EFFERENT ACTIVITY OF THE SPINAL CORD IN POSTISCHEMIC EXTENSOR CONTRACTURE OF THE HIND LIMBS

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Ventral root potentials in the caudal segments of the spinal cord were studied in acute experiments on cats with an extensor postischemic contracture. The latent period and amplitude of the monosynaptic potentials in response to stimulation of the dorsal roots in animals with a contracture were the same as in the control. The polysynaptic component of the ventral root response was absent after ischemia. Depression of the action potentials of the ventral root during repetitive stimulation in animals with contracture was less marked than in the control. The spontaneous activity of the motoneurons, a characteristic feature of these animals, did not disappear during single and repetitive stimulation of the afferent fibers and was increased after stimulation ceased. Prolonged circulation of impulses along neuron chains is one cause of the postischemic potentiation of motoneuronal activity.

Chronic inactivation of the interneurons in caudal segments of the spinal cord as a result of temporary ischemia leads to an extensor contracture of the hind limbs, accompanied by sensory and motor paralysis [6-10].

The object of this investigation was to study the mechanisms of this phenomenon, in the knowledge that it will shed light on the genesis of certain clinical contractures of central origin.

EXPERIMENTAL METHOD

Experiments were carried out on 24 cats weighing 2.5-3 kg. A marked contracture of the hind limbs was produced in 13 of the animals by compression of the abdominal aorta in the epigastric region for 25-30 min. The experiments were carried out 24 h after occlusion of the aorta under intraperitoneal pentobarbital anesthesia (40 mg/kg). Tracheotomy was performed, followed by laminectomy at the level L4-S1, and the nerves to the left hind limb were dissected. The animal was placed in a stereotaxic apparatus, and the spinal cord and dissected nerves were flooded with mineral oil warmed to 37°C. During the dissection and experiment the animal was heated.

The nerves and dorsal roots were stimulated through the isolating transformer of the stimulator of the Medicor electromyograph. The stimulus duration for single and repetitive stimulation was 0.5 msec. Segments L5, 6, and 7 of the spinal cord were tested. Ventral root potentials were detected by monopolar electrodes and recorded from the screen of the Medicor electromyograph.

EXPERIMENTAL RESULTS

The increase of extensor tone after occlusion of the abdominal aorta for 25-30 min began as soon as the animal came round from the anesthetic. Marked contracture of the muscles developed gradually over a period of several hours and led to the characteristic posture in which both extended limbs were crossed at the ankle. In some cases disturbances of the functions of the pelvic organs were observed.

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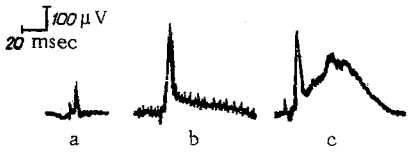

Fig. 1

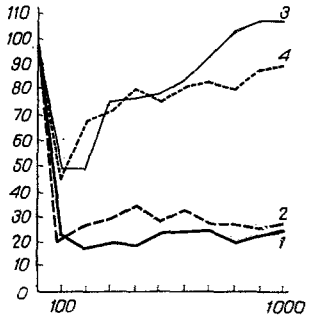

Fig. 2

Fig. 1. Responses of ventral root of segment L_7: a) to stimulation of nerve to gastrocnemius; b) to stimulation of dorsal root in animal with extensor contracture; c) to stimulation of dorsal root in a normal preparation.

Fig. 2. Dynamics of amplitudes of action potential (AP) of ventral root of segment L_7 during repetitive stimulation of dorsal root. Abscissa, time of tetanization, in msec; ordinate, amplitude of potentials in % of first AP; 1) frequency of stimulation 30/sec (control); 2) frequency of stimulation 100/sec (control); 3 and 4) the same, with contracture present.

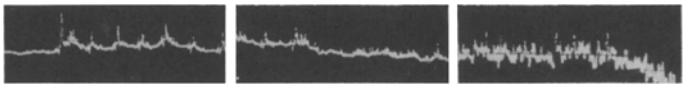

Fig. 3. Increased intensity of spontaneous discharges in ventral root after tetanization of peroneal nerve: a) beginning; b) end of tetanization for 1 sec at 50/sec; c) electrical activity of ventral root 1 sec after end of tetanization.

Pentobarbital anesthesia did not completely abolish the contracture of the muscles. Spontaneous discharges recorded in the ventral roots of the spinal cord subjected to ischemia [1, 2, 8] did not disappear, despite the use of anesthesia. These asynchronous and low-voltage (30-50 V) potentials persisted after division of the dorsal root. Spontaneous activity of the ventral root was unchanged in response to both single (Fig. 1a) and repetitive afferent stimulation.

In animals with an extensor contracture stimulation of mixed and muscular nerves evoked a low-amplitude response in the ventral root of the corresponding segment (Fig. 1b). In response to stimulation of the dorsal root a high monosynaptic potential appeared in the ventral root (Fig. 1c). The threshold intensity of stimulation under these circumstances was the same as in the control. Increasing the strength of stimulation by 3 to 4 times was not accompanied by any increase in the polysynaptic discharges as occurred characteristically in the control. In four experiments stimulation of the nerves and dorsal roots did not evoke ventral root responses in animals with a contracture and with disturbances of the functions of the pelvic organs.

The latent period of formation of the monosynaptic potential during dorsal root stimulation was 1.2 ± 0.04 and 1.3 ± 0.17 msec, respectively ($P > 0.5$), in segment L_7 of the animals after ischemia and in the control group. The amplitude of this potential in the animals with contracture was not significantly different from the control (355 ± 25 and 350 ± 25 V; $P > 0.5$).

During repetitive stimulation of the afferent inputs the amplitude of the monosynaptic potentials in the ventral roots was reduced in the control. This depression was characterized by a sharp initial decrease in the amplitude of the action potentials (AP) followed by a persistently low amplitude. In the animals exposed to temporary ischemia of the spinal cord, this depression was less marked [9].

The dynamics of the amplitudes of the monosynaptic potentials during and after tetanization for 1 sec at frequencies of 30 and 100/sec is shown in Fig. 2. By the end of tetanization at 30/sec not only was the

amplitude of the AP completely restored, but the magnitude of the first response also was increased. With high-frequency stimulation (100/sec) no restoration of AP amplitude to its initial level was observed at the end of tetanization. The weakening of depression may be due to blocking of the presynaptic mechanisms of inhibition [4, 5] or of delayed dendritic inhibition [3].

In some preparations the postischemic changes took the form that repetitive stimulation led to the appearance of asynchronous discharges which continued for a few seconds after the end of stimulation (Fig. 3). In animals with extensor contracture qualitatively similar changes were found in all segments of the spinal cord studied, but they increased in severity in a caudal direction. For instance, in segment L_7 during stimulation of the dorsal root the reflex response could be absent, in L_6 a small potential was recorded, but in L_5 of the same preparation a high-amplitude monosynaptic AP readily appeared. This shows differences in the degree of severity of injury to the caudal segments of the spinal cord after temporary ischemia and the possibility that extensor contracture of some muscles may coexist with flaccid paralysis of others.

The genesis of the postischemic contracture of the hind limbs is complex. Spontaneous discharges of motoneurons maintaining muscle tone arise chiefly in cells which have lost many of their intracentral connections. The strengthening of electrical activity recorded in the ventral root after repetitive stimulation evidently takes place as a result of the prolonged circulation of impulses around residual neuronal circuits. This is one cause of the postischemic strengthening of motoneuronal activity.

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