MICROPHYSIOLOGICAL ANALYSIS OF ELECTRICAL ACTIVITY OF SPINAL NEURONS OF VARIOUS TYPES IN EXPERIMENTAL TETANUS

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Generation of polysynaptic action potentials of motoneurons is facilitated in cats in the early stage of tetanus, while in a later stage they lose their ability to generate action potentials in response to monosynaptic excitation. In the latter case,the latent periods of monosynaptic excitatory postsynaptic potentials and of antidromic action potentials were considerably increased over the control. Background spike activity of internuncial neurons of the spinal cord showed no changes in the course of tetanus poisoning.

It is considered that in the late stage of tetanus disturbances of electrical activity are connected with degenerative processes developing in the motoneurons and cells of the spinal ganglia, reflected most clearly in function ring of the peripheral processes of these neurons.

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In the late stage of tetanus poisoning, a considerable slowing or cessation of axoplasm transport is observed in the thick medullated nerve fibers composing the sciatic nerve [3], and the fibers themselves become unable to conduct impulses rapidly and their lability falls sharply compared with the control. It has been suggested that these disturbances of function are due to degenerative processes developing in corresponding motor nerve centers under the influence of tetanus toxin.

It was therefore decided to study whether the electrical activity of different types of spinal neurons is disturbed in the course of tetanus poisoning.

EXPERIMENTAL METHOD

Experiments were carried out on healthy and poisoned cats. Local tetanus was produced by injection of tetanus toxin in a dose of 0.2 mg/kg (one lethal dose for mice is 0.00001 mg of the dry toxin) into the gastrocnemius and tibialis anterior muscles of the right hind limb. The animals were used in the experiment on the 3rd-4th day after poisoning (early stage of tetanus) or on the 9th-16th day (late stage).

The method of intracellular recording of action potentials (APs) of motor and internuncial neurons was described by the writers previously [4].

EXPERIMENTAL RESULTS

In the experiments of series I in the early stage of tetanus poisoning, the character of anti- and orthodromic excitation of α-motoneurons of the anterior horns of the spinal cord was studied after destruction of the corresponding nerves to the lower limb. At this stage, only facilitation of generation of polysynaptic APs compared with the control was observed, presumably as the result of disturbance of postsynaptic inhibition of the motoneurons [2, 5, 7, 8].

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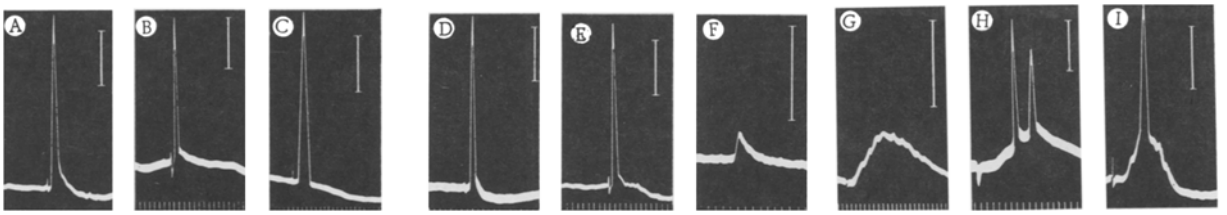

Fig. 1. Electrical activity of motoneurons in the course of tetanus poisoning. A, D, G—control: A) antidromic AP of motoneuron in nucleus of peroneal nerve, D) monosynaptic AP of same neuron, G) polysynaptic EPSP of motoneuron in nucleus of nerve to gastrocnemius muscle; B, E, H—early stage of tetanus poisoning; B) antidromic AP of motoneuron in nucleus of nerve to gastrocnemius, E) monosynaptic AP of motoneuron in nucleus of posterior tibial nerve, H) polysynaptic discharge of same neuron; C, F, I—late stage of local tetanus: C) antidromic AP of motoneuron in nucleus of posterior tibial nerve, F) monosynaptic EPSP of same neuron, I) polysynaptic AP of motoneuron in nucleus of nerve to gastrocnemius. Calibration: amplitude 25 mV, time 2 msec.

In the late stage of local tetanus, a similar investigation showed that, unlike the control, monosynaptic stimulation of motoneurons led to generation only of an excitatory postsynaptic potential (EPSP) of subthreshold amplitude, with a latent period increased (2.98 ± 0.02 msec) compared with the control (2.36 ± 0.03 msec; $P < 0.001$) (Fig. 1). To determine more precisely the degree of impairment of monosynpatic excitation of the motoneurons, experiments were carried out with stimulation of the central ends of the divided dorsal roots. In these experimental conditions, a monosynpatic AP could be recorded in each motoneuron on the intact side in response to stimulation of the corresponding dorsal root. On the side of tetanus, under the same conditions, only subthreshold EPSPs (4-7.5 mV) could be recorded in most motoneurons, and in about one-quarter of the neurons investigated, evidence of monosynaptic excitation could not be found.

The study of antidromic excitation of motoneurons in the late stage of local tetanus showed that, despite the ability of all investigated motoneurons to generate an antidromic AP, its latent period was increased. Lengthening of the latent period of the antidromic AP (up to 1.95 ± 0.02 msec) was particularly marked in cases when peripheral nerves were stimulated at a distance of 10-15 cm from the spinal cord (1.47 ± 0.03 msec in the control; $P < 0.001$). During stimulation of the ventral roots, on the other hand, the duration of the latent period of the antidromic AP showed hardly any increase (0.33 ± 0.01 msec, control 0.3 ± 0.008 msec).

The most significant changes in electrical activity of the α-motoneurons were thus observed only in the late stage of local tetanus. Under these circumstances disturbance of the function of the thick afferent nerve fibers [3] led to a defect of monosynaptic excitation of the motoneurons and lengthening of the latent period of monosynaptic EPSPs. The fact that the soma of the motoneurons in the late stage of local tetanus can generate an AP of the same amplitude as in the control in response to antidromic and polysynaptic excitation shows that tetanus toxin does not produce any gross changes in the level of polarization and excitability of the cell, despite appreciable disturbance of its trophic function relative to the axon.

The results described indicate that mechanisms of injury of central parts of the motor innervation of skeletal muscles in tetanus differ significantly from those in botulism [4], and attempts to regard them as identical [10] cannot be justified.

To assess the state of function of the internuncial neuron system, an investigation was made of their background spike activity, one of the characteristic features distinguishing this group of nerve cells [1, 6, 9].

These experiments showed that the character of background activity of the spinal neurons showed no significant change compared with the control in either the early or late stages of tetanus poisoning. This evidently indicates that the internuncial neurons are not subjected to the pathogenic action of tetanus toxin.

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