation was not altered in 2 animals (Fig. 7).

Propranolol (2-3 mg/kg, i.v.) failed to antagonize to inhibitory effect on the rectum produced by LSN or LCN efferent stimulation in 3 animals.

DISCUSSION

It has been reported that, because of the considerable increase in colonic motility resulting from lumbar sympathectomy, lumbar sympathetic nerves may exert a tonic inhibitory influence on colonic motility (2-8). During rectal distension efferent discharges of LCN increased by activation of LCN afferents are supraspinally restrained by rectal pelvic afferent discharges evoked simultaneously (1, 9). It is thus supposed that PAS reduces tonic inhibitory discharges of sympathetic nerves supplying the rectum, leading to the rectal contractions. Our results strengthen this assumption, since rectal contraction produced by PAS was abolished after transection of the spinal cord at T13 or sympathetic denervation to the rectum in animals in which parasympathetic innervation to the rectum was severed, while LCN efferent stimulation inhibited spontaneous rectal contractions.

Pelvic-colonic rectal contraction produced by PAS was abolished by the muscarinic antagonists, atropine sulphate and atropine methylbromide, and the adrenergic neuron blockers, guanethidine and No. 865-123. The site of action of these drugs was the intramural myenteric plexus since discharges of LCN innervating the rectum were eliminated by PAS and this response was preserved after application of atropine, guanethidine and No. 865-123 (unpublished observations). Pelvic-pelvic rectal contraction mediated by postganglionic cholinergic nerves was enhanced after inactivation of adrenergic nerves and postganglionic

sympathetic nerve (LCN) stimulation inhibited rectal motility. Two alternate mechanism of the initiation of rectal contraction by PAS may be proposed : the restraining of inhibitory action of postganglionic adrenergic nerves on the intramural cholinergic neurons in the myenteric plexus or the depressing of adrenergic inhibitory action on the rectal smooth muscle itself. If the rectal response to PAS was caused by the latter mechanism, both α-and β-adrenoreceptor antagonists should have partly reduced the rectal excitation induced by PAS and inhibition due to LCN stimulation. However, while the α-antagonist, yohimbine, completely abolished rectal responses to PAS and LCN stimulation, the β-antagonist, propranolol, failed to abolish it. The former mechanism, therefore, is probably responsible for initiation of rectal contraction due to the depression of LCN tonic activity by PAS. Convincing arguments for the former mechanism are offered by the following investigations. Histochemical studies have revealed that adrenergic fibers in LCN end in the myenteric plexus of the guinea-pig distal colon and that non-fluorescent neurons in the ganglia of the myenteric plexus are supplied by adrenergic terminals (10, 11). The inhibitory action of sympathetic postganglionic nerves on intramural cholinergic neurons was demonstrated by Beani *et al.* (12); they showed that sympathetic stimulation and catecholamines, adrenaline and noradrenaline reduced acetylcholine release during pelvic nerve and transmural stimulation in isolated guinea-pig colon. Intrinsic autonomic neurons innervating the intestinal smooth muscle have α-adrenoreceptors, activation of which supresses the release of acetylcholine (13).

In conclusion, PAS de-inhibits cholinergic neurons in the myenteric plexus by restraining the inhibitory action of LCN on α-adrenoreceptors, leading to increased acetylcholine release from nerve endings, thereby producing an intense rectal contraction. Such an action of PAS facilitates the initiation of recto-rectal propulsive contractions during defecation.

Acknowledgements. The authers wish to thank Mr. M. Watanabe for his excellent technical assistance and Prof. Y. Misu for his kind gift of No. 865-123.

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