# CHANGES IN REFLEX RESPONSES OF THE SPINAL CORD DURING URINARY BLADDER STIMULATION UNDER NORMAL AND PATHOLOGICAL CONDITIONS

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In acute experiments on cats, stimulation of the mechanoreceptors of the urinary bladder (pressure 60-120 mm Hg) in most cases depressed reflex responses of centers for the flexor and extensor muscles and also the potential of the dorsal surface of the spinal cord. Afferent impulses from the inflamed urinary bladder strengthen monosynaptic reflexes of the extensor centers; reflex responses of the flexor centers are depressed after prolonged strengthening. Polysynaptic discharges are depressed under these circumstances. At the same time, changes are found in the dorsal surface potential and duration of posttetanic potentiation. In most experiments the electrical responses of the spinal cord to interoceptive stimulation are weakened if inflammation is present.

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Stimulation of visceral nerves or interoceptive zones causes changes in mono- and polysynaptic reflex responses of motor centers of the spinal cord [1, 8, 10, 14, 15, 17]. A number of investigators have analyzed the degree of participation of afferent structures and of mononeurons themselves in these changes [3, 4, 6, 8, 9].

In this investigation changes in reflex responses of the spinal cord under the influence of stimulation of urinary bladder mechanoreceptors were studied under normal conditions and in the presence of a local pathological process in the receptor zone.

## EXPERIMENTAL METHOD

Experiments were carried out on 27 cats anesthetized with urethane (1 g/kg intraperitoneally). After laminectomy in the lumbosacral region the animal was securely fixed to the metal frame. Monosynaptic discharges of centers for extensor muscles were recorded from ventral roots $L_7 - S_1$ in response to stimulation of the central ends of the divided nerves to the gastrocnemius muscle. To record reflex discharges of the centers for the flexor muscles, the deep branch of the peroneal nerve was used, and the divided dorsal roots of $L_6$ were stimulated. A current of above-threshold strength for fibers of the first group was used. The potential of the dorsal surface of the spinal cord (DSP) was recorded by a silver electrode from segment $L_7$, the second electrode being implanted into the vertebra. Single stimuli were applied to the ipsilateral nerve to the popliteus muscle. Single square pulses, 0.2 msec in duration, generated by a stimulator with radiofrequency output, were used for stimulation. Responses were recorded on a CRO with ac amplifier. To prevent drying and cooling, the spinal cord and nerves were covered with a layer of saturated mineral oil, the temperature of which, like the animal's body temperature, was maintained at 36-37° by means of an ultrathermostat.

Stimulation of the urinary bladder mechanoreceptors was carried out by inflating a rubber balloon (pressure 60-140 mm Hg) for 5 min. Acute inflammation of the mucous membrane of the bladder was produced by irrigation with 6% silver nitrate solution.

The numerical results were subjected to statistical analysis by the difference method [7].

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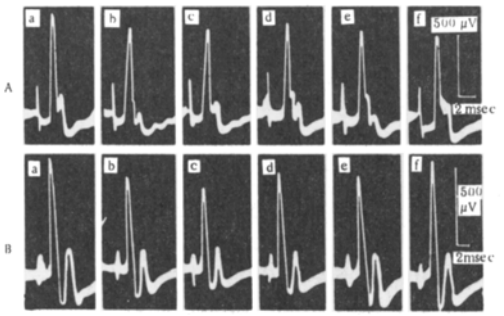


Fig. 1

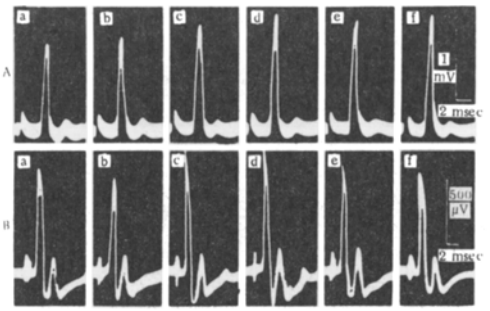


Fig. 2

Fig. 1. Changes in reflex responses of extensor (A) and flexor (B) centers of the spinal cord to interoceptive stimulation. a) Initial reflex response; b, c, d, e) responses 1, 2, 3, and 5 min respectively after beginning of stimulation of urinary bladder mechanoreceptors (pressure in balloon 120 mm Hg); f) 5 min after stopping interoceptive stimulation.

Fig. 2. Changes in reflex responses of centers for extensor (A) and flexor (B) muscles under the influence of impulses from inflamed urinary bladder. a) Initial reflex response; b, c, d, e, f) responses 30 min and 1, 2, 2.5, and 3 h after irrigation of urinary bladder mucous membrane with silver nitrate.

## EXPERIMENTAL RESULTS

In most experiments stimulation of the urinary bladder mechanoreceptors (pressure up to 120 mm Hg) caused very slight depression of monosynaptic extensor reflexes, the severity of which depended directly on the intensity of interoceptive stimulation. The effect usually reached a maximum after 1-3 min, when the amplitude of the responses was reduced by 10.8±3.4%; $P < 0.02$ (Fig. 1A). Strong interoceptive stimulation (140 mm Hg), on the other hand, was accompanied by a gradual increase in amplitude of the monosynaptic reflexes by 18.6±3.01% ($P < 0.001$). Polysynaptic reflexes recorded under these conditions in response to all intensities of interoceptive stimulation used in the experiment were reduced in amplitude. Changes in reflex responses persisted to different degrees for 7-15 min after the pressure in the balloon had returned to zero.

The amplitude of monosynaptic discharges in the reflex arc of the flexor muscles was reduced by stimulation of the urinary bladder (60 mm Hg) on the average by 12.1±4.9% ($P < 0.01$); the changes were significant only at the second minute of stimulation. During stronger distention of the bladder (120 mm Hg) inhibition of the reflexes was observed starting from 5-10 sec, and continuing throughout the period of stimulation, reaching a maximum at the third minute (amplitude reduced by 21.4±7.6%; $P < 0.05$). In the first minutes after stopping stimulation the amplitude of the monosynaptic reflex discharges returned to its initial level. Polysynaptic reflexes, after some initial depression, gradually strengthened; at the fifth minute of interoceptive stimulation their amplitude was increased by 20.3±7.4%; $P < 0.05$ (Fig. 1B).

In the presence of inflammation of the urinary bladder wall, in most experiments a distinct increase in amplitude of the monosynaptic discharges of centers for the extensor muscles was observed. Facilitation of the reflex responses appeared at the 10th-30th minute after irrigation with silver nitrate solution and usually persisted until the end of the experiment. Facilitation reached a maximum at the third hour (mean increase in amplitude 26±5.5%; $P < 0.001$ (Fig. 2A).

Afferent impulses from the urinary bladder associated with the presence of the pathological process in the organ also caused changes in reflex responses of the centers for the flexor muscles, and these were biphasic in character (Fig. 2B). During the first 1.5-2 h after production of inflammation, the amplitude of the monosynaptic discharges was increased by 20.4±6.7% ($P < 0.02$), and this was followed by development of gradually increasing inhibition of the reflexes. The amplitude of the responses 3-4 h later was reduced by 29.9±6.7% ($P = 0.001$). In most experiments changes in the polysynaptic reflexes were in the same direction.

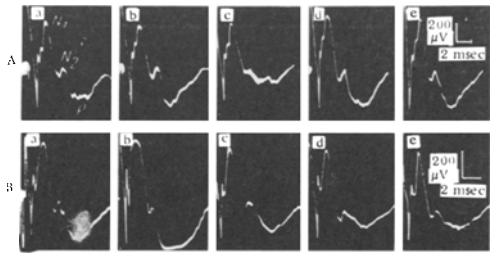


Fig. 3. Changes in potential of dorsal surface of spinal cord during interoceptive stimulation (A) and under the influence of impulses from the inflamed urinary bladder (B). In upper row: a) initial DSP; b, c, d) 10 sec and 1 and 5 min respectively after beginning of stimulation of urinary bladder mechanoreceptors (pressure in balloon 60 mm hg). In lower row: a) initial DSP; b, c, d, e) 1, 2, 3, and 3.5 h respectively after irrigation of urinary bladder mucous membrane with silver nitrate.

Stimulation of the pathologically changed receptive zone (urinary bladder) in most experiments caused less marked electrical responses of the spinal cord than in healthy animals. This weakening of the visceromatic reflexes was evidently due to depression of the functional state of the receptor apparatus of the urinary bladder.

The latent period of the spinal cord reflex responses was only very slightly changed by interoceptive stimulation under both normal and pathological conditions.

To determine the character of changes in the afferent parts of the reflex arc under the influence of a pathological process in the urinary bladder, the next stage was to study the potential of the dorsal surface of the spinal cord, reflecting presynaptic and postsynaptic processes in the interneurons [11-13]. In these experiments a typical multicomponent potential was recorded (Fig. 3).

Distention of the urinary bladder (pressure 60-120 mm Hg) led to an initial decrease in amplitude of the negative postsynaptic components ($N_1$ and $N_2$) and of the positive phase of the DSP, followed by an increase (Fig. 3A).

Under the influence of afferent impulses from the inflamed urinary bladder, during the first 1-2 h a marked increase (by 25-40%) in the amplitude of the negative components and the positive phase of the DSP was observed, although toward the end of the observation the amplitude of all its components (especially the positive wave) was below its initial value (Fig. 3B).

Changes in the duration of posttetanic potentiation of the monosynaptic flexor reflexes were also studied during development of the inflammation (stimulation of the same dorsal root at 200-400/sec for 10-15 sec was used). This phenomenon is connected with prolonged hyperpolarization of the presynaptic terminals of afferent fibers [16]. An increase of 1-4 min in the duration of potentiation was found in the phase of facilitation of the monosynaptic responses and a decrease by 1-3 min in the phase of depression.

As the facts described above show, stimulation of the urinary bladder mechanoreceptors under normal conditions most frequently have a very slight inhibitory effect on mono- and polysynaptic reflex responses and the DSP of the spinal cord (especially at the beginning of stimulation). Bearing in mind modern views of the origin of the DSP components [11-13], it may be considered that the observed interoceptive inhibition is presynaptic in nature. Such a suggestion has been put forward by other investigators [3, 4, 6, 10]. However, the possibility of participation of a mechanism of postsynaptic inhibition evidently cannot be ruled out here, and evidence in its favor is given by published reports of changes in the membrane potential of motoneurons themselves under the influence of stimulation of visceral nerves or of interoceptive stimulation [3, 4, 9].

During the development of inflammation of the urinary bladder wall, at first the monosynaptic reflexes were distinctly increased, regardless of whether they were evoked from a nerve to extensor or to flexor muscles, i.e., the effects of pathological stimulation of interoceptors are generalized. Only the duration of the facilitation effect differed: in the case of an extensor reflex the increase was more stable. Meanwhile an increase in amplitude of the negative components and of the positive phase of the DSP was observed, and at the same time the duration of posttetanic potentiation was slightly increased. Evidently, impulses from a focus of inflammation in the urinary bladder initially produce steady hyperpolarization of the primary afferent endings, improvement of transsynaptic transmission of excitation, and an increase in amplitude of reflex responses. However, 2-2.5 h after stimulation the amplitude of the monosynaptic discharges of centers for the flexor muscles decreased appreciably. Meanwhile a decrease was observed in all components of the DSP, especially its positive phase, together with a decrease in the duration of posttetanic potentiation. It may be considered that the initial hyperpolarization was replaced at this time by deepening and prolonged depolarization of the primary afferent elements. This suggests that the inhibition of reflex responses developing under these conditions is presynaptic in nature. Most probably restriction of the flow of afferent impulses from the pathological focus takes place at the level of the first synaptic relays.

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