PULMONARY CIRCULATION DURING THE ACUTE PERIOD OF EXPERIMENTAL MYOCARDIAL INFARCTION IN DOGS WITH INTACT THORACIC CAGE

(UDC 616.127-005.8-036.11-07:616.24-005-092.9)

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(Presented by Academician V. V. Parin)
Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 60, No. 8, pp. 21-24, August, 1965
Original article submitted September 24, 1963

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 catherization 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 cavity 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 electrokymograph 0-36 [3]. Hemodynamic indices were recorded by water-mercury [2], membrane and electronic manometers on an electrokymograph 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 significiant 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.

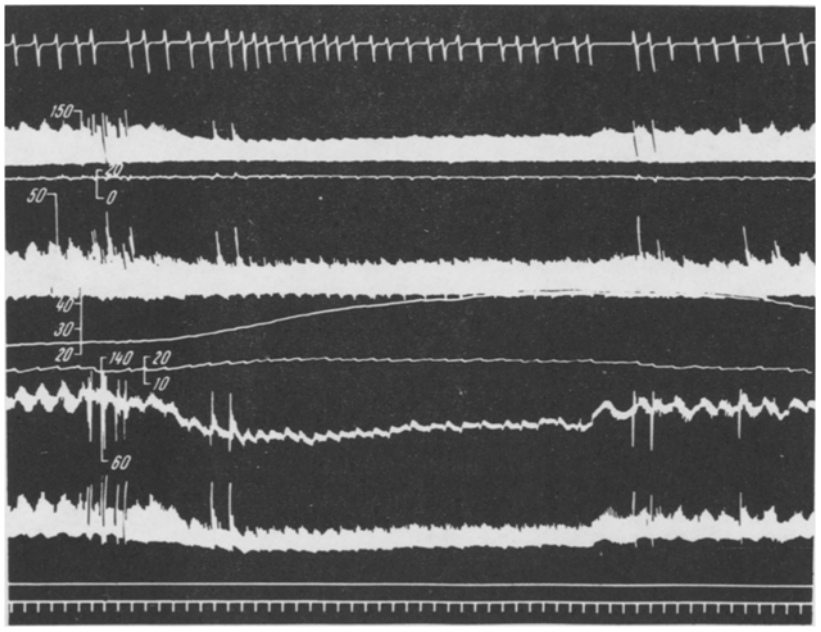

Fig. 1. Change in hemodynamics when the lumen is occluded (first mark) and when blood flow is restored (second mark) in the circumflex branch of the left coronary artery. Significance of curves (top to bottom): respiratory variation in pressure in the trachea, left ventricular pressure (in mm Hg), pulmonary artery pressure (in mm Hg), right ventricular pressure (in mm Hg), mean left atrial pressure (in mm H_2O), inferior vena cava pressure (in mm H_2O), 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 H_2O, in 43 experiments it rose by 10-20 mm H_2O and in 7 experiments it remained unchanged. Only in 5 experiments did the mean pulmonary artery pressure rise by 30-40 mm H_2O. 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.

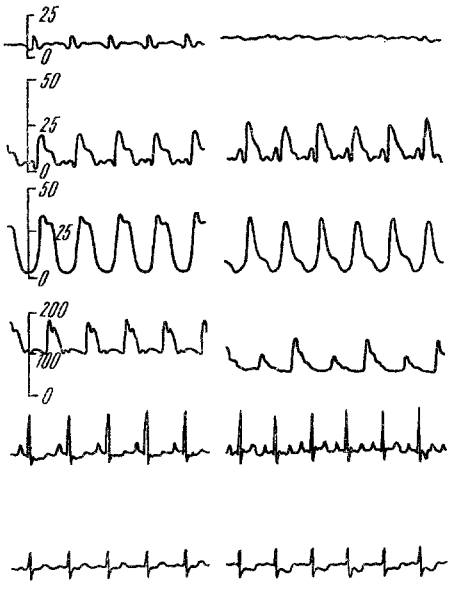

Fig. 2. Change in hemodynamics when the lumen of the circumflex branch of the left coronary artery is occluded. Left) initial curves; right) at 25 sec after thrombosis of the coronary artery. Significance of curves (top to bottom): left atrial pressure, pulmonary artery pressure, right ventricular pressure and femoral artery pressure (in mm Hg). EKG (lead II and V4).

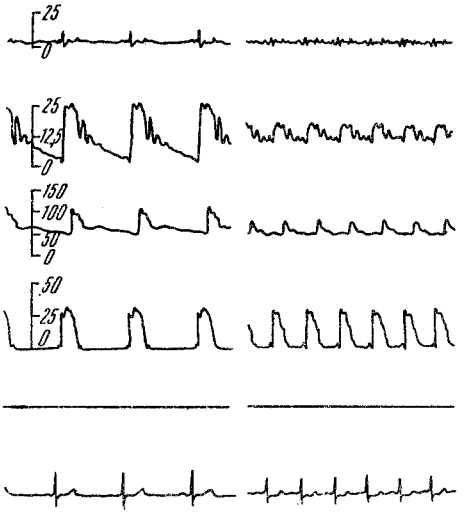

Fig. 3. Change in hemodynamics with occlusion of the circumflex branch of the left coronary artery. Left) initial curves; right) at 40 sec after thrombosis of the coronary artery. Significance of curves (top to bottom): left atrial pressure, pulmonary artery pressure, femoral artery pressure and right ventricular pressure (in mm Hg), tracheal pressure (respiratory pauses noted), EKG (lead V4).

In Fig. 2 are represented the hemodynamic changes which are accompanied by an expressed increase in the systolic and diastolic pressure in the pulmonary artery on a background of decrease in systolic and increase in diastolic pressures in the right ventricle, decrease in arterial pressure and increase in left atrial pressure at a constant cardiac rate.

In another case (Fig. 3), with a small drop in systolic arterial pressure and a negligible increase in left atrial pressure, decrease in right ventricular systolic pressure and a significant increase in the cardiac rate, the pulmonary arterial systolic pressure falls while the diastolic pressure rises significantly. A similar drop in systolic and rise in diastolic pressure in the pulmonary artery is accompanied in several experiments by a clearcut rise in the right ventricular systolic pressure.

Thus, a change in the pulmonary artery pressure during such a reaction is not found to be directly dependent on changes in arterial pressure, right heart activity and left atrial pressure.

Changes in left and right ventricular function were also heterogeneous. Even with a abrupt failure of the left ventricle, which is accompanied by a rise in the left atrial and fall in arterial pressures by 30-40%, right ventricular failure does not develop in most instances. The venous pressure recorded in the opening of the vena cava is not significantly altered. The maximal increase in most of the experiments did not exceed 5-10 mm Hg.

The pronounced changes in rate and depth of respiration observed in certain experiments appeared to have a significant effect on the absolute values of pulmonary artery pressure without producing marked changes on the mean pulmonary artery pressure.

Such relatively small changes in the mean pulmonary artery pressure and in arterial blood oxygen saturation with marked total disruption of the hemodynamics and respiration directly after cessation of blood flow in the coronary artery indicates the extremely high adaptability of the systemic vascular system to major alterations in blood circulation.

SUMMARY

Experimental myocardial infarction was induced in experiments on dogs without opening the thorax. The closure of the coronary vessel was immediately followed by a decline in the arterial pressure, a reduction in the activity of the right and left cardiac ventricles and comparatively slight changes in the mean pressure in the pulmonary artery and in oxygenation of the arterial blood.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
