

## Electrical Activities of the Anterior Mesenteric Vein of the Guinea Pig and Effects of Potassium Ion on Them

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(Received. December 1, 1966)

Spontaneous electrical activities of mammalian vascular muscles have been reported to be similar to those of visceral smooth muscles although they may differ in some respects (1,2,3,4). In general, the present experiments confirm the previous reports except that the larger action potentials (50mV or more) sometimes reaching near 0-level could be recorded. In addition the membrane potential could be shown to be determined primarily by potassium concentration gradient as in visceral smooth muscles.

Isolated longitudinal strips of the anterior mesenteric vein from guinea pig were used, 4-6mm length and 1-1.5mm width, mounted in an organ bath of 5mls. The adipose tissues and the adventitia were carefully removed under binocular microscope. Intracellular recordings were made from flexibly mounted glass microelectrodes with high tip resistance 30-50M $\Omega$  inserted into the longitudinal muscle layer from the outside of the vein. The preparations were examined histologically following the experiments to ascertain that the adventitia had been properly removed and that the muscle layer was not injured.

The normal Krebs's solution used in the experiments contained (mM): Na 137.4, K 5.9, Mg 1.2, Ca 2.5, Cl 134, H<sub>2</sub>PO<sub>4</sub> 1.2, HCO<sub>3</sub> 15.5, glucose 11.5 and was aerated with 95% O<sub>2</sub> + 5% CO<sub>2</sub>. The solution flowed continuously at the rate of 8-10ml/min., and at a constant temperature of 37°C.

By the time when the adventitia had been removed the preparation exhibited spontaneous contractile activity.

The membrane potentials varied from about 45mV to 56mV and the action potentials from 35mV to 55mV, sometimes reaching near 0-level. Overshoot was seldom observed.

Spike discharge occurred singly, in pairs or in bursts (Fig. 1). The train discharge appears to be initiated by a propagated impulse and maintains some level of depolarization upon which fast spikes and slow waves are superimposed (Fig. 1a). Single spikes associated with slow potentials can be seen irregularly firing at any phase of the slow wave (Fig. 1b). Slow waves that do not initiate fast spikes can also be seen sometimes with an amplitude of up to 20mV.

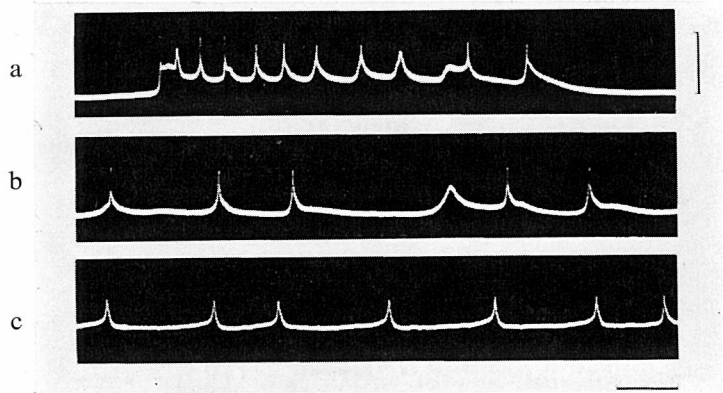


Fig. 1. The spontaneous discharge patterns. a: A typical record of the train discharge. b: Repetitively firing single spikes. c: The pacemaker potentials. Calibration, 50mV and 1sec.

Fig. 1c shows typical pacemaker potentials which depolarize the membrane gradually following the previous spike discharge to initiate an action potential at a critical level.

Thus the spontaneous spike discharge pattern of the anterior mesenteric vein was quite similar to those of visceral smooth muscles, i. e. the mammalian uterine smooth muscle or the taenia coli of the guinea pig, except that pacemaker potentials could be more often recorded than in visceral muscles and that overshoot of the spike potential occurred seldom. In previous reports, the maximal spike height observed with the sucrose gap method was 5mV or less (2,6). This fact may be due to poor intracellular conduction and asynchronous activity. Multiple pacemaker sites would seem necessary in order to maintain some degree of contraction in these poorly conducting tissue.

The mode of change of the membrane potential was observed in various potassium concentrations (0mM, 5.9mM, 12mM, 29mM, 59mM, 118mM). Both KCl and  $K_2SO_4$  were used.  $Ca^{++}$  content was doubled to keep adequately free Ca ions in the sulphate solution.

In Fig. 2 the external potassium concentration was increased to ten times normal (59mM) by KCl. The membrane potential began to decrease immediately, accompanied by an increase in spike frequency. Subsequently the spike discharges changed to the oscillatory ones and in a short time ceased.

A potassium free solution initially also caused a decrease in the membrane potential and an increase in spike frequency in a similar manner as did the potassium excess one. This phenomenon lasted several minutes, but subsequently the membrane potential gradually increased and after about 30 minutes spike discharge ceased (Fig. 3).

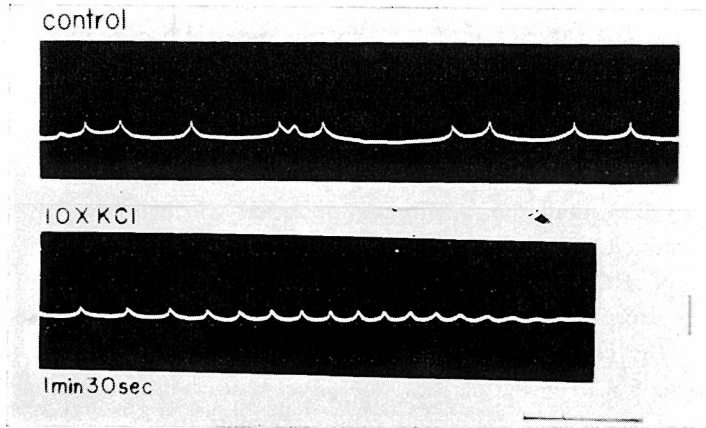


Fig. 2. The effect of potassium excess solution (59mM). Calibration, 50mV and 1sec.

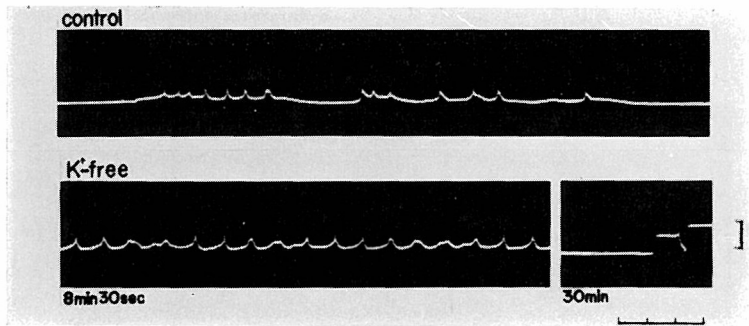


Fig. 3. The effect of potassium free solution. Calibration, 50mV and 1sec.

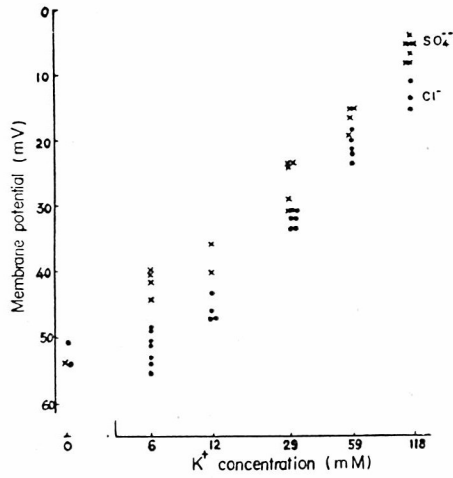


Fig. 4. The relation between the membrane potential and the logarithm of the external potassium concentration in the presence of Cl ion and SO<sub>4</sub> ion.

The relation between the membrane potential which was measured in the steady state and the logarithm of the external potassium concentration is shown in Fig. 4. The relation for the higher potassium concentration from 12mM to 118mM is nearly linear. Below 12mM, is not linear. The maximum slope of the linear part is about 35mV per tenfold change in potassium concentration. This value is nearly the same as that of the taenia coli of the guinea pig (7). This result suggests that the membrane potential of this vascular muscle may be also determined primarily by  $K^+$  concentration gradient. When  $K_2SO_4$  was used instead of KCl the membrane potential decreased more at all potassium concentrations except 0mM, indicating that  $SO_4$  ion is less permeable than Cl ion as is also the case in other mammalian visceral muscle (7).

Tetrodotoxin,  $5 \times 10^{-5}$  g/ml, did not affect either the slow component nor the fast one of the action potential in this vascular tissue.

We are indebted to Dr. A. Nakajima for his help and advice during this work, and we wish to express our gratitude to Dr. K. Ito who kindly prepared histological sections of the preparations employed in this work.

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