THE NATURE OF SPINAL SHOCK

COMMUNICATION I. THE STATE OF HYPERPOLARIZATION IN SPINAL SHOCK

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It was shown by Hermann as long ago as 1868 – 1872 that the excited area is electrically negative and the resting area electrically positive [10, 11]. In experiments by I. M. Sechenov [5, 6], stimulation of motor and sensory nerves was accompanied by a negative fluctuation of current led from the surface of the spinal cord in a longitudinal direction or from a transverse section of the spinal cord. During stimulation of cutaneous nerves, and also during voluntary movements of the limbs in animals, B. F. Verigo [2] observed that the brachial or lumbar enlargement of the spinal cord becomes electrically negative in relation to the midbrain. Similar results were obtained also by N. A. Mislavskii [4]. Among modern work may be mentioned the research of V. E. Delov and D. A. Lapitskii [3], who also showed a decrease in the resting current of the spinal cord (negative wave) in response to faradic stimulation of the sciatic nerve. In individual experiments by these authors, when the resting current showed the reverse trend, stimulation of the sciatic nerve caused a small increase in the resting current of the spinal cord (positive wave).

In connection with the development of the classical teaching of N. E. Vvedenskii on parabiosis [1], one of the foremost diagnostic indications of stationary focal excitation – parabiosis – was the negative electrical potential of the parabiotic area. The method of measurement of the electrostatic potentials of the spinal cord before, during and after chordotomy permits the study of the trends of the polarization of the spinal cord during spinal shock and to test the concept of the parabiotic nature of this state, as put forward by several workers.

In the present investigation we studied the potential difference between the spinal cord and the denervated sural muscle in association with chordotomy.

EXPERIMENTAL METHOD

Laminectomy was performed in a frog (Rana chensinensis D.), and the spinal cord exposed in the region of the lumbar and brachial enlargements. The experiment was commenced after one hour or later, when the frog's pose, jumping and defensive movements appeared to be within normal limits. The frog was fixed securely in the prone position on a cork board on a small table. The sciatic nerve was divided in the right hind limb, the vessels ligated and the sural muscle dissected out.* The tendon at the distal end of the denervated sural muscle was divided and the muscle half immersed in a bath of Ringer solution, in which also hung the wick of a nonpolarizing electrode (Zn + $ZnSO_4$). The thigh, knee-joint and paw of the right limb were firmly fixed through bone to the cork with pins. Such fixation ensured constant contact of the wick with the Ringer solution and the muscle. In some experiments we added 3% agar-agar to this solution to fix the wick in the gel thus formed. The wick of a second nonpolarizing electrode was firmly held in place by adhesion with a fibrin clot to the surface of the spinal cord or to one of its roots. For contact with the root, a loop was made from the wick of the

* The muscle was denervated to exclude the influence of the central nervous system on the demarcation current of the muscle [7 – 9].

Relationship Between Changes in Polarization and Reflex Excitation of the Spinal Cord After Chordotomy

Reflex excitation	Polarization of the spinal cord			
	increase of polarization	no change	relative depolarization	total
Spinal shock	35	2	4	41
No change	17	11	5	33
Increase of reflex excitation	6	1	11	18
Total	58	14	20	92

electrode, through which the root was threaded. The frog, together with the cork-topped table was placed in a Pfluger screened humid chamber; the interior of the chamber was abundantly moistened with water at room temperature. The potential difference between the spinal cord and muscle was indicated by the light on the scale of a mirror galvanometer, with a constant of $1.4 \cdot 10^{-10}$ A/ mm/m, a period of damping of 4 seconds, and R_{kr} = 40,000 ohm. In order to equalize the resistance of the external circuit we included a resistor of 20,000 ohm in parallel with the frog.

The potential of the muscle, with rare exceptions, was electrically negative, whereas the potential of the spinal cord was nearly always positive. Usually the potential difference of the resting current fell slowly and gradually. The change in the voltage of the current taking place in the course of the first minutes after chordotomy was probably due entirely to changes in the potential of the spinal cord, since the denervated sural muscle was unable to react in any way to this change of potential. In the course of the experiment, at the same time as we recorded the potential difference we also measured the threshold of reflex stimulation of the left semitendinosus muscle in centimeters of the induction coil. Electrodes from the induction coil were fixed with sticking plaster to the skin of the dorsal surface of the foot of the left hindlimb. The index of contraction of the left semitendinosus muscle was movement of the Engelman lever situated outside the chamber. In order to prevent any effect of the stimulating current on the loop, an earthed metal ring is placed over the leg of the left hindlimb. Chordotomy was performed above the brachial enlargement of the spinal cord. In the first 63 experiments the spinal cord was divided with a scalpel, but later with a woman's hair which was carefully introduced beneath the cord and tied around it in a loop. A thread, attached to the hair, was passed through a glass tube to the outside of the humid chamber. By pulling on the thread, without opening the lid of the chamber and without altering the humidity of the air inside it, we could rapidly sever the spinal cord. This method of division to a large extent guaranteed against displacement of the electrodes at the time of chordotomy and avoided contact of metal with the spinal cord; however the introduction of the hair beneath the spinal cord sometimes inflicted additional trauma which affected the results of the experiments.

EXPERIMENTAL RESULTS

In all 128 experiments were carried out, including 17 controls. In the latter, observing all the conditions described above, chordotomy was performed with a hair on dead frogs. In these experiments the conditions for fixation of the wick of the electrode on the surface of the brain were less satisfactory, because no fibrin clot is formed in dead frogs.

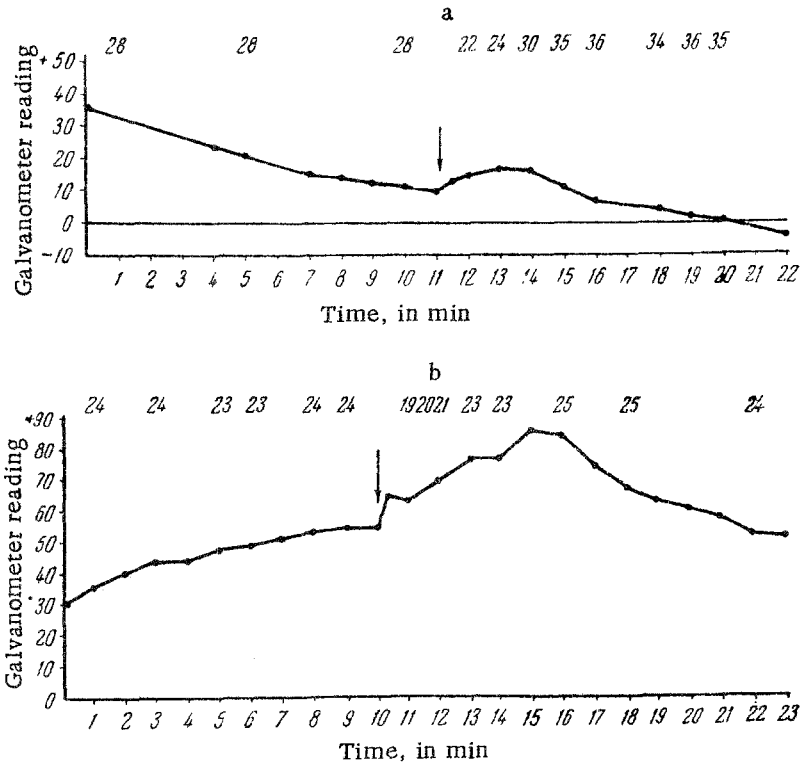
The results of 92 experiments were included in the analysis, nineteen being excluded on account of general traumatic shock and death of the frog after chordotomy, with manifestations of a progressive fall in reflex excitation.

The basic results of the investigations are shown in the Table. It can be seen from the Table that in the great majority of cases (58 experiments out of 92) chordotomy is accompanied by the increase of polarization of the spinal cord, i. e. by anelectrotonic changes. Under these circumstances the reflex excitation is more often diminished, showing the picture of spinal shock. In the minority of cases (20 of the 92 experiments) relative depolarization of the spinal cord (catelectrotonic shift) is accompanied by mainly an increase in reflex excitation.

In order to discover the degree of significance of the connection which was found between the changes of reflex excitation and the polarization of the spinal cord after chordotomy, we used the "chi-square" method, calculating the coefficient of contingency C. Analysis showed that for the given experimental results "chi-square" was equal to 35.00, and $p < 0.01$. Consequently $C = 0.61$, i. e. the existence of a connection between spinal shock and increased polarization of the spinal cord, and also between the increased reflex excitation and relative depolarization of the spinal cord is definite enough and statistically significant.

Spinal shock was observed in 41 experiments. In 35 of these experiments, in which spinal shock was combined with increased polarization of the spinal cord, reflex excitation was restored after 1 – 6 minutes (on the average after 2.9 minutes) to the original level (28 experiments) or above, into a state of exaltation (7 experiments). Spontaneous recovery of the failing reflex excitation in this way is an absolute sign of spinal shock. It must be pointed out that in 30 experiments we found complete agreement in time between the duration of spinal shock and the development of an anelectrotonic state of the spinal cord (from 1 to 7 minutes, on the average 2.9 minutes) this suggests a functional link between these phenomena (see Figure). It is not without interest that in 4 frogs spinal shock coincided with relative depolarization of the spinal cord.

In 19 of 111 experiments (about 18% of cases), as we have already stated, the frogs developed general traumatic shock. In these cases, as a rule (in 17 frogs), chordotomy caused a transient (from 30 seconds to 3 minutes) increase in polarization, after which the galvanometer indicator light began to move steadfastly along the scale, indicating the increasing depolarization of the spinal cord. At the same time a general areflexia developed, with paralysis of respiration, and the heart stopped in systole.



The influence of chordotomy on the polarization of the spinal cord and the threshold of reflex excitation.
a) Experiment dated March 8; b) experiment dated March 10. The upper row of figures indicates the value of the threshold as the distance in cm between the induction coils. The arrow indicates the moment of chordotomy. Below – the zero line of the galvanometer scale.

The chordotomy which was performed in 16 of the 17 frogs which died produced no changes of any sort in the potential difference; before and after chordotomy it remained at the same level. Only in one experiment was there a rise in the potential difference between the anterior root of the spinal cord and the muscle. Direct excitation of the spinal cord was absent. The cause of the change in potential in this experiment remained undetected.

Summarizing the results obtained it may be stated that:

1. High chordotomy causes spinal shock in 39% of frogs, and in 20% of cases a primary increase of reflex excitation.

2. Statistical analysis of the results by the "chi-square" method demonstrates with sufficient precision and reliability the existence of a connection between spinal shock and the increase of polarization (anelectrotonic state) of the spinal cord, and also between an increase in reflex excitation and relative depolarization (catelectrotonic state) of the spinal cord.

3. The duration of spinal shock (from 1 to 6 minutes, on the average 2.9 minutes) in 30 experiments corresponded in time to the rise in polarization (from 1 to 7 minutes, on the average 2.9 minutes).

4. It must be assumed that general traumatic shock is associated with the development of deplorization at different levels of the central nervous system.

5. Chordotomy, when performed on dead frogs, causes no changes in the general dynamics of the polarization current.

SUMMARY

One hundred and twenty-eight experiments were conducted on frogs. The difference of electrostatic potentials (rest current) between the exposed surface of the spinal cord and the denervated muscle was measured. Chordotomy carried out below medulla oblongata increased the polarization of the spinal cord in 58 experiments (of the 92) and caused relative depolarization in 20. Statistically authentic correlation was established between the increase of polarization of the spinal cord and development of spinal shock, as well as between the relative depolarization and the increase of the reflex excitation ($X^2=35.00$, $p<0.01$). General traumatic shock (19 experiments) is connected with increasing depolarization of the spinal cord. Chordotomy carried out in dead frogs (17 control experiments) does not change the electrostatic potentials.

LITERATURE CITED

[1] N. E. Vvedenskii, "Excitation, inhibition and anesthesia." Complete Collected Works, vol. 4, Leningrad State University Press (1953).*

[2] B. F. Verigo, Vestnik Klin. i Sudebn. Psikhiatr. i Nevropatol. No. 7, 82 (1889).

[3] V. E. Delov and D. A. Lapitskii, Transactions of the Brain Institute, pp. 70 – 78, (1935).*

[4] N. A. Mislavskii, Vrach (Kazan'), No. 5 (1894).

[5] I. M. Sechenov and M. Pflüg., Arch. 25, 281 (1881).

[6] I. M. Sechenov and M. Pflüg, Arch. 27, 524 (1882).

[7] M. N. Farfel', Byull. Eksptl. Biol. i Med. 23, 6, 419 (1947).

[8] M. N. Farfel', Byull. Eksptl. Biol. i Med. 24, 3, 183 (1947).

[9] L. G. Khrolinskii, Fiziol. Zhur. SSSR 40, No. 4, 472 (1954).

[10] L. Hermann, Untersuchungen zur Physiologie der Muskeln und Nerven I – III, Berlin, 1868.

[11] L. Hermann, Grundrisse der Physiologie, 1872, 4, Aufl. S. 323.

* In Russian.