5.2 Pathophysiology in Pulmonary Embolism

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There has been a widespread and detailed work about pulmonary embolism. It seems well accepted that in cardiopulmonary healthy persons hemodynamic changes are due to the degree of obstruction of the pulmonary vascular bed (1, 5, 6). Our so far available investigation in patients with angiographically proven massive pulmonary embolism confirmed the findings of McIntyre and Sasahara (5, 6) and of Miller and Sutton (7). Since the earliest time we have investigated our patients has been at least one and a half hours after the clinical event of pulmonary embolism, we believe, that these patients are already a selection of those who primarily survived. Data on the immediate response to massive pulmonary embolism in man are therefore not available. The central problem appears to be the ability of the right ventricle to compensate for a sudden increase in pulmonary vascular resistance. Therefore it seemed most interesting to us to get some information about the development of right ventricular force in the first essential seconds and minutes. Thus we evaluated the first reaction post embolization in dogs.

Methods

Experiments were carried out in 12 dogs of 35-70 kg. The animals were thoracotomized in nembutal anesthesia to insert the force probes into the myocardium of the left and right ventricle (4). Furthermore catheters were brought into the pulmonary artery, left ventricle and aorta to monitor the respective pressures. Additionally cardiac output was measured by a thermodilution catheter in the aorta. Heart rate was determined from ECG registration and breathing frequency from intrapleural pressure changes. Before and shortly after the embolization blood samples were taken from the aorta for determination of epinephrine and norepinephrine (2).

The embolus was prepared two hours before injection in form of 50 ml autologues blood which was mixed with tantal powder. After the death of the animal the lung was inflated with air and an x-ray was taken for localization of the embolus. A big catheter in the jugular vein allowed us to inject the embolus directly into the right atrium with a minimum of destruction of the thrombus. After all dogs had been prepared in this way anesthesia was terminated and the dogs were allowed to awake and move. At that time the embolus was injected.

Results

4 out of 12 dogs died within the first min. 8 survived for about one hour and were than killed by a nembutal overdose.
In all animals we observed a steep increase of right ventricular force followed by an increase in pulmonary arterial pressure. Simultaneously a decrease of aortic pressure and local left ventricular force occurred. In the dogs dying immediately the systolic pressure in the pulmonary artery fell after about 30 seconds while diastolic pulmonary arterial pressure remained stable or even increased. Myocardial force of the right ventricle than decreased rapidly by 200% in 15 sec. Systemic pressure measured in the left ventricle and the aorta was diminished and left ventricular force fell. Cardiac output is found to be zero few seconds after embolism. The typical cardiovascular response to large emboli in dogs which survived is demonstrated in Fig. 1. As shown before pressures of the pulmonary artery and the local force of the right ventricle increased. This is again accompanied by a decrease of the aortic pressure and left ventricular force. After 20 sec force of the right ventricle and pressures in the pulmonary artery began to decrease. In the aorta we measured a further decrease of pressure and left ventricular force. 50 sec after embolization pulmonary arterial and aortic pressures rose again as well as the forces of both ventricles. When the pressure in the pulmonary artery had reached a second peak, it then declined more slowly. At the moment the pulmonary arterial pressure starts to rise again to the second peak cardiac output and arterial pressure increased continuously close to the initial values. Calculation of the resistance of the pulmonary vascular bed reveals a tremendous increase immediately after embolization. This was followed by

![Typical Cardiovascular Response to a Massive Pulmonary Embolisation in a Conscious Dog](image)

**Fig. 1.** Relative changes in pulmonary arterial pressure, left and right ventricular local force and pressure in the aorta. (40 is f.e. 4 times the initial value). Above: cardiac output in percent of the value shortly before embolism.