EVIDENCE FOR INHIBITORY VASOMOTOR CONTROL AT THE SPINAL LEVEL

M. I. Dorokhova, O. S. Medvedev,
Ya. A. Reznikova, and V. A. Tsyrlin

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The possibility that inhibition of vascular tone may take place at the spinal level in response to afferent activation and to direct electrical stimulation of various structures of the spinal vasomotor center was investigated. Inflation of the urinary bladder and electrical stimulation of the spinal cord in the region of the ventral funiculi (at the lumbar level) in spinal animals can lead to depressor responses of the systemic arterial pressure, to a decrease in the perfusion pressure in the regional vessels, and to inhibition of electrical activity as recorded in the renal nerve.

Investigations [1, 2, 6-8] have shown that the segmental system can maintain neurogenic vascular tone and is the level at which vasomotor reflex arcs are closed. There is much less information about the possibility of inhibitory vasomotor control in spinal animals and such observations that have been made are highly contradictory [6, 9-11].

The object of this investigation was to examine whether inhibition can take place at the spinal level after afferent and direct stimulation of various structures of the spinal vasomotor center.

EXPERIMENTAL METHOD

Experiments were carried out on 24 cats in which the spinal cord was divided at the lower cervical level 24 h beforehand. In one series of experiments the tonic and evoked activity was recorded in one branch of the renal nerve in response to gradual stimulation of the interoceptors of the urinary bladder by inflation. In the other series the systemic arterial pressure and the perfusion pressure in the vessels of the fore- and hind limbs were recorded with the blood flow stabilized [5], and the volume velocity of the blood flow was recorded in the femoral veins with the aid of a drop counter and intervalograph [3] in response to unipolar electrical stimulation of various structures of the spinal cord (at the level L_{1-2}) through Constantan electrodes 50 μ in diameter (30-350 μA, 1-50 stimuli/sec, 1 msec) and to inflation of the urinary bladder. During the experiments the animals were artificially ventilated (after injection of muscle relaxants) and heated.

EXPERMENTAL RESULTS AND DISCUSSION

Inflation of the urinary bladder under a pressure of 20-60 mm Hg in five of 10 experiments led to a decrease of 5-10 mm Hg in the systemic arterial pressure (mean level 71 ± 11 mm Hg) after a latent period of 5-8 sec. The vascular tone of the hind limbs changed in various directions: in some experiments a pressor response of the vessels was observed (5-15 mm Hg), in others a depressor response. Marked dilatation of the limb vessels was confirmed also by the results of experiments to record the volume velocity of the blood flow. Inflation of the urinary bladder (Fig. 1) increased the blood flow in the hind limbs by 2-4 ml/min.

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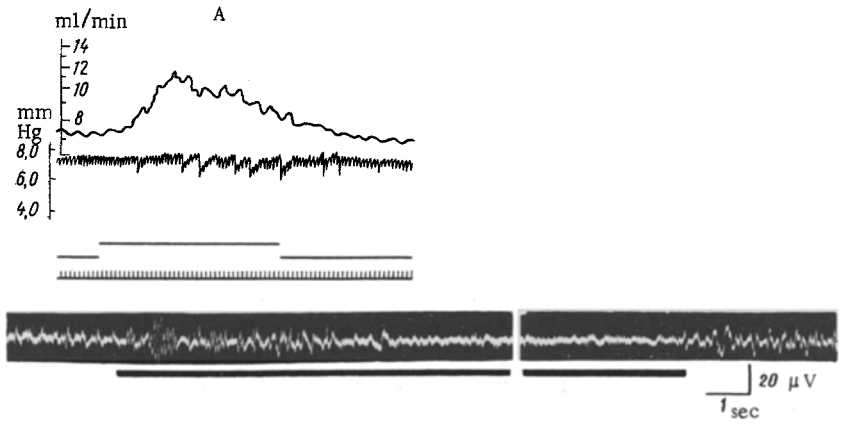

Fig. 1. Changes in volume velocity of blood flow in hind limb (A) and in electrical activity of renal nerve (B) in response to inflation of the urinary bladder: A) from top to bottom: volume velocity of blood flow in hind limb, systemic arterial pressure, marker of stimulation, time marker (1 sec). In B) black line under tracing is marker of stimulation (15 sec).

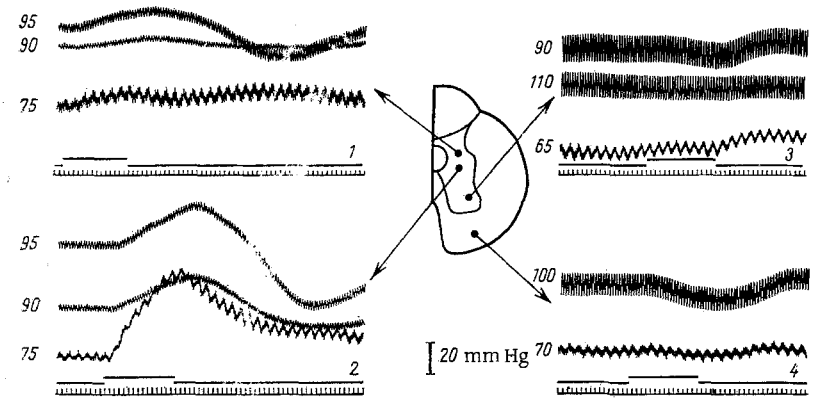

Fig. 2. Responses of limb vessels and systemic arterial pressure to stimulation of various spinal cord structures. On records 1-3, from the top to bottom: perfusion pressure in vessels of hind and forelimbs, systemic arterial pressure, marker of stimulation, time marker (1 sec). Numbers on records: values of original arterial and perfusion pressure.

Electrical activity in the renal nerve of the spinal animals is characterized by low-voltage discharges not synchronized with the pulse waves of arterial pressure. In three of five experiments a distinct dynamics of bioelectrical activity was observed in response to activation of the interoceptors of the urinary bladder (inflation under a pressure of 20-50 mm Hg). The increase in amplitude and frequency of electrical activity arising at the beginning of inflation (lasting 3-4 sec) was followed by considerable (Fig. 1) inhibition of activity, which persisted throughout the period of inflation. After the end of stimulation spontaneous electrical activity was restored.

If the stimulation electrode was localized in the intermediate zone of gray matter of the spinal cord biphasic responses (constriction followed by dilatation), characteristic of the hind limb vessels, appeared; the vasodilator response of the hind limb could amount to 30 mm Hg. With further advance of the electrode in the ventral direction and with its localization in the region of the anterior horns of the spinal cord, stimulation as a rule led to a decrease in the vascular resistance in both the ipsilateral and contralateral (relative to the position of the electrode) hind limb (Fig. 2). The decrease in perfusion pressure began 4-5 sec after the beginning of stimulation; in individual experiments the depressor response reached 25-30 mm Hg. Depressor responses were observed in spinal animals even after blocking of the peripheral cholinergic

receptors (oxyphenonium bromide, 2 mg/kg, intravenously). To exclude the possibility of activation of the descending vasodepressor fibers two experiments were carried out on chronic spinal cats (8 and 9 days after division of the spinal cord), in which depressor responses of the hind limb vessels also were obtained. It is interesting to note that the systemic arterial pressure, despite marked vasodilator responses in the hind limb, either was unchanged or showed a distinct pressor response.

These observations showed that processes of inhibition of vasomotor structures controlling vascular tone may be observed in spinal animals. The irregularity of appearance of depressor responses of the systemic arterial pressure and perfusion pressure in the regional vessels and their low value are evidently connected with a marked lowering of the original neurogenic tone in spinal animals. A decrease of neurogenic tone can be a cause of the absence of dilator responses of regional vessels in animals with an intact nervous system also [13]. There is as yet insufficient evidence to allow the character of inhibition in the segmental vasomotor structures to be determined. However, in preganglionic neurons, prolonged after-hyperpolarization does not take place [14] while in sympathetic ganglia direct inhibition is not found [4]. This suggests that dilatation of the regional vessels and depression of electrical activity in response to afferent activation are the result of direct inhibition of the spinal vasomotor structures.

The depressor regional responses to stimulation of the anterior horns of the spinal cord may be connected both with activation of the intrinsic inhibitory mechanisms of the spinal cord and with excitation of the descending "sympatho-inhibitory" tract [12]. The author considers that the dilator response of the blood vessels in the present experiments occurred through activation of true inhibitory mechanisms at the spinal level. Evidence in support of this conclusion is given by experiments on chronic spinal animals (the time after transection was sufficient to enable degeneration of the descending fibers) and by the possibility of the appearance of dilator responses (with simultaneous inhibition of electrical activity in the vasomotor nerve) in response to afferent stimulation. Ineffectiveness of the block of peripheral cholinergic receptors with respect to the appearance of depressor responses also rules out any possibility of the activation of cholinergic vasodilators.

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