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The action of adrenalin, pituitrin and acetyl-cholin on the coronary arteries of the rabbit.

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The present investigation is a preliminary study of the action of the sympathetic and vagus nerves on the coronary arteries of the rabbit. The effect of adrenalin and acetyl-cholin on the coronary flow of the perfused heart of the rabbit was studied because of the action that these drugs are said to have on the extrinsic cardiac nerves. Pituitrin was employed for the purpose of comparing smooth muscle stimulation with that of the sympathetic nerve action of adrenalin.

Adrenalin was introduced into the perfusate by injecting it with a hypodermic syringe directly into the tubing at its attachment leading into the aorta. The concentration of adrenalin used was 1-10,000,000, 0.2 cc. of this being injected at a rate which would produce a dilution of approximately 1-200,000,000 in the coronary arteries. The above low concentration was employed to avoid the complicating effects produced by the extreme cardiac stimulation resulting from the higher concentrations that are ordinarily introduced. Adrenalin in this dilution produced a definite decrease in the rate of coronary flow. The decrease in the rate of perfusion varied from 12 to 22.5 per cent. The diminished perfusion rate began immediately after the introduction of the drug, and persisted up to the point of maximum cardiac stimulation. At this point the rate of coronary flow was slightly augmented for a short time. Even with these low concentrations of adrenalin there was a very distinct stimulating effect on the heart, as indicated by the acceleration in heart rate and increase in amplitude of contraction.

Pituitrin, in concentrations of 1-50,000 regularly produced a striking decrease in the rate of perfusion. This change in the perfusion rate persisted for some time after the drug was discontinued. In those experiments in which the cardiac rate was not controlled, the injection of pituitrin was followed by a reduction in heart rate of about 20 beats to the minute. In some instances there was associated with the decrease in heart rate a slight diminution in amplitude of cardiac contractions. In those experiments in which the heart was driven by rhythmically induced break shocks, the decrease in coronary flow was not as striking, and the effect did not persist as long as in the heart with normal mechanism.

Acetyl-cholin was introduced into the coronary arteries in dilutions varying from 100,000 to 200,000 concentrations. The drug in these concentrations greatly reduced the heart rate, and in some instances produced cardiac standstill. Coincident with the reduction in the rate, the amplitude of the cardiac contraction was diminished. With the onset of the above changes in the heart, the perfusion rate was greatly augmented, in some instances amounting to 180 per cent. The slowing of the cardiac rate and the depression in amplitude of contraction were transient. The increase in the rate of perfusion, however, persisted for some time after the above effects on the heart had subsided. The administration of atropin sulphate in a dilution of 1-20,000 eliminated the effects of subsequent doses of acetyl-cholin on heart rate, and in a large measure the reduction in the amplitude of contraction. The atropin, furthermore, eliminated the action of acetyl-cholin on the perfusion rate except for very transient slight increase during the period in which there was some depression in amplitude of contraction. In the earlier experiments in which higher concentrations of acetyl-cholin were employed, the depression action of the drug on the amplitude of contraction was more striking and prolonged. In those instances the depression action on amplitude was not particularly altered by atropin, and the acceleration in the perfusion rate, even though greatly reduced, was still evident.

In those experiments in which the cardiac rate was controlled by rhythmical stimulation, thus eliminating, in a large measure, the depression in the amplitude of contraction, the same action of acetyl-cholin was observed.