Attempts to study the hemodynamics of the pulmonary circulation in patients who have suffered myocardial infarction [4] are fraught with great dangers. Data in the literature concerning the pulmonary blood circulation during experimental myocardial infarction are conflicting and do not yield clear representations of the hemodynamic interactions between the greater and lesser circuits [1, 5, 6].

In this connection we have carried out a study of the hemodynamics during the acute period of experimental disruption of the coronary circulation in dogs with intact thoracic cages.

METHODS

One hundred and twenty experiments were performed on 56 dogs under morphine-chloralose anesthesia (2.5 mg/kg of morphine, 50-100 mg/kg of chloralose). Experimental myocardial infarction was produced by artificial thrombosis of the circumflex branch of the left coronary artery using catheterization of the left coronary artery. To obliterate the lumen of the vessel we used artificial thrombi of round shape and external diameter of 1.5-3.5 mm. The pressure in the chambers of the heart and great vessels was recorded via catheters which were introduced into the left ventricle, left auricle and left coronary artery via the left carotid artery and into the right auricle, right ventricle pulmonary artery and superior vena cava via the right external jugular vein. A catheter was introduced into the inferior vena cava via the right femoral vein. The arterial pressure was monitored in the right or left femoral arteries. The position of the catheters was determined by the pressure level and nature of the pulse waves. At the end of the experiment the position of the catheters was verified at autopsy. The arterial blood oxygen saturation was recorded with a flow oximeter placed in the carotid or femoral artery. Recording was effected with an oxihemograph electrolystograph 0-36 [3]. Hemodynamic indices were recorded by water-mercury [2], membrane and electronic manometers on an electrolystograph or on a six-channel ink-writing polygraph, make "Al'var," synchronously with the curves of intratracheal pressure variation, thoracic cage excursion and arterial-blood oxygen saturation.

RESULTS

The introduction of a "thrombus" into the coronary artery produced different changes in hemodynamics in different experiments, in degree and in kind. Figure 1 represents one of the most typical reactions of the circulation and respiration to the occlusion of the coronary arterial lumen. The cessation of blood flow in the coronary artery is accompanied by a sharp decrease in arterial blood pressure. The mean pressure in the left auricle, venous pressure, and diastolic pressure in the right ventricle are increased, whereas the mean pulmonary artery pressure is unchanged.

In 99 experiments the arterial pressure gradually fell during the first minute after occlusion of the coronary artery by 20-30 mm Hg and more, which was accompanied by a significant speeding up of the cardiac rate. In 11 experiments the arterial pressure rose by 10-20 mm Hg during 5-10 sec and remained at this level for 1-2 min, then fell gradually. In 10 experiments no change was noted.
Fig. 1. Change in hemodynamics when the lumen is occluded (first mark) and when blood flow is restored (second mark) in the circumflex branch of the left coronary artery. Significance of curves (top to bottom): respiratory variation in pressure in the trachea, left ventricular pressure (in mm Hg), pulmonary artery pressure (in mm Hg), right ventricular pressure (in mm Hg), mean left atrial pressure (in mm H2O), inferior vena cava pressure (in mm H2O), femoral artery pressure (in mm Hg), femoral artery pressure (membrane manometer), stimulation marker, time marker (5 sec).

Changes in the mean and absolute pulmonary artery pressure after interruption of coronary blood circulation was neither in degree nor direction determined by changes in arterial pressure. In 60 out of 91 experiments the systolic pressure in the pulmonary artery fell by 3-5 mm Hg; in 20 experiments it rose by 2-4 mm and in 11 experiments it was unchanged, while the diastolic pressure in the majority of experiments increased by 1-3 mm Hg. (72% of all experiments.) However, even with the decrease in arterial pressure by 30-40% below the initial level, the mean pulmonary artery pressure was not significantly altered. In 15 out of 65 experiments the mean pulmonary artery pressure fell by 5-15 mm H2O, in 43 experiments it rose by 10-20 mm H2O and in 7 experiments it remained unchanged. Only in 5 experiments did the mean pulmonary artery pressure rise by 30-40 mm H2O. The decrease in mean pulmonary artery pressure was usually observed in experiments in which the arterial pressure was reduced more than 30-40% below the initial level.

The arterial blood oxygen saturation in all experiments performed with preservation of natural conditions of circulation in the thorax did not change after occlusion of the coronary arterial lumen. The decrease in this parameter observed in the majority of experiments did not exceed 2-5%.

In 110 experiments, during the first 2 min after occlusion of the coronary artery a marked depression of left ventricular function occurred which corresponded to the decrease in systolic and increase in diastolic left ventricular pressures and to the decrease in arterial blood pressure. As a result of left ventricular failure the mean and diastolic pressures in the left atrium were increased in all experiments.

In certain experiments the mean and absolute left atrial pressures increased twofold over the initial level and reached 7-10 mm of Hg.

However, the degree of loss in contractility of the left ventricle, being one of the main causes of the total complex changes in circulation, did not appear to be the factor which completely determined the hemodynamic change in the lesser circulation.