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Effect of Epinephrine on Potassium Balance in the Perfused Hind Limbs of the Frog.

J. CLIFFORD STICKNEY. (Introduced by Ancel Keys.)

From the Laboratory of Physiological Hygiene, University of Minnesota Medical School, Minneapolis, Minn.

Intravenous injection of epinephrine produces an immediate transitory rise in the level of plasma potassium.[1, 2, 3] The duration and magnitude of this effect differs in different animal species and a subsequent fall in [K]s to below normal is equally marked and less transitory, especially in man.[4] The initial rise in [K]s seems to originate in the liver[2] but it appeared possible that the skeletal muscle might be involved in the slower and more sustained decline.

Perfusion preparations of the isolated hind limbs of the double-pithed frog were made. The perfusion fluid was a 3% gum acacia solution with the salt content and pH adjusted to correspond with normal frog Ringer's solution except for K which was somewhat high (5.18 to 6.03 m.eq./l). A perfusion pump supplied pulsating pressure to the inflow cannula entering the terminal aorta. The outflow was collected from cannulae in the renal portal veins, all other egress being prevented by ligatures. [K] was determined[5] in arterial and venous samples collected at intervals during a period of 2½ to 3½ hours. Rates of flow were measured throughout.

In preliminary experiments single injection of epinephrine into the arterial inflow gave somewhat inconstant but essentially negative results with regard to the [K] in the venous outflow. In all cases the [K] in the arterial inflow was the most important factor in determining the direction and rate of K exchange between the tissue and the perfusion fluid. When epinephrine was continuously infused into the arterial inflow (0.005 mg/cc) there was consistently a marked effect on the arteriovenous K difference, but almost no effect on the rate of K exchange between the tissue and the perfusing fluid. In other words, in these experiments, the movement of K from vascular bed to tissue proceeded at a rate independent of the total rate

1 D'Silva, J. L., *J. Physiol.*, 1934, **82**, 393.

2 Marenzi, A. D., and Gerschman, R., *Rev. Soc. argent. de biol.*, 1936, **12**, 424.

3 Brewer, G., Larson, P. S., and Schroeder, A. R., *Am. J. Physiol.*, 1939, **126**, 708.

4 Keys, Ancel, *Am. J. Physiol.*, 1938, **121**, 325.

5 Hartzler, E. R., *J. Biol. Chem.*, 1937, **122**, 19.

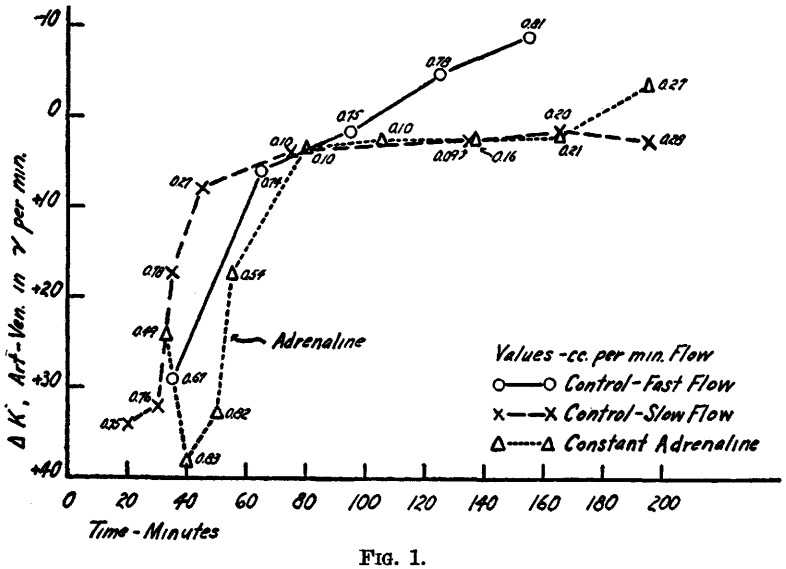

FIG. 1.

of flow. This was true whether the rate of flow was altered by epinephrine administration or by simple change in the perfusing pressure which was studied in separate experiments. These points are shown in Fig. 1 in which typical results are plotted.

These results show that when a small K gradient from blood vessel to tissue is applied in resting muscle, the rate of renewal of the blood phase is not, within physiological limits, a limiting factor for the K exchange, nor could a direct effect of epinephrine be seen. Further experiments with no net movement of K in control periods likewise failed to demonstrate a direct effect of epinephrine on K exchange or balance in resting muscle.