

PHYSIOLOGY

ELECTROTONIC POTENTIALS IN THE SIMPLEST REFLEX ARC IN STRYCHNINE POISONING

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(Received May 23, 1954. Presented by D. N. Nasonov, Active Member Acad. Med. Sci. USSR).

Two types of electrical potentials arise in the spinal roots in the course of reflex activity: spike potentials, representing the currents of action of the afferent and efferent impulses, and slow potentials, which, as opposed to the spike potentials, are spread along the roots electrotonically from intracordal formations and because of this die out at a few millimeters' distance from the spinal cord. Recently these electrotonic potentials have become the object of numerous investigations, since they probably are one of the manifestations of those central processes which develop in the spinal cord during reflex activity. However, the number of hypotheses as to the nature and significance of the electrotonic potentials of the spinal roots points to the fact that we still do not know which central process they express. Most probably they are a summary manifestation of various intracordal processes [3].

A study of the slow potentials in the simplest reflex arc — the arc of the stretch reflex which has two neurone connections, can be of a definite help in elucidating their nature and origin. However, even in such an arc the root electrotonic potentials, although more simple than, for example, in the flexor reflex arc, still consist of several components, since during the stimulation of the muscle nerve, other more complicated pathways, apart from the two-neurone ones, are called into play. Because of this it is expedient to study the changes in the slow potentials in such an arc under the influence of substances which show a selective action on the various elements of the spinal cord; this may aid in the elucidation of the origin of various components of the slow potentials and at the same time solve several vague problems of the action mechanism of such substances.

EXPERIMENTAL METHODS

The experiments were carried out on decerebrate cats and on cats with an intact central nervous system, under a light ether anaesthesia. The reflex reactions were elicited by stimulating the muscle nerve of the calf by single induction shocks. The vertebral canal was opened in the lumbar region, the outgoing electrodes were placed on the corresponding posterior and anterior roots (the latter were cut always distally to the outgoing electrodes to eliminate antidromal impulses). The electrical potentials were amplified by means of a symmetrical four-stage amplifier connected by a rheostatic capacity system (with a time-constant of 1 second) and registered on a double cathode ray oscillograph.

In the present report the results of experiments with the local poisoning of the dorsal surface of the 7th lumbar to the 1st sacral segments with a solution of strychnine in the dilution of 1:1000–1:5000 are presented.

In animals which had not been poisoned (and very regularly in the decerebrate), in response to a single stimulation of the muscle nerve in the corresponding anterior root, a well synchronized current of action was produced with a short latent period (about 3.5 millisecon), which doubtless represented a reflex discharge along two neurone pathways [4,7] — the so called "monosynaptic reaction". Subsequently with some increase in stimulation, a series of currents of action, more scattered in time, appeared, obviously representing the activity of the more complicated

multineuronic pathways ("polysynaptic reaction"). On placing the proximal outgoing electrode adjacent to the spinal cord, and with an adequate amplification, electrotonic potentials other than these spike potentials were registered; on these spike potentials were superimposed.

With a weak stimulation, which produced only a monosynaptic current, only a very weak and short negative electrotonic potential was registered from the anterior root, with a latent period of 3-4 millisecc, less than 50 μ V (during this time recordable electrotonic potentials were absent altogether from the posterior root). The latter did not suffer any noticeable changes during strychnine poisoning.

However, when the stimulation was increased, so that polysynaptic currents of action were produced, the character of the electrotonic potential was significantly complicated. On the anterior root there appeared a wave of electronegativity, considerable in size and duration, the latent period of which was 5-10 millisecc, the same as the latent period of the polysynaptic currents of action. If the proximal outgoing electrode was in contact with the cord surface, then, before the beginning of such a negativity a more or less pronounced wave of electropositivity was observed, (which corresponded in time with a short wave of electronegativity which developed on weak stimulation and often masked it completely). A prolonged phase of electronegativity was occasionally replaced by a phase of electropositivity; therefore all this complex of slow potentials corresponded approximately to that described by I.S. Beritashvili [1] during the stimulation of the big nerve stems, but it was much shorter. Simultaneously, with the electrotonic potentials in the anterior roots there appeared a wave of considerable electronegativity on the posterior roots, on those through which afferent impulses entered, as well as on the neighboring ones.

Such slow potentials which develop on increasing the stimulation, as opposed to the response to weak stimulation, changed abruptly soon after the beginning of the action of strychnine. On the ventral surface the primary positive phase, which took place immediately after the arrival of the most rapid afferent impulses into the spinal cord, was increased slightly. The phase which followed it increased sharply as soon as 30-60 minutes from the beginning of the action (sometimes tenfold or more), and was prolonged; its latent period became shorter. The negative electrotonic potential arising in the posterior roots immediately after the arrival of afferent impulses did not increase much under the influence of strychnine, but its later part was much more variable in poisoning (see also [5]). This part of the negative wave became more pronounced as the poisoning developed, although it did not reach the intensity of the negative phase in the anterior root. The time of appearance of such a second wave of electronegativity in the posterior root corresponded with the appearance of strong electronegativity in the anterior root.

As an example several oscillograms are shown in Figure 1 (experiment of November 28th 1953). On the first oscillogram (1) — the electrotonic potential of the 7th dorsal (upper curve) and ventral (lower curve) lumbar roots with single stimulations of the gastrocnemius nerve previous to poisoning is shown. On the upper curve, after a slight interval following stimulation (about 1.5 millisecc) a powerful spike could be seen — the current of action of the afferent impulses (a downward deviation, having gone beyond the limits of the screen). Immediately following the current of action, a prolonged negative electrotonic potential was registered (greater than 20 millisecc). During this period a small positive potential was passed from the anterior root, which after approximately 10 millisecc was followed by a long period of electronegativity, the termination of which took place beyond the limits of the oscillograph screen. The spike potentials from the ventral root were not registered from this specimen before poisoning.

The oscillograms 2-5 were taken in identical conditions of connection and stimulation, but during the development of the strychnine poisoning. On oscillogram 2, one can already see a definite additional negative wave on the upper curve, on the lower curve (anterior root) a significant increase in the negative phase is noted and also the appearance of rapid oscillations (apparently of a spike character) before a steep rise. This can be seen much clearer on oscillogram 3; finally, a complete development of changes can be observed on the following oscillogram; here it can be seen that the increase in the negative phase of the electrotonic potentials in the anterior root was several times greater than its increase in the posterior root (the lower ray has even overstepped the upper). Immediately before the beginning of the steep rise of the electrotonic potential on the anterior root (lower ray) a powerful spike potential was registered (the top of the spike went above the limits of the screen). The fact that this was a current of action was proved beyond doubt by its tapering off; after moving the proximal electrode further than 1 cm from the cord, then the electrotonic potentials could not be registered at all (oscillogram 6). Oscillogram 4 shows also that simultaneously with the beginning of the positive phase, a small spike potential, absent before poisoning, was registered from the anterior root. It represented the current of action (see oscillogram 6) and by its latent period (approximately 3.5 millisecc) corresponded to the monosynaptic current of action which manifested itself in strychnine poisoning. This current of action was completely unconnected with the appearance of a powerful negative

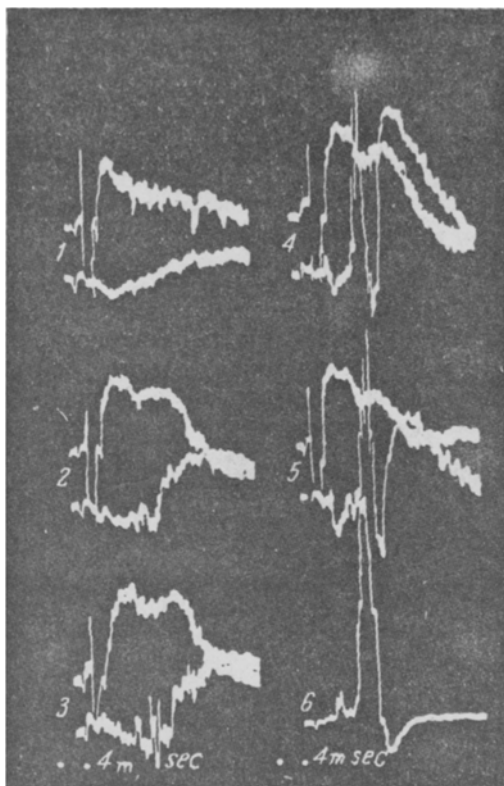


Fig. 1. The changes in electrotonic potentials of the 7th dorsal and ventral roots in strychnine poisoning. 1) Before the application of strychnine; 2-5) 20, 35, 50 and 65 minutes after its application. The upper ray is the lead from the dorsal, the lower from the ventral roots. The proximal electrode on the ventral root was in contact with the cord surface; 6) potentials of the anterior root on moving the proximal electrode 1 cm away from the cord. The upward deviation corresponds to the negativity of the proximal outgoing electrode.

under the influence of strychnine the spike potentials also increased or appeared if they had not been present before poisoning, but they did not increase uniformly. Strychnine provoked only a slight increase in monosynaptic spikes, which appeared in the anterior root much earlier than the powerful negative electrotonic potential, often already in the initial positive phase. Polysynaptic spikes increased in poisoning, often tenfold; this increase took place parallel with a powerful growth of the negative phase of the electrotonic potentials and the spike potentials arose either before its beginning, or during the period of its growth (sometimes at the very beginning and sometimes later).

Thus, strychnine acts mainly not on the processes which develop immediately after the arrival of a single wave of stimulation along the afferent pathways into the spinal cord, but mainly on the later processes. The main changes during the action of strychnine take place in the period of diminution of the negative electrotonic potential in the posterior roots and the development of a powerful electronegative wave in the anterior (developing 5-10 msec after stimulation). The earlier processes are changed to a far lesser degree, if at all. Similarly, strychnine mainly increases the later, polysynaptic, spike potentials, having a weaker action on the monosynaptic.

This was well seen in those cases when the stimulation of the muscle nerve before poisoning produced only a monosynaptic current of action, without the polysynaptic. Such a case is shown in Figure 2 (experiment from May 5th, 1953). The electrodes were placed at some distance from the cord and because of that the electrotonic potentials were not registered. Oscillogram 1 is the response in the 7th anterior root to the stimulation of the nerve of the gastrocnemius before poisoning. Only a monosynaptic spike with a latent period of approximately 3.5 msec can

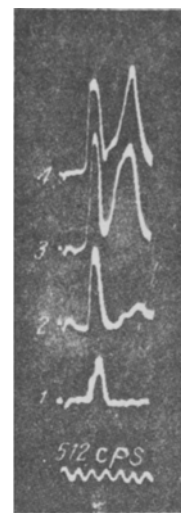


Fig. 2. Changes in the current of action of the 7th anterior root in strychnine poisoning (the proximal outgoing electrode at a distance of 1 cm from the cord). 1) Current of action before the use of strychnine; 2-4) 60, 85, and 110 minutes after its use. Oscillogram 4 was taken during decreased amplification.

electrotonic potential in the anterior root; it appeared much earlier, even in the beginning phases of the first electropositive wave, which was little changed during the action of strychnine. Oscillogram 5 was taken during a still deeper poisoning; since the rays deviated beyond the limits of the screen, the amplification was decreased — hence the reactions appear to be weaker. A true weakening of the electrotonic as well as spike potentials was observed only in the very deep stages of poisoning.

As can be seen from the oscillograms shown,

be seen. The following oscillograms were traced during an identical stimulation as the poisoning developed. Already on oscillogram 2 it can be seen that after a slightly increased monosynaptic spike a polysynaptic spike appeared, although still rather weak; but later it grew rapidly and "overtook" the monosynaptic (similar observations were also made by Sherrer [8]).

The changes in the electrotonic potentials under the action of strychnine doubtless confirm the deduction about the significant difference between the monosynaptic and the polysynaptic reactions, which occur during stimulation of the muscle nerves, action of strychnine being mainly on the intermediate neurones. The polysynaptic spike potentials which take place through the intermediate neurones, are at the same time especially sensitive to strychnine; there is a definite dependence between them and the powerful electrotonic potentials, which develop under the action of strychnine. The more intensive is the negative electrotonic potential which develops in the anterior root, the stronger are the polysynaptic spike potentials. The monosynaptic spikes are completely unconnected with the development of this electrotonic potential and arise long before its appearance. Obviously the authors were dealing with these monosynaptic currents of action, when they were describing the appearance of spike potentials without the electrotonic ones, during the stimulation of the muscle nerve [2], or the stimulation of the motor neurones without the summation of the local processes [1].

Therefore the strongest electrotonic potentials originate only with the participation of the intermediate neurones; no significant electrotonic potentials arise in the roots when the impulse passes along the straight two-neurone pathways. The intermediate neurones, the stimulation of which provokes the appearance of dorsal electrotonic potentials, obviously possess an unequal sensitivity to strychnine. Those in which the processes develop more rapidly, are less sensitive to poisoning than those in which excitation develops more slowly. The action of strychnine was most intense on the negative potential of the ventral surface, which was also noticed by other research workers [6]. It must be considered that the main source of such a potential is not the bodies of the intermediate neurones which are placed nearer to the dorsal surface of the spinal cord; it obviously develops in the area where the impulses of the intermediate neurones act on the motor cells.

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