CORONARY BLOOD FLOW IN CATS IN A MODIFIED GASEOUS ENVIRONMENT

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To analyze factors determining the partial pressure of oxygen in the myocardium, the blood supply of the myocardium has been studied by a thermoelectric method [2, 3, 6] and by the hydrogen clearance method [7]. By the first of these methods it is possible to study the dynamics of the blood supply but not to obtain any idea of quantitative changes in the blood flow. The second method, on the other hand, yields quantitative data on the local blood flow at a particular moment of the experiment but cannot shed any light on the dynamics of the blood supply. It was decided to attempt to study the coronary blood flow by an up to date technique, which not only yields accurate quantitative data but which can also be used to study the dynamics of blood supply and also the true character of phases in a coronary blood flow under different conditions.

The aim of this investigation was to study the coronary blood flow during hypoxia, hyperoxia, and hypercapnia.

EXPERIMENTAL METHOD

Acute experiments were carried out on 19 male cats weighing 3.5-5 kg, with open chest and artificial ventilation of the lungs, under pentobarbital anesthesia. Coronary blood flow was measured in the circumflex branch of the left coronary artery (in some experiments the blood flow in the corresponding coronary vein was recorded). The cardiac output was measured simultaneously. The coronary blood flow and also the blood flow in the ascending aorta were investigated by an ultrasonic method [4, 5]. The ultrasonic transducer used to study the coronary blood flow had an internal diameter of 1-1.5 mm and a length of 2.5 mm. The cardiac output was measured by transducers located on the ascending aorta (diameter of transducers 6-8 mm). The stroke volume and cardiac output values were estimated by means of an electronic integrator. The blood flow was recorded on the Mingograph-81 and N-338 instrument and also on magnetic tape. In some experiments the degree of saturation of arterial blood with oxygen (S_aO_2) was determined with a micro-Astrup apparatus. In the original state (after induction of anesthesia) it averaged 96%. To create the conditions of hypoxic hypoxia and hypercapnia, the animals were made to breathe gas mixtures with reduced oxygen concentrations (10 or 5% O_2 in nitrogen) or with an increased carbon dioxide concentration (5 and 10% CO_2 in air), and also pure oxygen. The gas mixtures were administered from Douglas bags by means of an artificial respiration apparatus.

EXPERIMENTAL RESULTS

The volume velocity of the blood flow in the circumflex branch of the left coronary artery averaged 9.1 ml/min (with variations from 6 to 13 ml/min in different experiments. The cardiac output averaged 380 ml/min (from 320 to 450 ml/min in different animals). The stroke volume averaged 2.4 ml (from 2 to 2.7 ml). The linear velocity of the blood flow in the coronary artery averaged 23 cm/sec (from 12 to 37 cm/sec).

The values obtained for the linear velocity of the blood flow in the coronary arteries, incidentally, are relatively low compared with those observed in other vessels (57 cm/sec in the lobar arteries of the lungs, 41 cm/sec in the pulmonary veins [8], and 60 cm/sec in the carotid arteries). The reason is probably that the blood flow along the coronary vessels takes place chiefly during diastole of the heart, when the velocity of the blood flow in the aorta is close to zero [11-13]. This fact may have definite biological importance, namely

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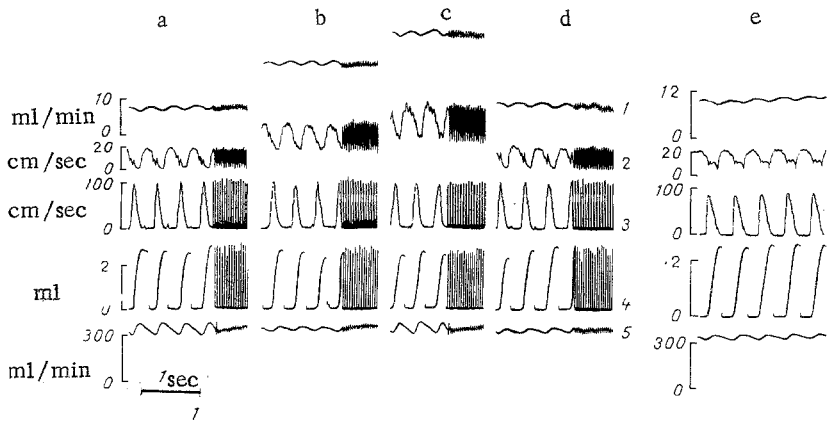

Fig. 1. Effect of hypoxia on coronary blood flow and cardiac output. 1) Mean values of flow in circumflex coronary artery; 2) phasic coronary blood flow in circumflex artery; 3) shape of pulsatile blood flow in ascending aorta; 4) stroke volume (readings of electronic integrator); 5) cardiac output. a) Before procedure (inhalation of atmospheric air); b, c) 3 and 10 min after beginning of inhalation of gas mixture containing 5% O_2; d) 10 min of recovery; e) phasic blood flow in coronary vein during inhalation of air (second curve from top). Considerable elevation of curve of phasic coronary blood flow during hypoxia above zero line indicates increase in resistance of coronary vessels to their extravascular compression during cardiac contraction.

that the relatively slow blood flow along the coronary vessels creates favorable conditions for more complete extraction of oxygen from the coronary capillaries than in other organs [11, 12]

The coronary blood flow in cats is clearly pulsatile in character. During synchronous recording of the blood flow in the aorta and the phasic blood flow in the coronary artery, it is clear (Fig. 1a) that the maximal coronary blood flow is observed during diastole. Minimal values of the coronary blood flow, close to zero, occur at the end of systole. At the beginning of systole a more or less well marked rise (a small wave or peak) can be distinguished on the coronary blood flow curve, and it coincides in time with the beginning of the increase in blood flow in the ascending aorta and is evidence that the blood pressure in the aorta, which is rising at that moment, is able to overcome the source of extravascular compression of the coronary arteries due to contraction of the myocardium. A similar character of phasic coronary blood flow was observed previously in experiments on dogs [12, 13]. The blood flow in the coronary vein is similar in its quantitative and temporal parameters and also in its shape to that observed in the coronary artery (Fig. 1e).

During inhalation of gas mixtures with a reduced oxygen concentration an increase in coronary blood flow was observed, depending on the severity of hypoxia: during inhalation of a gas mixture containing 10% O_2, when S_{aO_2} averaged 90% (from 87 to 92%) an increase in coronary blood flow took place — on average by 18.5% (from 7 to 33%) of the initial level. A mixture containing 5% O_2 caused an increase in the coronary blood flow on average to 218% of the initial level (from 155 to 333% in different experiments); S_{aO_2} under these circumstances fell on average to 66.5% (from 51 to 81%). In the majority of experiments the cardiac output was unchanged under these conditions or increased very slightly (by 5-10%), i.e., by a much lesser degree than the coronary blood flow. This is evidence of active and considerable dilatation of the coronary vessels under hypoxic conditions [1, 10].

Dilatation of the coronary vessels was reflected in the curve of coronary blood flow by a marked rise above the zero line, and thus by the appearance of a new, or appreciable increase in an existing, constant component. Comparison of the dynamics of the blood flow in the aorta and coronary artery leads to the conclusion that such a marked increase in the coronary blood flow took place on account of both its diastolic and its systolic components. This is evidence that under conditions of hypoxic hypoxia the resistance of the dilated coronary vessels to their extravascular compression rises considerably [9], whereas during inhalation of air, this compression almost completely blocks the blood flow in them during cardiac contraction. Hyperoxia (inhalation of oxygen) caused a reduction of 10-15% in the coronary blood flow.

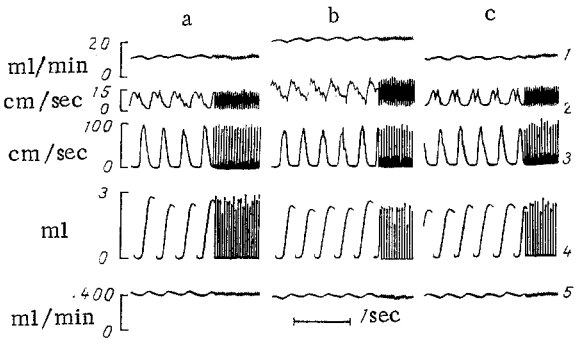

Fig. 2

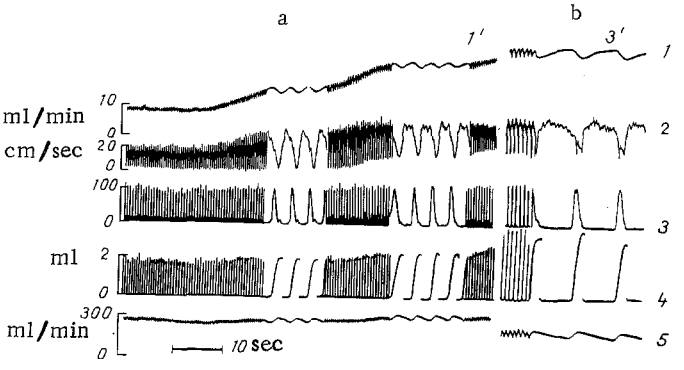

Fig. 3

Fig. 2. Effect of hypercapnia on coronary blood flow and cardiac output. a) Before procedure; b) 10 min after beginning of inhalation of gas mixture containing 10% CO_2; c) 10 min of recovery. Remainder of legend as to Fig. 1.

Fig. 3. Effect of asphyxia (cutting off artificial respiration) on coronary blood flow and cardiac output. a) Beginning of asphyxia; b) 3 min after beginning of asphyxia. Remainder of legend as to Fig. 1.

Hypercapnia, caused by inhalation of gas mixtures containing 5 and 10% CO_2 in air, is always accompanied by slowing of the cardiac contractions, but because of an increase in stroke volume of the heart in the majority of experiments the cardiac output rose somewhat (by 15-20%). Only in a few experiments did it fall slightly. Meanwhile during hypercapnia the coronary blood flow always was increased — on average to 129% of its initial level (with 5% CO_2) and to 156% (with 10% CO_2) (Fig. 2). In hypercapnia, just as in hypoxia, the curve of the coronary blood flow rose appreciably above the zero line together with the constant component of the flow, and systolic ejections of blood appeared (Fig. 2b).

During asphyxia (Fig. 3), when the action of hypoxia was combined with that of hypercapnia, the coronary blood flow rose very considerably (six-tenfold) compared with its initial level. The latent period of this response could be quite long (15-30 sec). The maximal increase of blood flow was observed between 1.5 and 2.5 min after the beginning of asphyxia, then followed a gradual fall of the blood flow to zero values (in the course of 5-6 min). The cardiac output under these circumstances increased a little in some experiments (by 15-20%), but in others, immediately after the beginning of asphyxia it began to fall gradually. The shape of the coronary blood flow was changed. During asphyxia those tendencies observed during isolated exposure to hypoxia and to hypercapnia were seen particularly clearly: the appearance of a new, or increase in an existing, constant component of the blood flow, the value of which depended directly on the total increase in blood flow under these conditions, and the appearance of a systolic wave on the coronary blood flow curve. In the terminal phase of asphyxia only systolic ejections could be seen on the curve — the diastolic flow was absent in this phase. The linear velocity of the blood flow under the conditions studied in these experiments, incidentally, changed much less than the volume velocity of the coronary blood flow, and this difference was particularly marked in asphyxia.

Thus, in hypoxic hypoxia and hypercapnia a considerable increase in the coronary blood flow is observed, and is evidently due to dilatation of the coronary vessels. Changes in shape of the phasic coronary blood flow arising under these circumstances indicate an increase in resistance of the coronary vessels to their extravascular compression during cardiac contraction and, consequently, an increase in the contribution not only of the diastolic, but also of the systolic fraction to the total increase in coronary blood flow under these conditions.

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TREND OF CHANGES IN CATECHOLAMINE CONTENT AND LIPID FATTY ACID COMPOSITION OF THE LUNGS IN TRAUMATIC SHOCK

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Profound disturbances of hormone, mediator, and enzyme metabolism in the lungs in various types of shock are now familiar. Considerable shifts have been found in lipid metabolism and the content of surfactants of lipid nature in the lungs has been shown to be reduced in shock [2, 3, 9, 11, 13]. Changes in metabolism in the lungs are closely interconnected with the balance between several hormones and mediators that are actively removed by the lungs from the blood stream or are synthesized in them [10, 12, 14, 15].

Considering the importance of the sympathico-adrenal system in the pathogenesis of traumatic shock it was decided to study to what extent changes in its activity are reflected in the metabolic status of the lungs. The aim of this investigation was a comparative study of trends in catecholamine content and lipid fatty acid composition in the lungs at different stages of traumatic shock.

EXPERIMENTAL METHOD

Experiments were carried out on 64 adult male dogs. Traumatic shock was produced by the method described previously [8]. Intact animals (series 1) served as the control. The animals were killed at once (series 2), 1 h after trauma (series 3), or in the terminal stage of shock (series 4). Adrenalin and noradrenalin in tissues of the lungs, pulmonary arteries and veins, and also in the blood samples taken from the carotid artery (arterial blood) and the right atrium (venous blood) were determined quantitatively by the method in [5]. Lipids were extracted from the lungs by a chloroform–methanol mixture (2:1 by volume) and triglycerides and nonesterified fatty acids (NEFA) were obtained by thin-layer chromatography on silica-gel; the fatty acid composition of the lipids was studied by gas–liquid chromatography [4].

EXPERIMENTAL RESULTS

The adrenalin concentration in the lungs remained unchanged throughout the experiments, but in the pulmonary arteries it rose immediately after trauma. A considerable increase in the adrenalin concentration in the pulmonary veins, observed throughout the torpid phase of shock, should be noted. The noradrenalin level in the lungs fell immediately after trauma, returned to the initial value after 1 h, and fell again considerably in the terminal period. Similar changes in noradrenalin concentrations also were observed in tissues of the pulmonary arteries and veins (Table 1).

Comparison of the catecholamine concentrations in arterial and venous blood shows that immediately and 1 h after trauma the venous blood adrenalin level was higher than the

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