PHYSIOLOGY

ELECTROTONIC POTENTIALS OF THE SIMPLEST REFLEX ARCS IN PHENOL POISONING

P. G. Kostyuk

From the Department of General Physiology (Chairman: D. S. Vorontsov, Corresponding Member, Acad. Sci. Ukr. SSR), Institute of Animal Physiology (Director: Candidate Biol. Sci. P. G. Bogach), Kiev University

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It was shown in our previous communication [4] that the various components of spinal root electrotonic potentials arising from a single stimulation of a motor nerve are affected differently by strychnine. These data provide convincing evidence of the complex nature of the electrotonic potentials associated with even the simplest reflex arc – the extensor reflex arc.

It was of interest to study these variations for the same reflex arc in the presence of other substances which act selectively on various elements of the spinal cord. One such substance is phenol, which enhances the excitability of the spinal cord, to the extent of causing convulsions. Baglioni [8] has shown that phenol, in contrast to strychnine, raises the excitability of anterior horn cells. A. V. Palladin [5] found that phenol, similarly to strychnine, raises the excitability of sensory, but not of motor formations of the spinal cord. Yu. M. Uflyand [6] also found enhanced reflex excitability after local application of phenol solutions to the dorsal surface of the spinal cord. However, A. A. Ukhtomsky and I. I. Kaplan [7] have shown that phenol poisoning can raise the excitability of both the motor and the internuncial neurons, the differences being of degree only.

Examination of changes in electrotonic potentials under the action of phenol may contribute to the elucidation not only of the nature of these potentials, but also of the still unresolved question of the mechanism of action of phenol on the central nervous system.

The experimental techniques used were the same as were described in our previous paper [4]; 0.1-0.2% phenol solution was applied locally to the dorsal surface of the 7th lumbar or 1st sacral segments of the spinal cord. A stimulus was applied to motor nerves of the leg, and the electrical reaction was registered from leads from the posterior root (cut distal to the leads, or intact) and the anterior root (in all cases cut below the leads).

Experiments in which the action potentials of the anterior roots were registered without complicating electrotonic potentials (proximal lead placed at a distance of more than 1 cm from the spinal cord) showed clearly that phenol strengthens both monosynaptic and polysynaptic action potentials. In contrast to the action of strychnine, no selective effect on either of these forms of reflex reaction could be observed – both were intensified equally; only occasionally was the monosynaptic action potential enhanced to a slightly greater degree. The maximum effect due to phenol was somewhat smaller than with strychnine. The phase of enhancement of potential was fairly soon followed by one of depression of both the mono- and polysynaptic action potentials, until finally all electrical reactions were abolished.

The changes in the spike potentials due to phenol poisoning are shown clearly by the oscillograms (Fig. 1), registered under constant conditions of stimulation of the motor nerve to the sartorius muscle, leading from the anterior root of the 7th lumbar segment. As the toxic effect of the phenol develops, we see enhancement of both monosynaptic (first large spike) and polysynaptic action potentials, followed by suppression of both.

Fig. 1. Changes in the action potentials of anterior roots (L_7) following local application of 0.1% phenol to the dorsal surface of the segment (proximal recording electrode placed 1 cm from the spinal cord). Oscillograms 1-5 were taken 30, 40, 75, 90, and 105 minutes, respectively, after application of phenol. Below: time scale (512 cps).

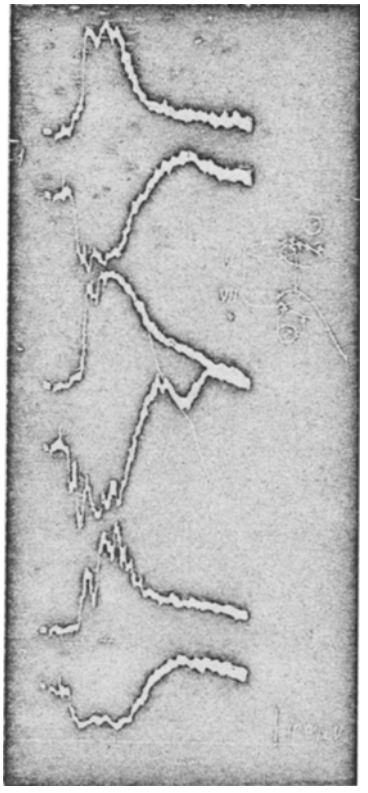

Fig. 2. Changes in electrotonic potentials of the posterior root (L_6) and anterior root (L_7), in phenol poisoning. The proximal electrode at L_7 was in contact with the surface of the spinal cord. 1) before application of phenol; 2 and 3) 30 and 60 min after. On the right: diagrammatic representation of arrangement of recording electrodes. Below: time scale (512 cps). Upward deviations correspond with negative potentials of the proximal recording electrodes. The upper tracings are from the dorsal, and the lower ones from the ventral side.

Like the spike potentials, the electrotonic potentials are to some extent enhanced by phenol, although not as powerfully as by strychnine. A negative electrotonic potential appeared in the posterior roots immediately after entry into the spinal cord of afferent impulses due to stimulation of an intensity sufficient to evoke polysynaptic reflex reaction. The amplitude of this potential was unaffected by phenol, the only perceptible effect being a slower return to the zero line, without the appearance of a secondary negative wave.

With such powerful stimulation of the nerve a prolonged electrotonic potential arose in the anterior roots, consisting of an initial positive phase (particularly well marked when the proximal recording electrode was in contact with the surface of the spinal cord), and a negative phase. The initial positive potential was unaffected by phenol, whereas the negative potential was noticeably increased; in some cases it took a sinuous course. The rise in the negative electrotonic potential was, however, never as considerable as with strychnine poisoning. After a longer or shorter time of action a general decline in the amplitude of both the anterior and posterior root electrotonic potentials set in (Fig. 2).

The first oscillogram (1) of Fig. 2 was recorded before application of phenol. Since, in this experiment, the recording electrodes were placed on the central section of the cut posterior root (L_6), and the afferent impulses from the sartorius muscle nerve entered the spinal cord through the intact roots (L_7 - S_1), the action potentials of the afferent impulses were not recorded, and, after the latent period (duration about 2.7 msec), a considerable

electrotonic potential appeared immediately, uncomplicated by spike potentials. Almost simultaneously with this, a powerful positive potential appeared at the ventral surface of the spinal cord, changing after over 10 msec.to a negative phase, the termination of which was beyond the limits of the oscillograph screen. This intense electrotonic potential was accompanied by barely perceptible spike potentials which, moreover, appeared in the initial stages of the positive phase; the negative wave was practically entirely free of spike potentials.

The second oscillogram (2) was recorded during the period of enhanced excitability due to phenol poisoning. The amplitude of the electrotonic potentials is not perceptibly augmented on the dorsal side, but a delay in return to the zero position is noticeable. The intensity of the initial positive phase of the ventral electrotonic potential is also not increased, not is its duration, but the negative phase is markedly augmented, and has a wave-like character. Apart from the changes in electrotonic potentials, this oscillogram also shows clearly the changes in spike potentials, which are greater than those seen in the preceding oscillogram (1). The first of these (monosynaptic action potential) was registered together with the descending limb of the positive potential, and subsequent ones registered before the start of the abrupt transition from the positive to the negative phase (polysynaptic action potentials). The weak potentials, also obviously spike potentials, were also recorded on a background of the first wave of electrotonic negative potential. As in the case illustrated by Fig. 1, the mono- and polysynaptic potentials are uniformly augmented.

The third oscillogram (3) was recorded after a considerable duration of action of phenol, in the period of commencing suppression of excitability of the spinal cord. We see here a weakening of all phases, both dorsal and ventral, of electrotonic potentials, with disappearance of spike potentials.

We thus see that the processes which arise soonest in the spinal cord, in association with electrotonic potentials in the posterior roots, are even less sensitive to phenol than to strychnine poisoning. Both these substances exert their action chiefly on the later processes, arising during the stage of reflex discharges of motoneurons. As opposed to strychnine, however, phenol intensifies the various types of reflex reactions in the anterior roots fairly uniformly. Preferential augmentation of either electrotonic or spike potentials, connected with the activity of two-neuron links, is not observed, nor is there any such marked increase in the negative electrotonic potentials and the efferent action potentials due to excitation of internuncial neurons, as is found with strychnine poisoning. It is evident that phenol differs from strychnine in not acting selectively on complex multineuronal links.

It might be thought that the nature of the action of phenol on the extensor reflex arc depends either on its action on motoneurons (raising their excitability with regard to impulses arriving by direct paths as well as from internuncial neurons, as was supposed by Baglioni), or else on its uniform action on presynaptic formations (arborizations of afferent fibers and axons of internuncial neurons and motoneurons).

The second assumption seems the more probable, if we take into consideration the fairly high speed of appearance of changes in reaction after local application of phenol to the dorsal surface of the spinal cord (after 15-20 minutes). This time seems insufficient for diffusion of adequate amounts of phenol to the anterior horn cells. Additional evidence in favor of the second assumption is afforded by our previous findings (3), concerning changes in inhibitory processes in a two-neuron arc in phenol poisoning. Whereas in strychnine poisoning the threshold for inhibition of the monosynaptic reaction to a single stimulation of the nerve to an antagonistic muscle is raised, this threshold is lowered in the case of phenol poisoning. This inhibitory effect is effected through a direct path, by the direct action of impulses at the terminations of afferent fibers on motoneurons. If only the excitability of motoneurons were to be raised in phenol poisoning, this should obviously result in hindrance, rather than in facilitation of inhibition; increase in the activity of "presynaptic" formations should have an inhibitory effect.

The above data allow one to draw the conclusion that the electrotonic potentials associated with the simplest reflex arc are of a complex nature, and are made up of the activities of various elements of the spinal cord. The sources of the intense negative potentials of the anterior and posterior roots, which appear only when internuncial neurons are drawn into the reaction, are seen to be diverse, as has been shown for other reflex arcs by D. S. Vorontsov [2] and I. S. Beritashvili [1]. The origins of the initial positive potential on the ventral side (particularly well marked when the recording electrodes are in direct contact with the spinal cord), and of the subsequent slow potential, are also diverse. The initial positive potential is produced by the same processes as is the negative dorsal potential; inversion of its sign in leading off from the ventral surface of the spinal cord is an effect peculiar to the recording of potentials through a capacious conductor – the spinal cord. For this reason, the potential is well defined only when the proximal recording electrode is in direct contact with the surface of the spinal cord; it does not express electrotonic changes in motor elements.

In view of this, the attempts of some workers to regard electrotonic potentials as direct expressions solely of postsynaptic processes, responsible for the initiation of impulses in motoneurons [9,10], must undoubtedly be considered as an oversimplification of the relations actually prevailing in the spinal cord.

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