A NEW FORM OF TETANUS HYPERRFLEXIA: THE PATHOLOGICALLY INCREASED SCRATCH REFLEX

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In rats with relatively local tetanus poisoning of the cervical and upper thoracic segments of the spinal cord, produced by migration of the toxin along the regional neural pathways of one of the forelimbs, application of a stimulus to the receptive fields of this limb evokes a response of the ipsilateral hind limb, in the form of a pathologically increased scratch reflex. It is postulated that this pathological response is due to the action of tetanus toxin on interneurons in the arc of the scratch reflex located in the cervical and upper thoracic segments of the spinal cord.

In animals with ascending tetanus produced by injection of tetanus toxin into the muscles of one of the hind limbs the application of a stimulus to that limb is accompanied by a generalized paroxysmal response of the extensor type, whereas stimulation of all other receptive fields evokes only a local response ("departure station" phenomenon) [1, 2].

It was interesting to investigate the character of the motor response in animals in cases in which the "departure station" of the tetanic paroxysmal activity is created in the brachial and not in the crural portion of the spinal cord [3, 4, 5].

EXPERIMENTAL METHOD

Experiments were carried out on 100 albino rats. The "departure station" phenomenon was produced by injecting tetanus toxin in a dose of 3-4 MLD into the triceps brachii muscle and with simultaneous injection of antitetanus serum (0.025 a.u.) With this method of poisoning the tetanus toxin travels along the regional neural pathway [1] into the brachial segments of the spinal cord.

The responses of the rats to natural stimuli (touch, pressure, pinching) in various receptive fields were investigated. Analysis of the sequence and character of the rapid movements was made by taking motion pictures at a speed of 48 frames/sec (the "Krasnogorsk" motion-picture camera). In some animals visual assessment of the motor response was supplemented by recording of the electrical activity of the muscles on an 8-channel encephalomyograph (San'Ei). The electromyogram (EMG) was recorded in animals with an intact spinal cord and after cordotomy at the level C_2. Uninsulated steel needles were used as the recording electrodes.

EXPERIMENTAL RESULTS

On the 3rd-5th day the muscles of the limb into which the toxin was injected, the muscles of the neck and upper part of the chest on the side of injection of the toxin, and also some muscles of the contralateral forelimb were rigid. The digits of the "tetanic" paw were clenched into a fist. Rigidity of the hind limbs

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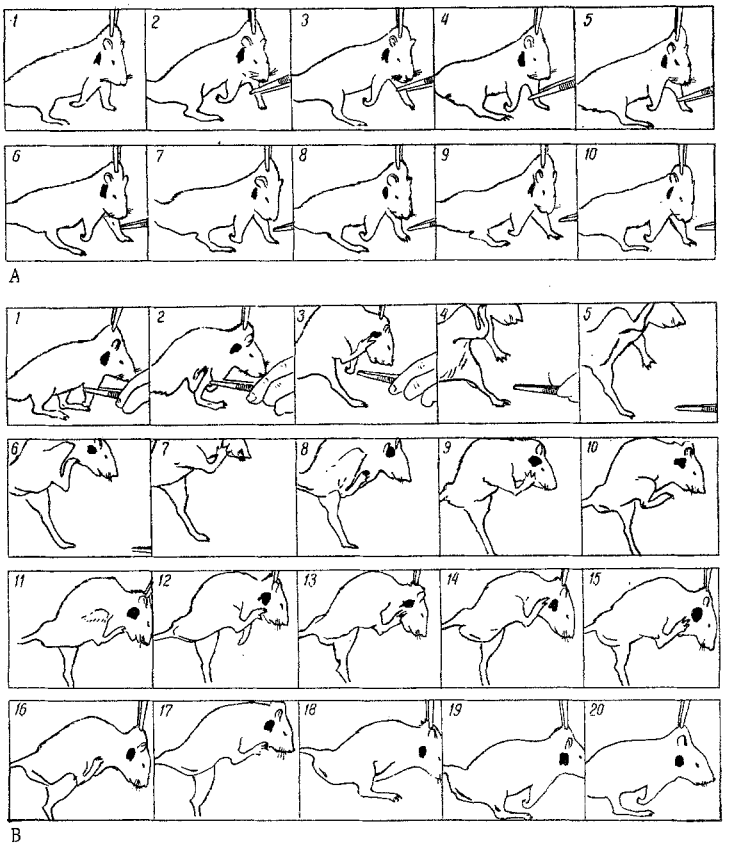


Fig. 1. Motor response of rat with "departure station" phenomenon evoked by stimulation of the forelimbs. Response: A) to stimulation of forelimb on side opposite to injection of toxin; B) to stimulation of forelimb into which tetanus toxin was injected.

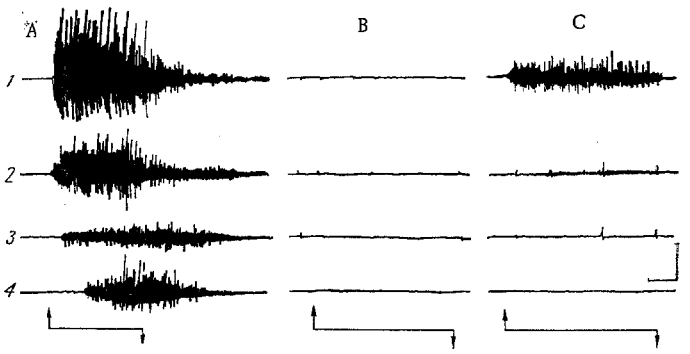
was absent. The responses of the hind limbs to stimulation of the receptive fields of these limbs and their participation in the animals' walking were undisturbed.

The animal's response to stimulation of the receptors of the "tetanic" forelimb and of the forelimb on the side opposite to injection of the toxin was different. This is clear from the frames of the motion pictures recording the animal's behavior during stimulation of the forelimbs. Compression of the forelimb with forceps on the side opposite to injection of the toxin evoked only a small change in the animal's posture (Fig. 1A). Stimulation of the same intensity applied to the "tetanic" forelimb was accompanied by an energetic response of many of the body muscles. The trunk was flexed toward the "tetanic" side, the contralateral hind limb was extended, the ipsilateral hind limb was drawn towards the anterior part of the body, and it performed regular scratching movements. The toes and foot of the ipsilateral hind limb performed particularly fast and frequent movements (Fig. 1B).

In the early stage of poisoning movements of the hind limb were well coordinated and aimed at removing the stimulus applied to the "tetanic" forelimb. If the rigidity of the muscles of the anterior part of the body was slight, the scraping movements were directed precisely at the stimulated "tetanic" limb in the late stage of poisoning also (Fig. 2D).

The later stage of poisoning differed from the earlier by the following features of the scratch response. The duration of the motor activity was much longer than that of stimulation or, in other words, the response was characterized by a prolonged aftereffect (Fig. 1B; Fig. 2A, D). It will be clear from Fig. 1B (frames 4–17) that after the end of stimulation the scratching movements continued for a further 1.5 sec.

In the late stage of poisoning bursts of paroxysmal scratching movements began to appear "spontaneously." Repeated scratching caused damage to the skin (Fig. 1), and sometimes of all the underlying tissues down to the bone (Fig. 2D). If major blood vessels were damaged the animals quickly died from massive bleeding.



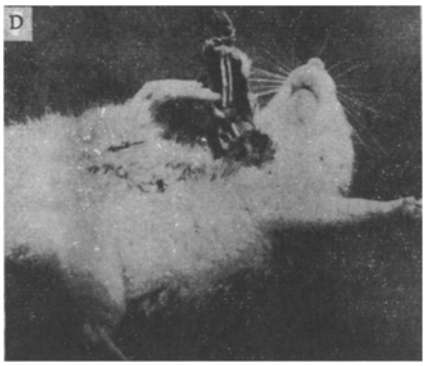


Fig. 2. Activity of hind limb muscles in a rat with "departure station" phenomenon. Tetanus toxin injected into triceps muscle of left forelimb. EMG of hind limb muscles recorded on side of injection of toxin: 1) tibialis anterior, 2) gastrocnemius; and on side opposite to injection of toxin: 3) tibialis anterior, 4) gastrocnemius. A) Strong compression of "tetanic" forelimb; B) the same, but stimulus applied to forelimb opposite to injection of toxin; C) as in A, but 1 h after cordotomy at level C_2. Time intervals during which forelimb is stimulated marked by segments of straight line beneath records of EMG, beginning and end of stimulation indicated by arrows. Calibration: signal 0.5 mV, time 1 sec; D) motor response of rat evoked by stimulation of "tetanic" forelimb.

Progressive rigidity of the muscles of the anterior part of the body made it impossible to oppose the hind limb to the stimulated "tetanic" forelimb. As a result the areas of injury arising on account of the vigorous scratching movements were found on the medial or lateral surface of the arm, on the neck, or on the head (Fig. 1).

It was interesting to study the level of closure of scratch reflex studied. Experiments showed that precollicular decerebration did not abolish the pathologically increased scratch reflex. In animals with chronic (3 days before injection of the toxin) ipsilateral hemisection at the level C_2 an increased scratch reflex arose at the same times as in the rats with an intact spinal cord, and the response was just as intensive.

In the acute spinal rat (transection at the level C_2) stimulation of the receptors of the "tetanic" forelimb did not evoke a scratch response but by recording the EMG it was possible to detect some increase in the activity of those flexor muscles in which it had predominated before cordotomy (Fig. 2).

The electromyographic data on predominant activation of flexor muscles obtained in experiments on animals fixed to a frame are in agreement with results of the analysis of movements of the unfixed animal. It is clear from Fig. 1B (frame 2) that the first response of the rat to stimulation of the "tetanic" forelimb was flection of the ipsilateral hind limb.

It follows from the remarks made above that the motor response arising in rats with a "departure station" phenomenon to stimulation of the "tetanic" forelimb is similar to the scratch reflex of the healthy animals. Differences between the responses of the animal in the early and late stages of poisoning (in the intensity and number of clonic movements, the duration of the aftereffect) were analogous to differences between the strong and weak scratch reflex. Predominant activation of the flexors and, in particular, the flexors of the ankle is characteristic of the scratch reflex under normal conditions also [7].

The experiments with transection in the CNS demonstrate the spinal origin of this response, in agreement with data in the literature on the spinal origin of the normal scratch reflex [7].

Special electrophysiological investigations confirmed the preservation of inhibition of the lumbar motoneurons and the uniform character of reflex influences of the brachial division on lumbar motoneurons in rats whose scratch reflex was tested and also in healthy animals [3-5].

Since inhibition of lumbar motoneurons in animals with a pathologically increased scratch reflex was not suppressed by the toxin, the increase in the response can be explained only by the action of the toxin on nonmotor elements in the reflex arc of the scratch reflex located in the cervical and upper thoracic segments.

Consequently, the type of tetanus hyperreflexia described above differs in principle from the well known hyperreflexia in local tetanus, which is based on the suppression of motoneuronal inhibition by the toxin [6, 7].

Some important conclusions follow from this fact. The action of tetanus toxin on nonmotor elements of the reflex arc may be an essential factor in the intensification of reflexes in tetanus. The special character of the reflexes intensified by the toxin is determined by the morpho-functional properties of the affected reflex arcs.

It has been postulated previously that wide irradiation of excitation and other features of the "universal departure station" phenomenon [1, 2] are attributable to the specific type of spread of excitation in a system with diffuse, yet relatively effective synaptic connections [5]. The unilateral character and precise exhibition of the pathological scratch reflex can be explained by the unilateral character and high efficiency of the long-axon propriospinal pathways responsible for the spinal scratch reflex.

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