REFLEX COMPENSATION AND DECOMPENSATION OF MOTOR FUNCTIONS AFTER PARTIAL INJURIES TO THE SPINAL CORD*

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(Received May 15, 1955. Presented by Academician L. A. Orbeli)

Inasmuch as compensation of lost functions in various parts of the central nervous system is a universal phenomenon, it follows that the restoration of such functions, to a greater or smaller degree, must involve the participation not only of the cerebral hemispheres (with regard to conditioned reflex mechanisms), but also of the lower levels of the central nervous system (with regard to unconditioned reflex mechanisms). E. A. Asratyan [1] believes that following partial damage to the spinal cord compensation of sensory-motor functions can be achieved without the active participation of the cerebral hemispheres, by means of the intrinsic mechanisms of the spinal cord itself, but no special study of this possibility has been made. We have attempted to elucidate this problem, by inflicting minor injuries on the spinal cord, applying various functional loads [3], and then removing the forebrain after achievement of compensation of motor functions.

EXPERIMENTAL METHODS

Incomplete lateral transection of the spinal cord, to a depth of $^1/_4$ of its diameter, was performed at T_4 in 4 rabbits, at C_{10-11} in 14 pigeons, and of the thoracic or lumbar segments of 28 frogs. The operations were performed aseptically, under ether anesthesia. Clinical observations were then made of impairment and recovery of reflex reactions to stimulation of a number of receptor fields, and impairment and recovery of motor functions were studied in detail. Changes in the rheobase and chronaxie of motor nerves of the upper and lower extremities of pigeons, on both sides, and before and after the operation, as well as during the course of development of compensatory reactions were followed.

In determining chronaxie in pigeons, the birds were immobilized in the same position in all cases (on their backs, on a special table). One electrode is inserted into the anus, and the other is placed on the upper or lower limb, in the region of the median or peroneus nerves (Fig. 1). The response reaction from the lower extremities was shown by movements of the middle claw (Fig. 2, 4), and of the upper extremities by movements of the first-order feathers of the appropriate wing (Fig. 2, 1, 2, 3). After full compensation of motor functions had been achieved, we examined the decompensatory effects of small doses of narcotics, motor loading, cooling of the lower limbs, and removal of the forebrain.

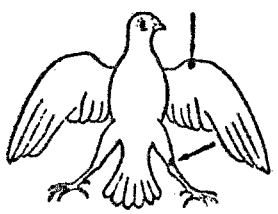

Fig. 1. Sites of application of the recording electrodes (indicated by arrows).

EXPERIMENTAL RESULTS

Injury to and recovery of motor functions. All four rabbits were unable to stand on the day following lateral transection of a quarter of the diameter of the spinal cord at T_4. They lay on the side on which transection was performed.

*The experiments on pigeons were performed in the Department of Physiology of the Central Nervous System of the V. M. Bekhterev Brain Institute under the guidance of E. A. Asratyan in 1939-1941.

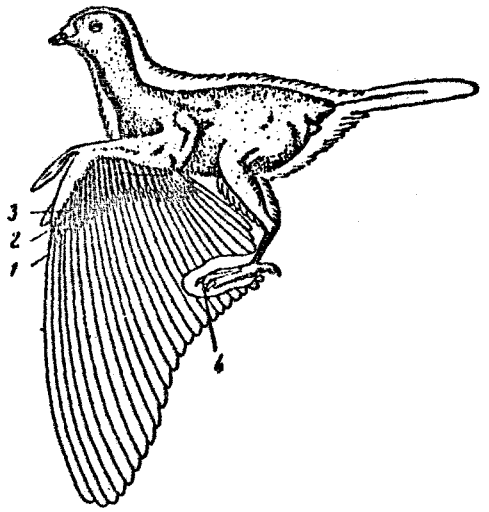

Fig. 2. Diagrammatic representation of the position of the extremities and feathers of a pigeon. Indicators of motor functions were movements of the middle claw (4) of the legs, and of the flight feathers (1,2,3) of the wings.

After 2-3 days they were able to stand, and to walk a little, with a lameness of the hind leg on the operated side; the reflex reaction from this leg was diminished.

Only after 20-22 days did the locomotor functions of the rabbits become normal.

The pigeons were able to stand on the day following the operation, and some even to walk, falling, however, towards the operated side; after 2-3 days they all walked, with only a slight limp on the side on which the spinal cord had been partly transected. They were not able to fly without being thrown up into the air, and could not balance on their perches. After 9-10 days they walked well, flew, and alighted correctly.

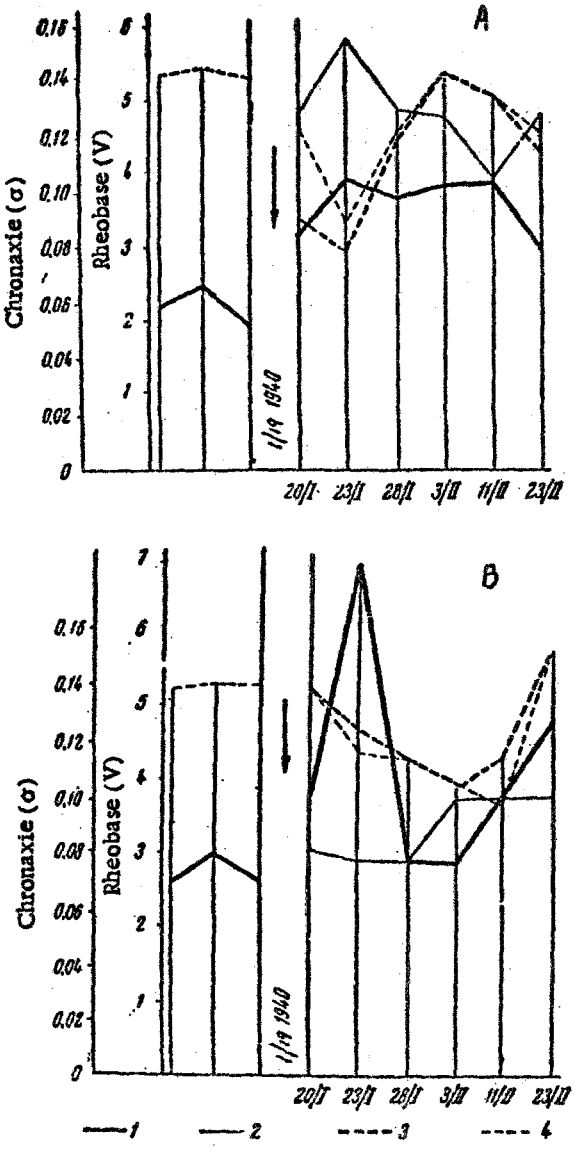

Fig. 3. Changes in rheobase (1 - injured side, 2 - intact side) and chronaxie (3 - injured side, 4 - intact side) of the motor nerves of the legs (A) and wings (B) after lateral transection (↓) of the spinal cord to a depth of $^1/_4$ of its diameter.

Considerable changes took place in rheobase and chronaxie after the operation. Preoperationally, the rheobase and chronaxie of the motor nerves of both wings and legs were at the same level. On the second day after the operation the rheobase of the motor nerves of both legs was enhanced, to a greater degree on the operated side. For the following 3 or more days the rheobase diminished, but did not revert to the initial level even a month later, although compensation of motor functions had been achieved after 9-10 days. Chronaxie of leg motor nerves was at first shortened on both sides, and then lengthened during the following 3 days, reaching the initial level after 14-15 days. The rheobase of the wing motor nerves, on the other hand, at first rose on the unoperated side, and fell slightly on the operated one. Later changes in the rheobase were in most cases inconstant. Wing motor nerve chronaxie varied in general in the same way as for the legs, although the initial level was regained much later (after 22-25 days). After the operation, reflex reactions of the leg to mechanical (pinching) or electrical (induction current) stimuli were at first weaker on the operated, and stronger on the unoperated, side. Similar effects were not clearly evident in the wings.

With recovery of motor functions (after 15-20 days), a levelling off of the reflex reactions of both legs took place. This time coincided with that of restoration of the original rheobase and chronaxie levels.

No marked chantes in locomotor function were observed in frogs after transection of $^1/_4$ of the diameter of the spinal cord. They walked and jumped, with only very slight trailing of the hind leg on the operated side. The latent period of the reflex to application of 3% acid to the leg on the operated side was markedly prolonged in most cases (from 10-12 seconds). After 3-4 days the frogs no longer trailed the affected hind leg, and reflex time reverted to the initial value.

Study of stability of the compensatory mechanisms. We have shown in our earlier papers [2,3] that even after full recovery of lost functions of animals subjected to lateral hemitransection of the spinal cord, decompensation is readily brought about by the agency of various "provocative" measures, such as small doses of narcotic substances (alcohol, ether, chloral hydrate, sodium amytal, etc.), muscular effort, cooling, and others.

We found that when the transection affects only a quarter of the cross section of the spinal cord, application of the same "provocative" measures did not lead to decompensation of function. The only differences from the initial state found in operated pigeons after application of narcotics or of muscular effort were in the rheobase of the motor nerves of both legs, which rose by 1-1.5 v, and in their chronaxie, which was prolonged by 0.04-0.06 σ (with hemitransection of the spinal cord we found marked differences for these values between the operated and the intact sides).

It thus appears that, notwithstanding full recovery of motor functions, application of "provocative" measures to animals which have suffered relatively serious injury to the spinal cord (hemitransection) reveals a residual instability of the compensatory mechanisms, which persists for a long time, and renders them vulnerable to stress. These compensatory mechanisms possess much greater stability when the injury to the spinal cord is of smaller degree.

Decompensation of motor functions caused by ablation of the forebrain.

If one cerebral hemisphere is removed from a pigeon following full recovery of motor functions after hemitransection of the spinal cord, partial loss of the recovered functions is observed, while ablation of both hemispheres causes total loss of these functions. When only a quarter of the diameter of the spinal cord is transected, removal of a cerebral hemisphere after full development of compensatory mechanisms has practically no decompensating effect. Subsequent ablation of the remaining hemisphere causes very little further deterioration. Simultaneous ablation of both hemispheres does, however, cause considerable decompensation of motor functions. During the first few days after the operation, some of the pigeons lie on the side on which partial spinal transection had been performed, while others sit on their shanks, leaning over to the same side. After 6-8 days, however, the pigeons were able to stand on their feet, only very seldom falling over to the affected side, with the wing slightly drooping on the same side. They began to walk after 10-12 days, limping slightly on the side of the spinal injury, and with considerable drooping of the wing on the same side; when thrown into the air they fall flat. The wing defect disappeared after 20-22 days, and the leg defect after 30-35 days. The pigeons began to fly without assisted takeoff only after 78-85 days.

Rheobase and chronaxie levels were determined for the motor nerves participating in reflex reactions of the legs and wings of pigeons after ablation of the forebrain, following recovery from lateral transection of the spinal cord, to a depth of $^1/_4$ of its cross section. No clear-cut differences were found between the values on the operated and intact sides, in contrast to birds which had been subjected to a spinal hemitransection.

Frogs were subjected to spinal hemitransection and, after recovery of motor function, the fore and intermediate brains were removed in two stages, at an interval of 8-10 days. This led to considerable disturbances of motor function (they rarely jumped, and they trailed a leg on the side of the spinal injury). These motor defects were not seen when the spinal cord was transected to a depth of only $^1/_4$ of its cross section, but simultaneous extirpation of the fore, intermediate, and the midbrain did affect motor function only, however, in causing restriction of voluntary movements (trailing of a leg was not seen). Reflex time was somewhat prolonged (2-3 seconds), but soon reverted to normal.

We thus see that lateral transection of the spinal cord to a depth of $^1/_4$ of its diameter causes disturbances of motor function, which are most pronounced in mammals, less so in birds, and least of all in amphibiae. Recovery of lost functions also proceeds more rapidly in amphibiae and birds than in mammals.

Whereas application of various types of functional stresses (motor stress, cooling, narcotics, and others) to animals which have fully recovered motor functions lost after spinal hemitransection causes recurrence of the previous defects, pointing to the instability of the compensatory mechanisms, practically no such effect is found when the transection involves only $^1/_4$ of the cross section of the spinal cord. In frogs, even one-stage removal of the fore, intermediate, and midbrain does not result in full recurrence of the previous defects.

The conclusion may be drawn from our experiments that compensation of lost motor function following partial transection of the spinal cord is achieved basically from the resources of the spinal cord itself, viz. unconditioned reflex mechanisms. Since the spinal cord is more stable than are the higher levels of the central nervous system, it is natural that it is more difficult to cause recurrence of previous defects (decompensation of function) by means of "provocative" measures than when compensation of function is developed largely in conjunction with the forebrain, by a conditioned reflex mechanism, as with more extensive injuries to the spinal cord.

LITERATURE CITED

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[3] V. D. Dmitriev, Fiziol. Zhur. SSSR 39, No. 3, pp. 293-299 (1953).

*In Russian.