THE EFFECT OF POTASSIUM ON THE CORONARY CIRCULATION

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A normal potassium ion concentration is of great importance for cardiac activity and blood vessel tonus. Increase in the blood potassium ion, according to the majority of investigators, leads to the constriction of peripheral vessels [2, 10, 19], however certain authors describe its action as vascular dilatation [7].

Recently a connection has been established between pathological EKG changes during myocardial infarction and migration of potassium ions into the heart muscle—their exodus from the zone of necrosis, accumulation in the border zone, and increased concentration in the blood [4, 8, 13]. On the other hand, when potassium chloride is given intravenously [22], its introduction into the coronary artery [12, 18], pericardium, or epicardium [20] temporarily causes EKG changes similar to those observed during myocardial infarction. When a necrotic piece of muscle tissue is apposed to a frog heart a monophasic electrogram appears [5], which phenomenon (as shown on rabbit heart) is connected with the egress of potassium ion from the damaged tissue into the undamaged myocardium [4].

In these papers, simultaneous registration of EKG and changes in the coronary blood flow during the action of potassium chloride was not done and it has not been established if there is an essential correspondence between these indices. There is a limited number of studies on the question of the effect of potassium on the tone of the coronary vessels, carried out on isolated, fibrillating hearts. Some authors have noted a dilatation of coronary vessels in response to the injection of small doses and constriction of biphasic reaction when large doses of potassium chloride are given [14-16]; others find only coronary dilatation independent of dosage [9, 11, 17]. The effect of an increased potassium concentration in the entire circulatory bed on the tone of the coronary vessels has not been studied, or the importance of applying different concentrations of KCL to the epicardium.

The investigations we carried out studied the changes in coronary circulation in the intact organism after injection of KCl into the systemic circulation, the coronary vessels and after applying KCl to the epicardium.

METHODS

The experiments were performed on 55 dogs of weight 14-20 kg under morphine-urethane anesthesia. Circulatory depression of the heart was studied by the method of thermoelectric registration of the volume velocity of the blood flow in the circumflex branch of the left coronary artery [1] and by the method of resistography, i.e., by registering changes in pressure in the coronary vessels under a constant volume of blood entering them [6]. In the latter case, the blood was taken from the carotid artery and returned via a perfusing pump into the circumflex branch of the left coronary artery through a cannula inserted into it. Intra-coronary injection of KCl solutions were done through the perfusion pump system through a triple valve. The application of a tampon soaked in KCl solution was made to the anterior surface of the heart and in some of the experiments also to the region supplied by the arteries (the tampon had an area of three cm² and was placed under the pericardium). The length of the application in experiments with perfusion was 1-2 min, and in experiments with thermoelectric method, 5-10 minutes. Circulatory supply of other organs was recorded with vascular thermoelectrodes or needles of the Gibbs type. Almost all experiments were carried out with artificial respiration. In many experiments the EKG was recorded on the standard leads.
RESULTS

After intravenous administration of various concentrations of KCl (one, 1.5, two, three and 5% dose 250-100 mg) an increase in coronary blood flow was invariably observed. When the KCl concentration was significantly increased, cardiac standstill occurred. In a portion of the experiments considerable arrhythmia developed prior to cardiac standstill, and during the arrhythmia coronary flow decreased (Figure 1, a). When isotonic KCl (1.15%) was injected very slowly the changes were less marked, but were not altered in character, i.e., a slight increase in blood flow was observed which fell during arrhythmia. It follows from experiments using constant volume perfusion of the coronary arteries that the increase in coronary blood flow is related not only to the increase in total arterial pressure but also to the decreased coronary vessel tone (Figure 1, b).

Injection of KCl directly into the coronary artery produced a fall in coronary vessel tone in all concentrations used in doses of 3.4-17 mg. When 0.5 ml of 5% solution (25 mg KCl) was injected, cardiac arrest always resulted (Figure 1, c). Only in one out of 10 experiments when KCl was injected in large concentration was a biphasic reaction observed. The EKG changes after intra-coronary injection were similar to those observed in myocardial infarction; they disappeared after injection was stopped (Figure 1, d).