of urine within 30 min. before and after muscular effort, its determination being carried out by the method of Sjoerdsma, Weissbach and Udenfriend[3].

Altogether 50 men, aged 19–50, were examined after muscular effort lasting from 5 min. to 3 hr. Twelve men ran an average of 1,250 m. for 5 min. at maximum effort on a laboratory treadmill. Eight men were examined after a 3-km. cross-country race and 21 men after a marathon race. Also included in the group examined were 9 football players after a football match.

In no case was sulphosalicylic acid proteinuria found before the effort. With the exception of 4 well-trained cross-country runners and 5 marathon runners, proteinuria could always be detected after the effort was made (ranging from slight turbidness to heavy protein precipitation). After a marathon race, proteinuria of 6–166 mgm./100 ml. (average value 47·1 mgm./100 ml.) was detected by means of Exton's method.

In no case, either before or after the effort, could any 5-hydroxyindoleacetic acid be detected. From this it can be concluded that, even when accompanied by considerable nervous excitement, muscular effort does not result in increased urinary excretion of this substance.

L. Schmid

Institute of Sport-Medicine,
Salmovská 5,
Prague 2.

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Anionic Permeability of the Inhibitory Postsynaptic Membrane of Motoneurones

It has been postulated that the inhibitory postsynaptic potential in motoneurones is caused by the movement of ions across patches of the postsynaptic membrane that are momentarily (1–2 msec.) made very permeable for ions less than a certain critical size[1]. A sufficiently increased intracellular concentration of anions capable of passing through the postsynaptic membrane would change the normally hyperpolarizing inhibitory postsynaptic potential into a depolarizing one. In accordance with the hypothesis it was shown that injections of the small ions, Br^-, Cl^-, NO_3^-, and SCN^-, but not injections of the large HCO_3^-, $CH_3CO_2^-$, SO_4^{--}, $H_2PO_4^-$ or HPO_4^{--}, were effective in transforming the inhibitory postsynaptic potential into a depolarizing response. The diameter of the largest effective anion (derived from limiting ion conductances[2] and expressed relative to $K^+ = 1{\cdot}00$) was 1·11 and that of the smallest not effective, 1·65 (ref. 1). A weakness in the experimental evidence was the relatively few ions tested and the gap in size between the effective, and not effective, ions. Furthermore,

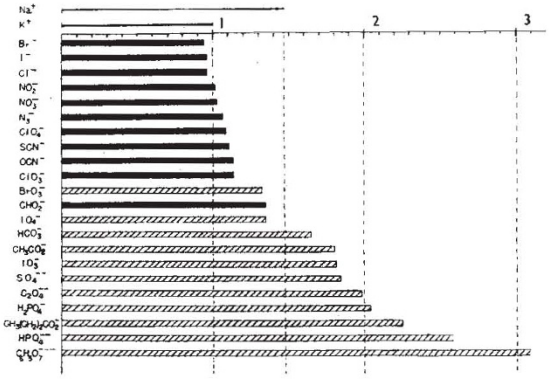

Fig. 1. The ions specified in the left column are arranged in order of hydrated ion size (derived from limiting ion conductances (ref. 2) and expressed relative to $K^+ = 1{\cdot}00$). The black bands indicate ions that are effective in changing the inhibitory postsynaptic potential into a depolarizing response; the hatched, those which are not effective. It should be noted that these results are consistent with the hypothesis that potassium ions can pass through the membrane but not sodium ions (ref. 1)

it was not possible to exclude a slight effect of the bicarbonate ion (diameter 1·65) (ref. 1).

We have now extended the previous investigation and tested 22 different species of anions. Microelectrodes containing the anion to be tested were inserted into motoneurones and used both for injection and recording. Most tests were made on knee flexor motoneurones that were inhibited by quadriceps group Ia afferents. The anions were injected by a steady current of 3–10 $\times$ 10^{-8} amp. during 1 min. or more. The effect of the injection was judged by the polarity and size of the inhibitory postsynaptic potential immediately following the injection. The effects of the injections were always reversible within a few minutes after the injection. This recovery is attributable to the outward diffusion of the injected ions across the cell membrane.

The results are illustrated by the histogram (Fig. 1) showing the ions arranged according to their diameters. It appears that all ions with a diameter less than 1·32 (bromate) were effective in producing the reversible change of the inhibitory postsynaptic potential into a depolarizing response. Larger ions were ineffective except for the only slightly larger formate-ion (1·35). Injections of bicarbonate ions or carbonate ions never resulted in an immediate change of the inhibitory postsynaptic potential. After several injections of any of these ions there was often a gradual decrease of the hyperpolarization and finally a change to a depolarizing inhibitory postsynaptic potential. This late cumulative change is presumably due to secondary effects of these ions and cannot be taken as evidence for a similar action as that exerted by the small ions. Hence our findings support the hypothesis that the activated inhibitory postsynaptic membrane has an increased permeability for all anions less than a certain critical size.

T. Araki
M. Ito
O. Oscarsson

Department of Physiology,
John Curtin School of Medical Research,
The Australian National University,
Canberra.

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