TRANSMEMBRANE IONIC CURRENTS IN SMOOTH MUSCLE OF THE PULMONARY ARTERY

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KEY WORDS: pulmonary artery; smooth-muscle cells; transmembrane currents; tetraethylammonium.

The smooth-muscle cells of the pulmonary artery under normal conditions do not generate action potentials and their membrane possesses marked rectifying properties [3, 4, 6]. In the presence of tetraethylammonium (TEA) these cells acquire the ability to generate action potentials, both spontaneously and in response to a depolarizing current [3, 6]. It has been suggested that the action of TEA is connected with inhibition of the potential-dependent potassium holding current, early activation of which in a normal solution prevents the development of action potentials [2, 3]. To test this hypothesis, we studied transmembrane ionic currents of smooth-muscle cells of the rabbit pulmonary artery under voltage clamp conditions.

EXPERIMENTAL METHOD

Transmembrane ionic currents were studied under voltage clamp conditions by the double sucrose gap method [1]. Circular muscle strips of the rabbit pulmonary artery, 300-400 μ wide, were used in the experiments. The width of the test portion of the muscle was 500 μ. The compoisition of the Krebs' solution used in the experiments was given previously [4].

The records of currents and graphs illustrating this paper were corrected for leakage current. It was assumed that leakage conductance is independent of membrane potential and time.

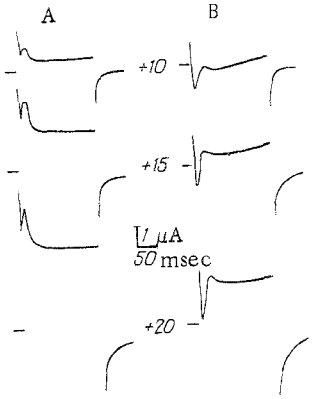

Fig. 1. Traces of transmembrane currents and smooth-muscle cells of rabbit pulmonary artery recorded in normal Krebs' solution (A) and in the presence of 10 mM TEA (B), with clamped depolarizing shifts of membrane potential by 10, 15, and 20 mV.

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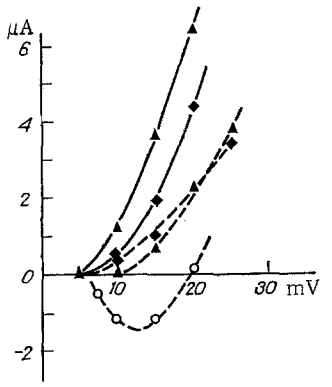

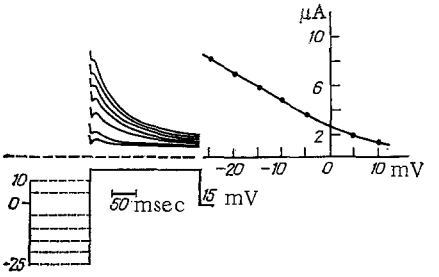

Fig. 2

Fig. 3

Fig. 2. Current-voltage characteristic curves of membrane of smooth-muscle cells of pulmonary artery in normal solution (continuous lines) and in presence of 10 mM TEA (broken lines) for fast outward current (triangles), steady outward current (squares), and inward current (circles).

Fig. 3. Action of conditioning hyper- and depolarization on transmembrane currents in normal solution. Records of currents and graphs shown on the same scale.

EXPERIMENTAL RESULTS

When shifts of membrane potential from the resting potential level were clamped, a fast spike of capacitive current was followed by the development of an outward current, which reached a maximum after 5-15 sec and then began to decrease approximately in accordance with exponential rule, until a steady level was reached (Fig. 1A). This complex pattern of transmembrane current is evidently due to activation of at least two ionic conductances: one responsible for the fast outward current, the other for the steady current. The amplitude of both fast and steady outward currents increased with an increase in the clamped shifts of membrane potential (Fig. 1A, Fig. 2). Under these conditions no signs of an inward current could be found. On the addition of 10 mM TEA, which blocks potassium conductance, to the Krebs' solution the pattern of the transmembrane currents was changed. The amplitude of the fast outward current was considerably reduced, and under these conditions a fast inward current, preceding the outward current, could be observed (Fig. 1B). The results of these experiments are summarized in Fig. 2, which shows the current-voltage characteristic curves for the fast inward, outward, and also the steady outward currents in normal Krebs' solution and under the influence of TEA. The current-voltage characteristic curve for the fast inward current had a region with negative resistance, and reversal of the current took place at a potential of about 20 mV. The channels of conduction for the fast inward current were completely activated by depolarizing shifts of membrane potential by 12-15 mV. The fast outward current was not suppressed completely by TEA, but more so than the steady current. The threshold for the fast outward current was raised by 5-7 mV.

The amplitude of the fast outward current arising in response to depolarizing shifts of membrane potential was dependent on the initial membrane potential level. An example of this dependence is illustrated in Fig. 3. The outward current was evoked by abrupt depolarization of the membrane by 15 mV, which was preceded by conditioning hyper- or depolarization to a varied degree for 120 msec. It will be clear from Fig. 3 that conditioning depolarization reduced the amplitude of the fast outward current, whereas hyperpolarization increased it as a near-linear function within the range 5-25 mV. Meanwhile, the steady outward current was almost unchanged by the action of conditioning polarization.

The results of these experiments indicate that the main cause of electrical inexcitability of the smooth-muscle cells of the pulmonary artery is early activation of the fast outward current. The experiments with TEA showed that activation of the fast outward current took place almost simultaneously with activation of the fast inward current, as is observed in other types of smooth muscles also (ureter [5], uterus [7]). However, a distinguishing feature of the smooth-muscle cells of the pulmonary artery is that under normal

conditions the conductance of the activated channels for the fast outward current is much greater than the conductance of the inward channels. For that reason, despite activation of the channels of the fast inward current, the resultant current is outward in direction during depolarization of the membrane. In the presence of TEA, when the channels of the fast outward current were partially blocked, a component of inward current appeared in the resultant current, the current-voltage characteristic curve of which included a region with negative resistance. Under these conditions the muscle cells acquired the ability to generate gradual action potentials [3, 6].

The dependence of the fast outward current on the membrane potential level in the smooth-muscle cells of the pulmonary artery was similar in character to that in other types of smooth muscles (ureter [5], uterus [7]), and it indicates that under normal conditions the overwhelming majority of channels of the fast outward current are in an inactivated state. During hyperpolarization, inactivation of the channels is abolished and the fast outward current increased. Depolarization, on the other hand, leads to even greater activation of these channels. This mechanism evidently plays a part in the regulation of excitability of muscle cells during a change in membrane potential under the influence of physiologically active substances.

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HIGH ERYTHROCYTE CONCENTRATION IN BLOOD CIRCULATING IN THE BRAIN

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An uneven distribution of erythrocytes in blood flowing through the vascular system was noted a long time ago. For instance, it is now 100 years since Kostyurin [1] found that the erythrocyte concentration in human blood flowing from the region of the clavicle is higher than in blood from the little toe. It was shown later [8] that blood circulating in the region of the renal tubules contains more erythrocytes than blood circulating in the renal cortex. On the basis of such investigations of phenomena of the microcirculation the following principle of redistribution of erythrocytes in the vascular system of organisms was formulated: Blood with a relatively higher concentration of erythrocytes is found in microvessels in parts of the body where the local circulation is intensified, and vice versa [2-4].

It might be supposed that blood in the brain, where the circulation is particularly intensive [6], would contain more erythrocytes than in peripheral parts of the circulatory system. The experiments described below were carried out to test this hypothesis.

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