# EFFECT OF CATECHOLAMINES ON VELOCITY OF BLOOD FLOW IN THE PULMONARY CIRCULATION DURING GENERAL ANESTHESIA

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UDC 612.215.8.014.46:[615.357.452.015.2:615.212.7

Experiments (42) on intact and anesthetized dogs showed that catecholamines, when administered to intact animals against the background of morphine, reduce the velocity of blood flow in vessels of the pulmonary circulation. After preliminary administration of atropine and chlorpromazine, catecholamines do not change the velocity of blood flow along the pulmonary vessels. Endotracheal anesthesia with ether and oxygen in stage $III_2$ increases the velocity of the pulmonary blood flow, but administration of catecholamines against the background of anesthesia has no effect on the pulmonary circulation time. Preliminary administration of morphine, atropine, and chlorpromazine has no effect on the velocity of the pulmonary blood flow under these conditions.

No unanimity yet exists regarding the velocity of the blood flow in the pulmonary circulation. The time taken for blood to pass through the vessels of the pulmonary circulation, determined by various methods, is between 3 and 11 sec. In the presence of lesions of the mitral valve or circulatory failure, the pulmonary circulation time may be increased to 28 sec [1, 5, 7, 8].

## EXPERIMENTAL METHOD

To study the pulmonary circulation time a platinum–hydrogen method was used; this is based on the ability of platinum to change its potential under the influence of hydrogen ions. The method was developed in 1967 in the laboratory of intracardiac research of the A. V. Vishnevskii Institute of Surgery, Academy of Medical Sciences of the USSR, by Yu. D. Volynskii, S. Ya. Berdikyan, and L. I. Abaskulieva. The suggested method differs from other methods using platinum [6] in that the indicator is injected into the heart and several platinum electrodes can be fitted, thus enabling indicator dilution curves to be recorded simultaneously from different parts of the vascular system.

Altogether 42 experiments were carried out to determine the effect of catecholamines on the velocity of the pulmonary blood flow in intact (series I) and anesthetized (series II) animals. Before the start of the experiment the dogs were injected intramuscularly with morphine solution (10 mg/kg body weight). After 30 min the femoral vessels were exposed and heparin (0.05 ml/kg) injected. Under x-ray control a platinum–hydrogen electrode was placed at the origin of the aorta from the left ventricle, and a catheter was introduced into the right atrium for injection of ascorbic acid, the source of hydrogen ions. A freshly prepared 5% solution of ascorbic acid (10-14 mg/kg) was used. The way from the right atrium to the origin of the aorta can be taken as the extent of the pulmonary circulation.

The dilution curves were recorded on a type 42-B 4-channel Elema myograph. Besides determination of the pulmonary circulation time, the systemic pressure in the femoral artery, the EKG, and the pressure in the femoral vein were recorded. After the initial parameters had been recorded, succinylcholine chloride

Laboratory of Pathophysiology, Research Institute of Clinical and Experimental Medicine, Ministry of Health of the Azerbaidzhan SSR, Baku. (Presented by Academician N. V. Antelava.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 69, No. 1, pp. 25-27, January, 1970. Original article submitted April 18, 1969.

(0.05 ml/kg) was injected intravenously, the animal was intubated, and artificial respiration of an ether–oxygen mixture was started. The depth of anesthesia was verified electroencephalographically. Because of the importance of hypoxia under the experimental conditions used, parallel observations were made on the indices of the acid–base balance. Catecholamines were injected into the femoral vein: adrenalin and noradrenalin in doses of 0.025 mg/kg, phenylephrine in a dose of 0.25 mg/kg. At the height of the hemodynamic reaction, 40-60 sec after administration of the catecholamines, ascorbic acid solution was injected into the heart. With the object of discovering the role of different components of the nervous system in the response reaction, in some experiments the muscarine-like (M-) and nicotine-like (N-) cholinergic structures were first blocked by intravenous injection of 0.1% atropine solution in a dose of 0.1 mg/kg, and in others the reticular formation of the brain stem was first blocked by intravenous injection of 2.5% chlorpromazine solution (10 mg/kg). The numerical results were subjected to statistical analysis by the method of direct and indirect differences, using Moldenhauer's formula [3].

## EXPERIMENTAL RESULTS

The pulmonary circulation time against the background of morphine was $6.1 \pm 0.74$ sec. Intravenous injection of adrenalin, noradrenalin, and phenylephrine into these animals increased this time to $13.56 \pm 0.91$ ($P < 0.01$), $9.1 \pm 0.88$ ($P < 0.05$), and $8.6 \pm 1.62$ sec ($P > 0.05$), respectively. Blocking of the M- and N-cholinergic structures, like blocking of the reticular formation, caused no significant changes in the pulmonary circulation time of intact animals: against the background of atropine this index was $6.2 \pm 1.68$ sec, and of chlorpromazine $5.6 \pm 0.63$ sec. No direct causal relationship could be established between the velocity of the blood flow and the heart rate.

When adrenalin was injected into animals against the background of atropine, the pulmonary circulation time was $5.1 \pm 1.17$ sec ($P > 0.05$), and the corresponding values for injection of noradrenalin and phenylephrine was $5.7 \pm 1.65$ sec ($P > 0.1$) and $6.9 \pm 0.97$ sec ($P > 0.1$). After preliminary blocking of the reticular formation by chlorpromazine, adrenalin decreased the pulmonary circulation time to $5.3 \pm 1.53$ sec ($P > 0.1$) and noradrenalin to $4.9 \pm 0.69$ sec ($P > 0.1$), while phenylephrine increased it slightly to $6.2 \pm 0.72$ sec ($P > 0.1$). Administration of catecholamines after blocking of M- and N-cholinergic structures and after blocking of the reticular formation of the brain stem thus caused no appreciable changes in the velocity of the blood flow in the pulmonary circulation.

Intratracheal ether–oxygen anesthesia at the $III_1$ level, associated with adequate ventilation, increased the velocity of the blood flow. At the $III_2$ level, the circulation time was $3.5 \pm 0.49$ sec ($P < 0.05$). Under these circumstances, administration of catecholamines did not slow the blood flow: when morphine premedication was given, the pulmonary circulation time after injection of adrenalin was $3.3 \pm 1.04$ sec, after noradrenalin $4.5 \pm 1.13$ sec, and after phenylephrine $4.1 \pm 0.52$ sec.

Preliminary atropinization or administration of chlorpromazine had no appreciable effect on the velocity of the pulmonary blood flow: in animals anesthetized after atropine the circulation time was $3.7 \pm 0.78$ sec, and after chlorpromazine $3.3 \pm 0.69$ sec.

Administration of catecholamines under these conditions did not significantly change the circulation time: against the background of atropine, after injection of adrenalin, noradrenalin, and phenylephrine it was $3.3 \pm 0.43$, $4.2 \pm 1.88$, and $3.3 \pm 0.69$ sec, respectively; against the background of chlorpromazine the results were very similar: after adrenalin $3.0 \pm 1.16$ sec, after noradrenalin $4.5 \pm 0.90$ sec, and after phenylephrine $4.3 \pm 1.80$ sec.

Comparison of changes in the velocity of the blood flow in the pulmonary circulation with changes in the height of the venous pressure revealed a definite parallel: in intact animals administration of actecholamines following preliminary injection of morphine was accompanied by a marked rise of venous pressure; preliminary injection of atropine and chlorpromazine abolished this reaction. During intratracheal ether–oxygen anesthesia, with all forms of premedication tested, after administration of catecholamines the venous pressure usually did not rise appreciably, in agreement with results obtained by other workers [2, 4], indicating the role of the inflow of blood from the systemic circulation in regulation of the pulmonary circulation.

The results of these experiments show that among the factors concerned in the response reaction of the pulmonary vessels to administration of catecholamines, an important role is played by the initial state of the nervous system.

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