CONTINUITY OF THE BULBAR LOCOMOTOR STRIP IS NOT ESSENTIAL FOR WALKING

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Bilateral destruction of the medulla, interrupting both "locomotor strips," was carried out on mesencephalic cats. After the operation walking could still be induced by stimulation of the more rostral (and also caudal) portion of the strip, although in some experiments the threshold of walking was raised. The connection of the "locomotor strip" with other structures of the brain stem and spinal cord is discussed on the basis of these findings.

KEY WORDS: medulla; locomotion; locomotor strip.

Electrical stimulation of a certain part of the brain stem induces the mesencephalic cat to walk on a treadmill. Points whose stimulation induces walking form the "locomotor strip" (LS), which stretches at a distance of 4 mm from the sagittal plane along the whole of the hindbrain – from the midbrain to the spinal cord [2, 5]. LS contains neurons giving synaptic responses to stimulation of adjacent portions of LS [3], although this does not rule out the presence of axons directly connecting the brain stem with the spinal cord in it. If this axonal component of LS plays a significant role, after interruption of LS stimulation of its more rostral portion ought not to induce locomotion. It is shown in the investigation described below that bilateral destruction of the bulbar LS does not abolish the locomotor effect of stimulation of its more rostral portion.

EXPERIMENTAL METHOD

Precollicular decerebration was performed on cats under ether anesthesia after tracheotomy and ligation of the carotid arteries. The ventral boundary of the incision passed between the mamillary bodies and the point where the third pair of cranial nerves emerges [1]. Part of the occipital bone and the tentorium cerebelli were removed. The cat's head was fixed and its body suspended so that the limbs touched the belt of the treadmill.

Stimulating electrodes (tungsten wire 20 μ in diameter, insulated with glass, outer diameter of electrode 40-100 μ) were introduced into the midbrain and medulla about 4 mm away from the sagittal plane. Monopolar stimulation was carried out with square pulses, of negative polarity, 0.2 msec in duration for the bulbar LS and 0.4 msec for the midbrain, with a frequency of 60 Hz. The electrode was moved in a dorsoventral direction until a point was found whose stimulation with a current of under 40 μA induced walking [2, 5, 3]. This point was then destroyed by passing a direct current through it, after which walking was again induced by stimulation of a locomotor point in the midbrain or in another part of the medulla. Next, in most experiments the symmetrical locomotor point was located in the medulla, it also was destroyed, and attempts were then made to induce walking by stimulation of the midbrain or other parts of LS. Altogether 22 experiments were carried out.

The location and size of the injuries were verified in photographs of sections cut on a freezing microtome. The Horsley–Clarke coordinates were taken from a stereotaxic atlas [4].

EXPERIMENTAL RESULTS

Neither unilateral nor bilateral destruction of the bulbar LS (P 11-13) prevented walking from being induced by stimulation of the locomotor point in the midbrain (P2) or of another point in LS, no matter whether it was situated rostrally (P 7-8) or caudally to the lesion (five experiments).

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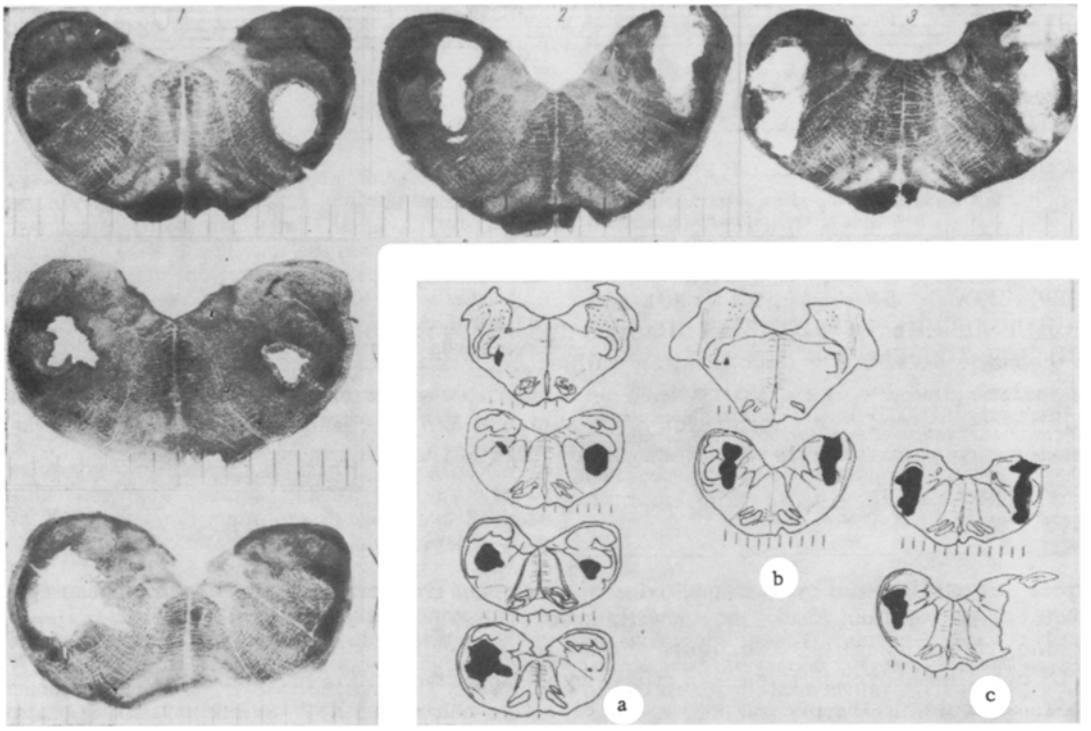

Fig. 1. Bilateral destruction of medulla at level P 11-13 following which walking was induced by stimulation of more rostral points of LS. 1, 2, 3) Frontal sections through medulla of three preparations; a, b, c) corresponding schemes. In top schemes a and b, coagulation tags indicate points whose stimulation induced walking after destruction of the medulla. 1) Corresponds to bottom schemes (a), 2) to bottom scheme (b), 3) to top scheme (c).

Foci of destruction of the medulla are indicated in Fig. 1 (three experiments). In one experiment (Fig. 1, 1) stimulation through the left electrode 16 mm from the surface of the cerebellum induced walking with all four limbs (25 μA), whereas at a distance of 16.5 mm from the surface walking was induced similarly but by a current of 35 μA. Stimulation through the right electrode 15.5 mm from the surface of the cerebellum induced dropping of the lower jaw and weak stepping movements with all four limbs (30 μA), at a depth of 16-17 mm it induced walking with all the limbs (20 μA), and at 17.5 mm walking changing into rapid spastic contraction of all the limbs (40-45 μA); with a smaller current no walking developed. After electrolytic destruction of all points whose stimulation induced walking (Fig. 1, 1a, the bottom three schemes) stimulation of the left LS 5 mm rostrally to the center of the left focus of destruction induced walking with all the limbs (25-30 μA; Fig. 1a, top scheme). The same effect was obtained in response to stimulation of a region 4 mm caudally to the focus of destruction (30 μA).

In two other experiments (Fig. 1) bilateral destruction was carried out at points stimulation of which by a current of 20-30 μA induced walking with all the limbs. Subsequent stimulation (35 μA) of a point located 4 mm rostrally to the left focus (Fig. 1, 2b) and stimulation (30 μA) 4 mm rostrally and caudally to the right focus (Fig. 1, 3c) induced walking by all the limbs. In both experiments walking could be induced after destruction by stimulation of the midbrain (25 μA in experiment 2, 100 μA in experiment 3).

Stimulation at levels P9-14 and in the midbrain (P_2) continued to induce walking even after destruction in the caudal part of the medulla (P16-18, four experiments). The foci of destruction carried out on the left at a point whose stimulation (25 μA) induced walking with all four limbs, and on the right induced walking complicated by general spasticity (20-25 μA), are shown in Fig. 2a. Destruction on the right was more extensive. After destruction stimulation at level P11 on the left (25 μA) induced stepping movements with the forelimbs, whereas on the right as before it induced locomotion complicated by general spasticity (20-25 μA). Foci of destruction produced in the other experiment are shown in Fig. 2b (bottom schemes). After them, walking could also be induced by stimulation of the more rostral point (top scheme), although the threshold of walking was increased from 40 to 60 μA.

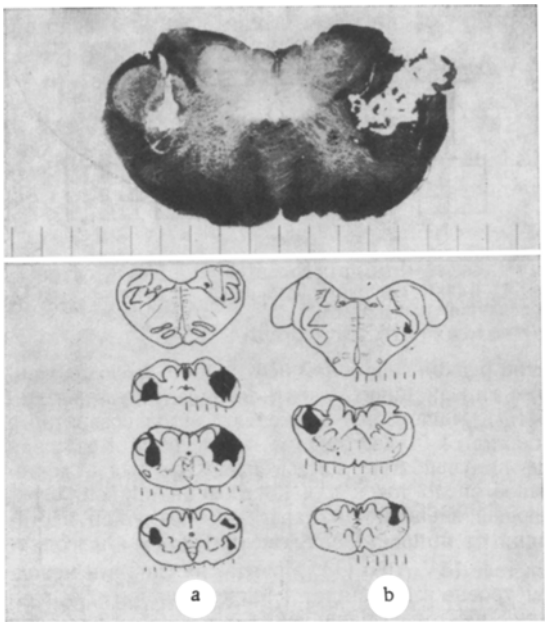

Fig. 2. Bilateral destruction of medulla at level P16-18, after which walking could be induced by stimulation of more rostral points of LS. a, b) Schemes for two different experiments. Top schemes show tags of points (on right) stimulation of which induced locomotion after destruction. Photograph corresponds to second scheme from bottom in (a).

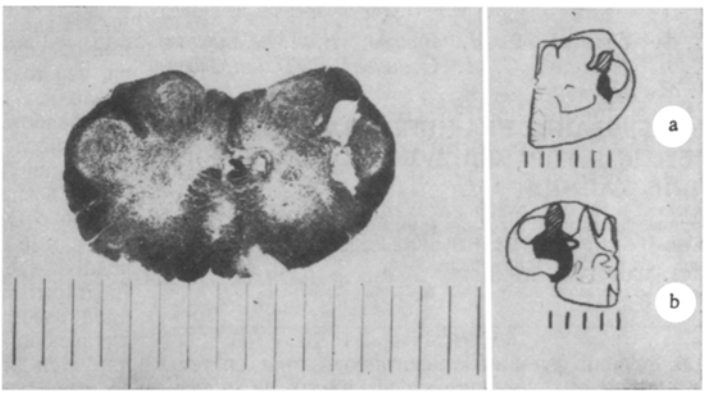

Fig. 3. Two coagulations carried out on one track (P17) at points whose stimulation induced walking. After destruction, stimulation on either side at level P 14 induced locomotion with all the limbs. a) Scheme of section shown in photograph; b) similar scheme from another experiment.

In four experiments the effect of destruction of the locomotor points on the effectiveness of stimulation of a neighboring point on the same track was studied (Fig. 3). To begin with coagulation was applied at a point whose stimulation induced walking without any other motor effects, after which a more dorsal point was stimulated. In one experiment stimulation at a depth of 13.5-14 mm from the surface of the cerebellum induced walking by all the limbs (in this case the forelimbs were spastic) and dropping of the lower jaw (15 μA). At a depth of 14.5 mm only opening of the mouth was observed, but at a depth of 15 mm walking took place without any side effects (13 μA). After coagulation at a depth of 15 mm (see the photograph in Fig. 3) stimulation at a depth of 14 mm induced stepping movement by the forelimbs, complicated by spastic contraction of their muscles (16 μA).

Locomotion induced by stimulation of the point dorsal to that from which walking could be induced without any motor side effects was usually accompanied by dropping of the lower jaw or by general spasticity (on more medial tracks). Often stepping movements were carried out only by the forelimbs, but noncyclic spastic contraction developed in the muscles of the hind limbs.

Destruction of the dorsal point (located within the spinal nucleus of the trigeminal nerve or on its dorsal boundary) had no effect on walking induced from the bulbar LS rostrally to the foci of destruction or from the midbrain.

In three experiments after unilateral destruction at level P 18, affecting an extensive region (including funiculi) ventrally to the spinal nucleus of nerve V, ipsilateral stimulation at more rostral levels did not induce locomotion. In one experiment stimulation of the midbrain (25-30 μA) induced walking after extensive ipsilateral destruction at level P 17-18 (Fig. 3b), but the hind limb on the side of stimulation did not participate. After extensive contralateral destruction, affecting a large region ventrally to the spinal nucleus of nerve V, stimulation no longer induced walking.

Stimulation at level P 16-18 (13-25 μA), incidentally, could induce walking with all the limbs, not just stepping movements by the ipsilateral hind limb [2]. In four cats, in which the ventral boundary of the incision through the brain stem passed along the anterior border of the pons, stimulation of LS also induced walking. In four experiments walking could be induced by stimulation at level P 11-12 even in animals with an incision in the brain stem passing through the middle of the superior colliculi and cutting off the anterior quarter of the pons.

Bilateral destruction of the bulbar LS at level P 11-13 thus did not prevent walking from being induced from other parts of the brain stem. The possibility of inducing locomotion still remained even after destruction at levels P 16-18, although the threshold of locomotion in some experiments was raised. However, in other experiments, after extensive destruction (even unilateral) it was impossible to induce locomotion.

The results of these last experiments was evidently determined not by interruption of the strip, but by injury to the pathways through which the various descending system of the brain stem exert their tonic influence on the spinal cord.

It must also be pointed out that in five experiments unilateral destruction of LS at level P 12-18, on the other hand, led to a shift in the animal's condition toward greater readiness to walk. Now it was sufficient to set the treadmill belt in motion for stepping movements to be induced even without electrical stimulation of the brain stem.

The possibility of induced locomotion by stimulating a point rostrally to foci of bilateral injury of LS is evidence against the view that LS is a descending tract running directly into the spinal cord and giving off no collaterals to structure in the brain stem. The results are more in agreement with the hypothesis that LS is a column of neurons connected not only with each other [3], but also with neurons located outside LS. These neurons can provide a "bridge" across the injured region of LS (its rostrocaudal extent in these experiments was usually 1-2 mm) or can transmit impulses to the spinal cord along pathways avoiding LS.

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