SUPRASPINAL CONTROL OVER SPINAL REFLEX PATHWAYS DURING WALKING

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The tonic flexor reflex to injury of the limb can be evoked in decorticated cats only when the animal is in a state of rest. As soon as the animal starts to walk, it cannot maintain flexion; the injured limb is excluded from walking and the flexor attitude gives way to extensor. The same result is found in response to electrical stimulation of a cutaneous nerve. The authors consider that descending influences of the central nervous system evoking locomotion inhibit spinal reflexes from cutaneous afferent fibers to flexor motoneurons.

Previous investigations showed that injury to the limb evokes a tonic flexor reflex, not only in animals with an intact nervous system, but also in decorticated and decerebrate animals [2-4]. Until recently, however, one important factor was lost sight of. Injury to the limb in an animal with an intact nervous system evokes a tonic flexor reflex which is of equal intensity when the animal is in a state of rest or is moving. As the experiments described below show, such an injury to the limb in decorticated animals evokes a tonic flexor reflex only if the animal is in a state of rest. During walking the flexor reflex is replaced by an extensor reflex. The same result was obtained in response to electrical stimulation of the cutaneous nerve (sural nerve) of one hind limb. Stimulation evoking a flexor reflex in an animal in a resting state evoked an extensor reflex as soon as the animals started to walk.

EXPERIMENTAL

Cats were decorticated under ether anesthesia. The carotid arteries were ligated. The dura was opened through a burr-hole in the skull and decortication carried out by Khananashvili's method [5]. At the end of the operation the administration of ether was stopped and the experiment began 2 h later.

The animal's head and vertebral column were fixed in a stereotaxic apparatus. The main plane of the bench was strictly horizontal so that the head was in a position in which influences from the labyrinth and neck on the limb muscles were minimal. The animal's lower limbs rested on the belt of a treadmill; when the belt was set in motion, these preparations began to perform spontaneous locomotion [6]. Electrical activity of the hind-limb muscles was recorded by needle electrodes. The limb was injured by injection of 1 ml hypertonic sodium chloride solution subcutaneously into the foot. The sural nerve was exposed under the tendo Achillis.

EXPERIMENTAL RESULTS AND DISCUSSION

In agreement with the previous experiment, injection of hypertonic sodium chloride solution subcutaneously into one hind limb of the decorticated animal in a state of rest evoked a tonic flexor reflex which lasted up to 10-15 min.

However, as soon as the animal began to walk, it could not maintain a flexor attitude of the limb. During active locomotion the flexor attitude was replaced by extensor. This extensor attitude was so strongly marked that it was difficult to flex the limb passively.

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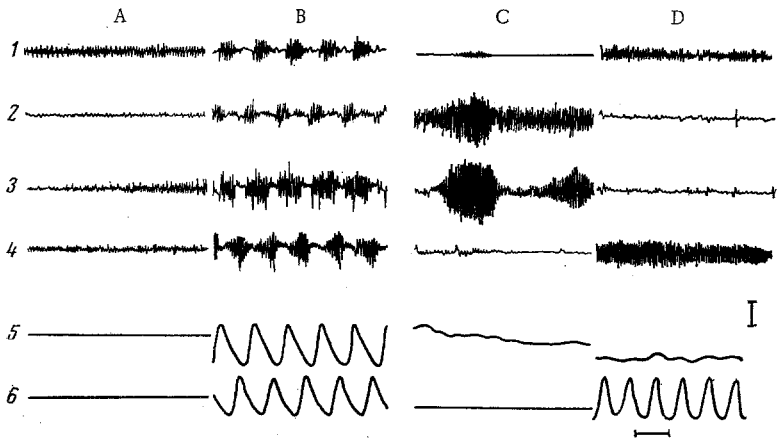

Fig. 1

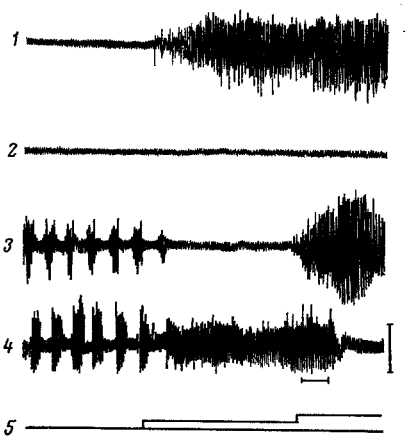

Fig. 2

Fig. 1. Mechanograms and EMG of limb muscles of decorticated cat before (A, B) and after (C, D) subcutaneous injection of hypertonic salt solution into the dorsal surface of the left hind limb: 1,4) EMG of extensors (gastrocnemius, vastus lateralis muscles); 2,3) EMG of flexors (gracilis, biceps femoris posterior muscles); 5,6) mechanograms of left and right hind limbs. Remainder of explanation in text. Calibration 500 μV, time 1 sec.

Fig. 2. EMG of limb muscles during stepwise increase in strength of stimulation of sural nerve: 1,3) EMG of flexor (biceps femoris posterior); 2,4) EMG of extensor (vastus lateralis); 1,2) EMG in animal at rest; 3,4) EMG in animal during locomotion; 5) marker of stimulation of sural nerve. Frequency of stimulation 100 Hz. Remainder of legend in text. Calibration 500 μV, time 1 sec.

The results of one experiment of this series are demonstrated in Fig. 1. When the treadmill belt was stationary and the animal in a state of rest (Fig. 1A) the mechanograms (A:5,6) showed absence of limb movements, while the electromyogram (EMG) of the flexors (A:2,3) and extensors (A:1,4) recorded muscular activity which corresponded to that position of the limb. When the treadmill belt was set in motion and the animal began to walk (Fig. 1B) cyclic movements of the hind limb were recorded on the mechanograms (B:5,6), with corresponding volley activity in the flexors (B:2,3) and extensors (B:1,4) on the EMG. The treadmill belt was then stopped and the animal was once again in a state of rest (Fig. 1C). Injection of 1 ml hypertonic sodium chloride solution subcutaneously into the dorsum of the foot evoked a flexor reflex of the limb and corresponding electrical activity in the flexor muscles (C:2,3), while electrical activity in the extensor muscles was absent C : 1,4). The treadmill belt was then again set in motion (Fig. 1D). Instead of the flexor reflex, an extensor reflex developed in the injured limb, with corresponding electrical activity in the extensors (D:1,4). No electrical activity was present in the flexors. Cyclic movements were recorded on the mechanogram of the uninjured limb (D:6). The injured limb did not take part in the walking movements (D:5).

The same pattern was observed in response to electrical stimulation of a cutaneous (sural) nerve. Stimulation which evoked a flexor reflex in an animal at rest evoked an extensor reflex in the same animal during movement (Fig. 2). When the animal was at rest (Fig. 2:1,2) the electrical stimulation of the sural nerve (of threshold strength and above, frequency 50-150 Hz) evoked a tonic flexor reflex. Strong electrical activity was recorded on the electromyogram of the flexor (Fig. 2:1), but activity was absent in the EMG of the extensor. The treadmill belt was then set in motion and the animal began to walk (Fig. 2:3,4). Volley activity corresponding to walking was recorded on the EMG of both flexor and extensor (initial segments of records 3 and 4). The sural nerve was then stimulated, using stimuli of the same parameters as those which evoked a flexor reflex at rest. When the strength of stimulation was 2-3 thresholds for the flexor reflex an extensor reflex appeared. Electrical activity was recorded in the extensors, but was absent in the flexors (middle segments of records 3 and 4). Only an increase in the strength of stimulation above four thresholds again evoked a flexor reflex in the animal: electrical activity was recorded in the flexor, no activity in the extensor (final segments of records 3 and 4).

There are reports in the literature of the conditions under which stimulation of cutaneous afferent fibers can induce a flexor or extensor reflex.

According to Sherrington [7], digital pressure on the strongly flexed limb of a spinal animal evokes an extensor jerk instead of flexion. Beritashvili [1] evoked an increase or decrease in the tone of the limb muscles by turning the head of a decerebrate animal. In the presence of strong extensor tone stimulation of a sensory nerve induced a flexor reflex, while weak stimulation induced an extensor reflex. Beritashvili explains the results of these experiments on the basis of Yxkuell's rule: centers of stretched muscles are activated in response to reflex stimulation.

The response to reflex stimulation of an afferent nerve may depend on the strength of stimulation. For instance, weak electrical stimulation of the radial nerve evokes an extensor reflex, but stronger stimulation of the radial nerve leads to the appearance of a flexor reflex [7]. The localization of the stimulus is also significant. During stimulation of the skin over the flexors mainly flexion develops, while stimulation of the skin over the extensors leads mainly to extension [8].

Predominance of the flexor or extensor reflex may also depend on the level of decerebration or transection of the spinal cord [9, 10]. In the present experiments all these conditions were ruled out. A flexor or extensor reflex appeared when the influence of reflexes from the labyrinth and neck on tone of the limb muscles was prevented, when the parameters of stimulation were the same, when the localization of stimulation was the same, and when the brain was divided at the same level (decortication). This suggests that in these experiments a fundamentally novel factor arose which would convert the flexor reflex into extensor, namely, active locomotion.

Analysis of this phenomenon must await further research. However, even at this stage it can be considered that one cause of the replacement of the flexor reflex by extensor during walking is that descending influences from the brain evoking locomotion inhibit the reflex pathways from cutaneous afferent fibers to flexor motoneurons.

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