Pelvic afferent reflex control of rectal motility and lumbar colonic efferent discharge mediated by the pontine sympatho-inhibitory region in guinea pigs

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Abstract. Rectal motility and the efferent discharge of lumbar colonic nerves (LCED) have previously been shown to be affected by reflex activity activated by rectal stimulation. The sensory limb of this reflex is represented by afferent fibers in pelvic nerves. The present study revealed that this reflex is modulated by supraspinal sympatho-inhibitory regions. Pelvic afferent stimulation led to rectal contraction through the withdrawal of a tonic inhibitory influence of lumbar colonic nerves. The supraspinal region responsible for this antagonism of the rectal-inhibitory colonic nerve activity was localized to the pons. Neither the intravenous administration of atropine nor that of guanethidine (and Eisai compound 865-123, another adrenergic neuron blocking agent) affected the ability of pelvic afferent stimulation to inhibit tonic discharge of lumbar colonic efferent nerves; nevertheless, both agents eliminated the mechanical response of the rectum to stimulation of pelvic afferents. These observations suggest that lumbar sympathetic nerves may tonically inhibit the release of acetylcholine from excitatory neurons in the rectal myenteric plexus. We conclude that descending fibers from the pons are activated as a result of pelvic aff erent nerve stimulation. These descending pontine fibers in turn inhibit the firing of sympathetic lumbar colonic nerves. Removal of this tonic restraint leads to rectal contraction.

Key words: Defecation reflex — Guinea pig — Lumbar colonic nerve — Pons — Pelvic afferent stimulation — Rectal motility — Supraspinal center — Sympathetic activity

Introduction

Lumbar sympathetic fibers in colonic nerves mediate an inhibitory reflex pathway that functions during defecation (Takaki et al. 1980, 1983). This sympathetic activity can be suppressed by a descending inhibitory pathway that originates in the pons. Afferents from the rectum are contained in pelvic nerves. de Groat and Lalley (1972) have demonstrated a supraspinal influence on the pelvic-hypogastric inhibitory reflex of the cat urinary bladder. They also demonstrated that intense stimulation of pelvic nerve vesical afferents evoked reflex efferent volleys in the lumbar colonic sympathetic nerves. This activity was noted in five of the 16 experiments, although the efferent activity in colonic nerves was less than that observed in the hypogastric nerves after the same afferent stimuli had been applied. The present investigation sought to analyze the recto-rectal reflex. The reflex was evoked by stimulation of pelvic afferent nerve fibers and monitored by measuring the activity of lumbar colonic efferent nerves. It was postulated that this activity could be modulated by a supraspinal sympatho-inhibitory region.

Materials and methods

Experiments were performed on 27 male guinea pigs (body weight 305—740 g). Preparation of the animals, and surgical and experimental procedures have previously been described in detail (Takaki et al. 1983). Briefly, the brain stem was exposed and transected in anesthetized animals. All branches of the pelvic nerves were bilaterally sectioned to prevent pelvic-pelvic reflexes. Rectal pelvic afferents could not be separated from other pelvic afferents although separation of vesical pelvic afferent could be achieved. Accordingly, all pelvic afferents (12/25 animals), or all except for those from the bladder (13/25 animals), were stimulated. Electrical stimulation of pelvic afferent nerve fibers (PAS) was accomplished using a bipolar platinum electrode (rectangular pulses; frequency 20—50 Hz; duration 0.1—2 ms; intensity 6—18 V). In most animals, the intermesenteric and hypogastric nerves were also sectioned; thus, the sole intact efferent pathway to the rectum was that of the lumbar colonic nerves (LCNs). In six of the animals, baroreceptors were denervated by bilateral section of carotid sinus, aortic depressor, and cervical vagus nerves. Systemic blood pressure was continuously monitored via a cannula inserted into the common carotid artery. The temperature of the abdominal cavity was maintained at 36—37° C by a heat lamp; room temperature varied between 25—26° C.

Rectal motility and rectal distension were measured using a 1.5 cm long balloon inserted into the rectum. Lumbar colonic efferent discharge (LCED) was recorded from a branch of the dissected nerve near the inferior mesenteric ganglion as previously reported. A frequency histogram of the LCED was obtained by counting pulses of neural discharge through a Nihon-Kohden discriminator.

Drugs used were atropine sulfate (Sigma, St. Louis, MO, USA), guanethidine sulfate (Tokyo-Kasei, Tokyo, Japan), hexamethonium bromide (C6) (Sigma, St. Louis, MO, USA), and 4-7-exo-methylene-hexahydroindoline-ethylguanidine hemisulfate, Eisai compound 865—123 (Eisai, Tokyo, Japan), an adrenergic neuron blocking agent (Misu et al. 1976).
Results

A. Effect of pelvic afferent stimulation (PAS) on rectal motility

Unilateral electrical stimulation (20 Hz, 1–2 ms, 10–12 V, 1 min) of a pelvic afferent nerve produced intense contraction of the rectum (20–120 cm H₂O) in 18 out of 25 animals, even after bilateral section of all of the branches of the pelvic nerves (Figs. 1A, 2C and D). Stimulation of all of the pelvic afferents caused a rectal contraction in 6 out of 12 animals, and stimulation of all pelvic afferents except for those from the bladder led to rectal contraction in 12 out of 13 animals. The latency and duration of these rectal contractions ranged from 4 to 45 s and 34 to 68 s, respectively. Neither of these types of pelvic afferent stimulation lowered blood pressure; occasional rises of 5–10 mm Hg were noted. The contraction of the rectum in response to pelvic afferent nerve stimulation was not affected by baroreceptor denervation in three animals (data not shown); however, contraction of the rectum was abolished by transecting the spinal cord at T13 or by sectioning the lumbar colonic nerves (LCNs). In contrast, pelvic afferent-induced rectal contraction was unaffected by sectioning the hypogastric and intermesenteric nerves (Neya et al. 1984). Stimulation of the efferent LCNs (20 Hz, 1 ms, 8 V), however, was found to produce a potent and long-lasting inhibition of rectal motility, as described previously (Takaki et al. 1983). These results suggest that the rectal contraction evoked by stimulation of pelvic afferent nerves was induced by a withdrawal (or inhibition) of lumbar colonic nerve activity.

B. Effect of pelvic afferent stimulation (PAS) on lumbar colonic efferent discharge (LCED)

The discharge rate of lumbar colonic nerves was reduced to less than 10 Hz by pithing the spinal cord (L1–4) in 3 animals. Administration of C6 (1 or 5 mg/kg i.v.) fully eliminated efferent activity in confirming that these are postganglionic fibers.

After observing a rectal contraction elicited by PAS, a lumbar colonic nerve was sectioned, and its action potentials were recorded. Pelvic and hypogastric nerves as well as the intermesenteric nerves were then severed bilaterally and fully in 10 animals. When this was done, stimulation of all pelvic afferents for 1 min inhibited LCED in 7 of the 10 animals. The frequency of spontaneous LCED also decreased from 30–170 Hz to 10–50 Hz. Duration of the response fluctuated between 2.4 and 20s (Fig. 2). The inhibitory pattern of LCED varied from animal to animal. This variation is thought to reflect contribution from vesical afferent stimulation, as has been reported by de Groat and Lalley (1972). The initial transient inhibition of LCED by PAS was always strong; that is, the impulse frequency in lumbar colonic nerves was reduced almost to zero, although the duration of this strongly inhibitory phase of the response to PAS was relatively short (between 2.4 and 12.0s) (Figs. 2A, 3). When the lumbar colonic efferent pathway to the rectum was partially preserved, the rectal contraction in response to stimulation of all pelvic afferent nerves was accompanied by an initial transient inhibition of LCED in 2 of 6 animals (Fig. 2C and D). Transsection of the spinal cord at T13 (2 animals) completely prevented the inhibition of LCED and also the simultaneous rectal contraction induced by stimulation of all pelvic afferent nerves. Cutting the spinal cord, however, did not facilitate LCED or did not cause rectal relaxation.

In contrast to the response to stimulation of all pelvic afferent nerves, stimulation only of rectal pelvic afferents (20 Hz, 2 ms, 14–16 V, 1 min) induced a constant inhibition of LCED in 8 of 9 animals (Figs. 4A, 5). Stimulation of rectal pelvic afferent nerves reduced the impulse frequency in lumbar colonic efferent nerves to one-third (25–30 Hz).

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**Fig. 1.** Effects of sectioning the brain stem on the pelvic-colonic reflex contraction in the rectum (A) and inhibition of lumbar colonic efferent discharge (LCED) (B) evoked by stimulation of all pelvic afferents except for those from the bladder (rPAS). Bilaterally the pelvic, hypogastric, and intermesenteric nerves were sectioned. Suprapontine transection (first arrow) and subsequently subpontine transection (second arrow), were performed (A). The time indicated was elapsed time after subpontine transection. The left trace in B was control LCED response by rPAS. The right trace in B was the response after subpontine transection. rPAS is designated by horizontal bars. Parameters of stimulation: 20 Hz, 2 ms, 14–17 V.