The present investigation confirmed that the CP increases the efficacy of cardioplegic protection and demonstrated that the most favorable conditions for metabolic cardioplegic protection of the myocardium are provided by normothermic injection of a cardioplegic solution with CP. Hence this leads to the conclusion, of great practical importance, that no useful purpose can be served by the use of hypothermia during cardioplegic protection of the myocardium, with the aim of preserving the existing solution and to ensure the most effective recovery of the exhausted HEP pool.

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BLOOD FLOW VELOCITY — A CONSTANTLY ACTING FACTOR IN DILATATION OF LARGE ARTERIES

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KEY WORDS: blood flow; sensitivity; velocity; diameter of artery.

The lumen of many main arteries of the systemic circulation in dogs and cats varies in response to a change in velocity of the blood flow [2, 4, 7, 9, 11]. However, in all investigations so far undertaken in which the sensitivity of arteries to blood flow velocity has been studied, dilatation of arteries was observed only in response to a considerable increase in blood flow velocity. It is therefore not yet clear whether there is continuous, uninterrupted regulation of the lumen of arteries in accordance with the blood flow velocity or whether changes in diameter take place only in response to considerable changes in blood flow.

The aim of this investigation was to determine whether arteries "monitor" changes in blood flow velocity continuously in the form of changes in their own diameter, and within what range of blood flow rates they exercise this property. The investigation was carried out on both the femoral and carotid arteries of cats, in which the presence of sensitivity to blood flow has been established [2, 9], and also on the renal artery, in which this property has not previously been found.

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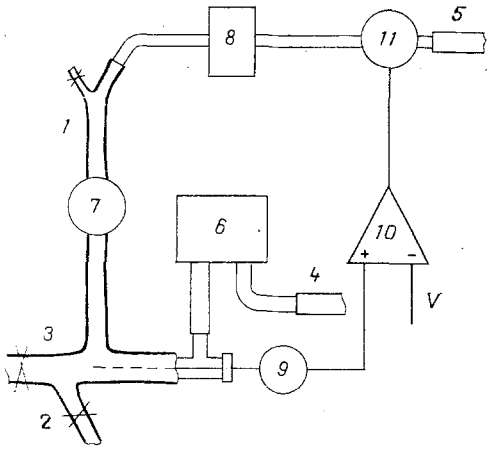

Fig. 1. Scheme of experiments. 1) Left RA, 2) right RA, 3) aorta, 4) carotid artery, 5) renal vein, 6) perfusion pump, 7) diameter transducer, 8) transducer of electromagnetic flowmeter. Pressure stabilization system: 9) electromanometer, 10) differential amplifier, 11) hydraulic throttle, V) constant voltage assigning level of pressure stabilization.

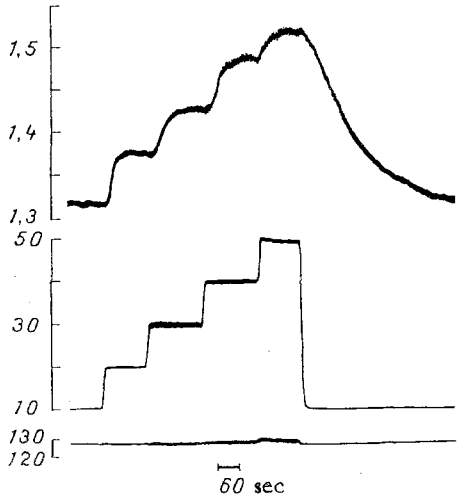

Fig. 2. Dilatation of RA in response to blood flow through it rising in steps of 10 ml/min. From top to bottom: diameter (in mm), volume velocity of blood (in ml/min), pressure in artery (in mm Hg).

EXPERIMENTAL METHOD

The procedure of measuring the diameter of the femoral and carotid arteries during changes in blood flow velocity was described fully previously [1, 2]. The scheme of the experiments on the renal artery (RA) is illustrated in Fig. 1. Laparotomy was performed on cats anesthetized with urethane and chloralose (0.6 and 0.04 g/kg body weight, respectively), the intestine was removed, and the left RA and a segment of the aorta 2 cm long distally to the origin of the renal arteries was isolated from the surrounding tissues, and the right RA was ligated. Heparin (1500 U/kg) was injected intravenously, after which the left RA was connected by rubber tubes with the right femoral vein. The aorta was ligated above and also 2 cm below the origin of the renal arteries. A cannula was introduced into the peripheral segment of the divided aorta toward RA, and connected to the outflow of a PN-3 perfusion pump [6]; the inlet tube of the pump was connected to the right carotid artery. The pressure in RA was stabilized at 100 mm Hg by means of an automatic tracing system [1], consisting of electromanometer, pressure transducer, power amplifier, and hydraulic throttle, with electrodynamic control. A segment of the tube connecting RA to the femoral vein was used as the throttle. The cross-section and, consequently, the hydraulic resistance of this tube could be varied so that the

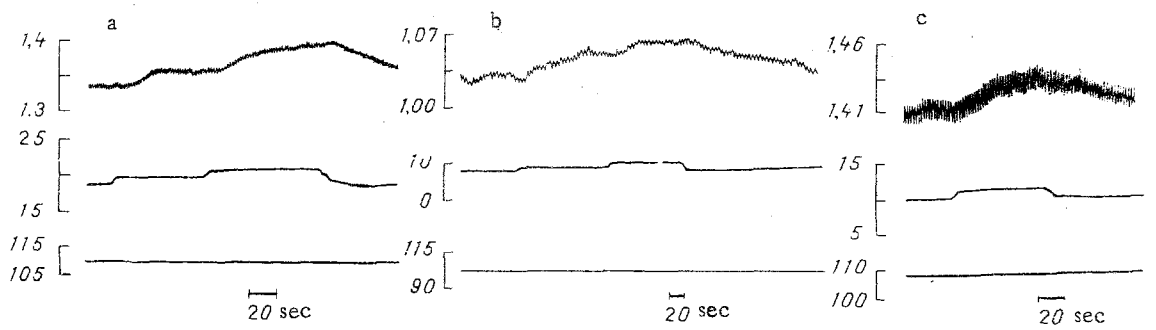

Fig. 3. Dilatation of renal (a), femoral (b), and carotid (c) arteries in response to increase of 1 ml/min in blood flow. From top to bottom: diameter (in mm), blood flow (in ml/min), pressure in artery (mm Hg).

pressure in the test artery remained unchanged. Stabilization of the pressure in the artery is essential since changes in diameter in response to small changes of flow (about 1 ml/min) must naturally be small, and for them to be recorded reliably, fluctuations of diameter connected with instability of arterial pressure must be abolished.

The blood flow in RA was measured by an RKE-1 electromagnetic flowmeter with D2M mainstream flow transducer, and the external diameter of the segment of this artery was measured with a contact capacitive displacement transducer [3]. Since respiratory movements lead to considerable displacements of the abdominal vessels, which make recording of diameter more difficult, before the beginning of the measurements the animals were artificially ventilated, pneumothorax induced, and during the subsequent course of the experiment, succinylcholine chloride was injected continuously into a vein (150 µg/kg/min). This procedure greatly reduces the amplitude of movements of RA connected with the act of respiration. A diameter transducer was then applied to the left RA, after which the peritoneal cavity was irrigated with Ringer's solution, the temperature of which was kept constant at 36-37°C throughout the experiment.

In each experiment 20-30 min after the end of the operation the blood flow rate in RA was increased from 20-25 to 40-45 ml/min and the presence of a reaction of the artery to an increase in the blood flow velocity was determined. Next, by increasing the input into the pump, the blood flow rate in the artery was increased from 10 to 40-60 ml/min in steps of 10 ml/min, and in that way dependence of the diameter of RA on the blood flow velocity could be determined. Finally, tests were carried out to determine whether RA responds by dilatation to an increase in flow rate of 1 ml/min, from an initial flow rate of 20, 30, and 40 ml/min. In the final stage of the experiment the blood flow was reduced to 2-5 ml/min to determine whether the artery remained capable of responding to a change in blood flow velocity at such low flow rates. The order of the measurements on the carotid and femoral arteries was the same.

EXPERIMENTAL RESULTS

The trace of changes in the diameter of RA caused by an increase in blood flow with 10 ml/min steps (Fig. 2) shows that each step of increased flow led to dilatation of the artery. Within the range from 10 to 40 ml/min the increase in diameter in response to every 10 ml/min of rise of blood flow was about equal. However, a further increase in blood flow was accompanied by a lesser degree of dilatation of the artery, the latent period of the dilator response increased, and the rate of increase of diameter was reduced.

The increase in diameter of RA due to a twofold increase in the blood flow rate (from 20 to 40 ml/min) was 23 ± 8% of the initial value, which corresponds to a reduction in hydraulic resistance of this RA by about 2.5-3 times. Dilatation of RA in response to a twofold increase in the blood flow rate can therefore be so great that the fall of pressure in it in response to such an increase in blood flow rate goes below the initial value.

The trace in Fig. 3a shows the response of RA to a change in volume velocity of blood flow in it by 1 ml/min: from 19 to 20 ml/min and from 20 to 21 ml/min. Changes in diameter of the femoral and carotid arteries in a response to an increase in blood flow of 1 ml/min are illustrated by Fig. 3b, c. In all cases such a small increase in blood flow caused a distinct and definite dilatation of the arteries. It can thus be concluded that the system of local regulation of arterial tone, which is sensitive to the blood flow velocity, acts continuously, bringing the lumen of the arteries to a size which corresponds to the rate of

flow of the blood in them. Incidentally, the response of an artery to an increase of 1 ml/min in its blood flow rate can be recorded not only at physiological blood flow values (25-35 ml/min for the renal and 10-20 ml/min for the femoral and carotid arteries), but also with very low flow rates, that are hardly practical under physiological conditions.

It thus follows from these experiments that arteries can respond by changes in their lumen to sufficiently small fluctuations of blood flow velocity, and as a result, they are continuously modifying their lumen to match the velocity. RA of cats, like all main arteries studied previously, is sensitive to the blood flow velocity. This result, together with those of previous investigations [2, 4, 7, 9, 11], leads to the conclusion that all main arteries of the systemic circulation possess sensitivity to blood flow velocity. This sensitivity ensures constant regulation of the lumen and, consequently, the hydraulic resistance) of the arteries to correspond to the blood flow velocity. This conclusion modifies existing ideas on the functional role of sensitivity of arteries to blood flow velocity.

In fact, starting with the first study [10] which showed dilatation of the femoral artery during working hyperemia of the leg muscles of cats, the view was held that dilatation of arteries is essential only for the largest possible blood flow to be achieved in intensively working skeletal muscles. The authors of [8], who recorded an increase of 3% in diameter of the femoral artery at the peak of working hyperemia of the lower limb muscles in dogs, postulated that this response prevents passive collapse of the artery due to the lowering of its transmural pressure as a result of the increase in blood flow. Dilatation of arteries during working hyperemia was thus regarded as an auxillary mechanism, facilitating achievement of maximal blood flow in intensively working muscles. These views are linked with the fact that no significant increase in diameter of the main arteries of skeletal muscles has been observed during a large and frequent increase in blood flow [4, 8].

The writers have shown that there is a significant increase in diameter of the main artery of the kidney, i.e., an organ not characterized on the whole by any considerable degree of working hyperemia [5] and, in addition, they have shown that dilatation of the renal, carotid, and femoral arteries during an increase in blood flow velocity is by no means an "emergency measure," effected only when the blood flow reaches values many times higher than initially, and always modifying the lumen of arteries to make it correspond to the velocity of blood flowing through them.

Consequently, the blood flow itself is a constantly acting factor in dilatation of arteries. Dilatation of arteries due to increased blood flow may be considerable, and this suggests that sensitivity to blood flow velocity plays an essential role in the regulation of the blood flow of an organ. To test this hypothesis direct experimental proof of the presence of such sensitivity must be obtained in small intramural arteries of organs.

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