RELATIONSHIP BETWEEN THE CONSTANT POLARIZATION POTENTIALS OF SKELETAL MUSCLE AND THE LEVEL OF TRANSECTION OF THE BRAIN AND SPINAL CORD IN FROGS

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The reflex activity of the muscular system of the hind limbs varies depending on the level of transection of the brain or spinal cord. I. M. Sechenov showed originally [3] in a series of experiments that the duration and depth of depression of the spinal reflexes are dependent on the level of section of the cord.

The investigations of G. N. Sorokhtin [4, 5] showed that after transection of the spinal cord a distinctive state of "atony of the nervous center"—i.e., of passive hyperpolarization—develops in the centers lying below the point of transection, as a result of deprivation of their usual supraspinal impulses. A similar state of passive hyperpolarization also arises on the skeletal muscle [1, 2, 4] when it is excluded from central influences by means of transection, and also by blocking of its nerve with procaine or cold.

The object of the present investigation was to discover what changes take place in the constant polarization potentials (CPP) of the gastrocnemius muscle of the frog as a result of transection of the brain and spinal cord at different levels, and also to determine whether passive hyperpolarization can develop after denervation of a muscle if this is performed on a spinal or bulbar frog.

EXPERIMENTAL METHOD

Experiments were carried out on grass frogs (Rana temporaria) in which the brain or spinal cord was exposed. Transection was carried out during the experiment at different levels: between the brachial and lumbar enlargements of the spinal cord (low cordotomy), below the medulla (high cordotomy), and above the medulla. The CPP were measured in the left gastrocnemius muscle by means of a nonpolarizing electrode, which served as indicator. A second nonpolarizing electrode, lying on the bones of the right lower limb which had previously been denervated and excluded humorally from the general circulation, was taken to represent zero. The CPP which were being studied were compensated by means of a type P-307 high-resistance potentiometer. A mirror galvanometer with a sensitivity of $10^{-9}$ A/mm/m was used as null apparatus.

EXPERIMENTAL RESULTS

Regardless of its level, transection of the brain and spinal cord always caused a brief (3-4 sec, on the average) period of depolarization as a result of the descending stream of impulses from the wound. Following this traumatic depolarization, in most experiments an increase in the electropositive potential was observed (see table).

It is clear from the data given in the table that the more caudal the level of transection, the more frequent the development of passive hyperpolarization, the longer its duration, and the greater its magnitude. In the case of the tested muscle, this relationship may be explained, we consider, by the size of the deficit of pulsed excitation corresponding to the level of transection of the brain or cord. Denervation of the gastrocnemius muscle in the intact frog caused, in 78-80% of cases, a well marked passive hyperpolarization, attaining 2-4 mV and sometimes lasting for several hours (Fig. 1, A). These results are in agreement with those found by other authors [1, 2]. Denervation, creating a maximal deficit of excitation for the muscle, also produced the maximal passive hyperpolarization. The
Effect of Level of Transection of Brain or Spinal Cord on Development of Passive Hyperpolarization of Gastrocnemius Muscle

<table>
<thead>
<tr>
<th>Level of transection</th>
<th>Total number of experiments</th>
<th>No. in which hyperpolarization developed</th>
<th>Duration of increase in electro-positive potential (in min)</th>
<th>Magnitude of electro-positive potential (in mV)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>max-</td>
<td>min-</td>
</tr>
<tr>
<td>Low cordotomy</td>
<td>27</td>
<td>20</td>
<td>20</td>
<td>4</td>
</tr>
<tr>
<td>Below medulla</td>
<td>27</td>
<td>17</td>
<td>16</td>
<td>5</td>
</tr>
<tr>
<td>Above medulla</td>
<td>27</td>
<td>15</td>
<td>10</td>
<td>4</td>
</tr>
</tbody>
</table>

Deficit of excitation after low cordotomy was greater still, for the influences from the anterior portion of the spinal cord and all the impulses from the brain were lost. After low cordotomy, passive hyperpolarization was observed in 74% of cases, but by comparison with denervation of the muscle it was weaker and shorter in duration (Fig. 1, B).

High cordotomy created a smaller deficit of excitation for the gastrocnemius muscle. As a result, passive hyperpolarization was observed less frequently in the muscles than after low cordotomy, and although it was equal in magnitude, it disappeared more quickly (Fig. 1, C). Finally, after transection of the brain stem above the medulla, the deficit of excitation for the muscle became minimal, since the preservation of the spinal cord and medulla in the bulbar frog enabled the vitally essential visceral and somatic functions to be maintained. In these experiments passive hyperpolarization developed more rarely still, and it was weaker and shorter in duration than after transection at the other levels (Fig. 1, D).

When transection of the brain or spinal cord was carried out after preliminary denervation of the gastrocnemius muscle, it did not lead to the development of passive hyperpolarization. In these control experiments the CPP of the muscle were unchanged. If, on the other hand, transection of the spinal cord or brain stem was performed after the preliminary isolation of the muscle from the general circulation, but leaving its nerve supply intact, the muscle reacted to the transection by the development of typical passive hyperpolarization.

After the potential had returned to normal and become stabilized, the sciatic nerve was divided. Denervation of the muscle after low or high cordotomy did not change the CPP, for the spinal frog was passive whatever the conditions of stimulation, and all its skeletal muscles were in a state of deficit of excitation as a result of the cordotomy (Fig. 2, A).

On the other hand, division of the sciatic nerve in the bulbar frog, capable of active tonic and locomotor reflexes, created an additional deficit of excitation for the denervated muscle, and thus favored the development of passive hyperpolarization (Fig. 2, B).