PRESSURE DROP STABILIZATION IN CONDUCTING ARTERIES IN RESPONSE TO INCREASED BLOOD FLOW IN RATS

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UDC 612.13-06:612.143

KEY WORDS: small arteries; pressure gradient; tone.

The blood flow in mammalian organs can vary many times over. If arteries conducting blood to organs behaved under these circumstances like rigid tubes, the pressure drop on them would change by the same number of times as the blood flow. Since the mean pressure in the cat femoral artery even at rest is about 10% lower than the mean pressure in the arch of the aorta [3], with a tenfold increase of blood flow in the hind limb the mean pressure in the femoral artery would fall virtually to zero. This great pressure drop could only be prevented by dilatation of the conducting arteries. According to calculation, for the blood flow along the femoral artery of a dog to be increased 2-3 times but the pressure in it not to fall, its internal diameter would have to be increased by 20-30% [4]. However, in corresponding experiments, the increase in diameter was only 3-4% [4], and this could not significantly reduce the pressure drop on the femoral artery. The technique used in these investigations may perhaps have been comparatively traumatic. Whatever the case, later results [1, 2] indicate a significantly greater degree of dilatation of the main arteries of cats and dogs in response to an increase in blood flow and, consequently, that this response could considerably reduce the increase in pressure drop.

This paper describes an attempt by direct measurement to determine by how much the pressure drop is increased in the conducting arteries during an increase in blood flow. For this purpose, dependence of the magnitude of the pressure drop between the arch of the aorta and the distal end of a comparatively small artery on the blood flow was investigated.

EXPERIMENTAL METHOD

Wistar rats of both sexes weighing 320 ± 30 g were anesthetized with pentobarbital (50 mg/kg, intraperitoneally, maintenance dose 17 mg/kg/h). Tracheotomy was performed. The body temperature was maintained automatically at 36.5-37.5°C. Blood clotting was prevented by injection of heparin (1500 U/kg intravenously, maintenance dose 150 U/(kg·h).

The experimental method is shown schematically in Fig. 1. A polyethylene cannula 2, blood from which passed through the throttle valve 3 of the flow control system, from which it

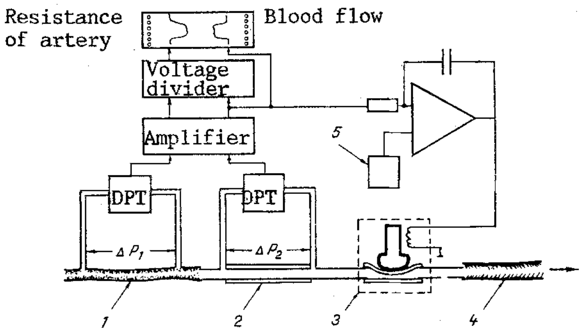

Fig. 1. Scheme of method of determining hydraulic resistance of an artery (explanation in text).

Laboratory of Biomechanics and Regulation of the Circulation, All-Union Cardiologic Scientific Center, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR V. N. Smirnov.) Translated from Byulleten' Éksperimental'noi Biologii Meditsiny, Vol. 104, No. 9, pp. 259-261, September, 1987. Original article submitted December 5, 1986.

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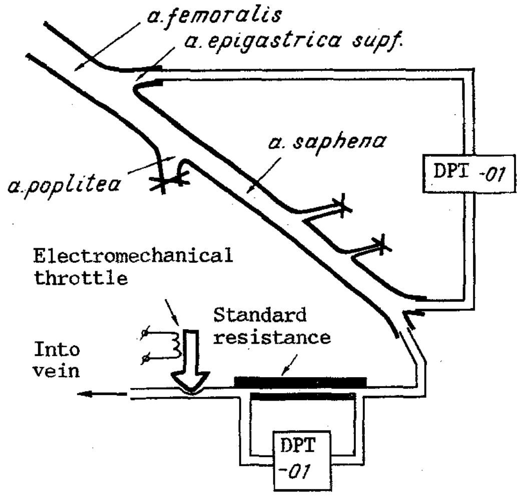

Fig. 2. Diagram of experimental setup (explanation in text).

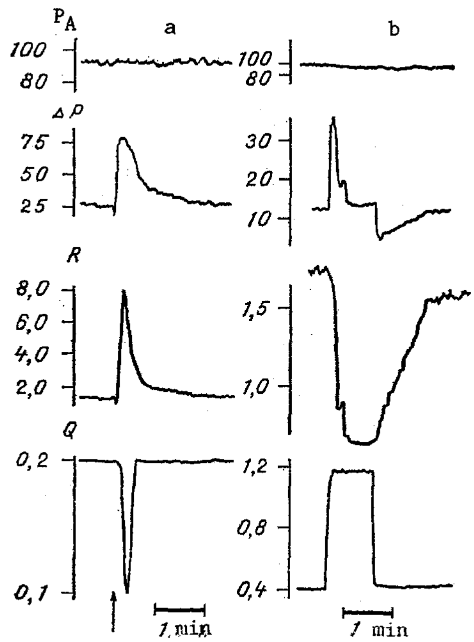

Fig. 3. Traces of responses of an artery to injection (arrow) of 10 μl of noradrenalin hydrotartrate solution in a concentration of 2×10^{-5} g/ml into the blood stream (a) and to an increase in blood flow along the artery (b). P_A) Pressure in arch of aorta (in mm Hg); ΔP) pressure drop on artery (in mm Hg), R) resistance of artery (in relative units), Q) blood flow (in ml/min).

passed through the bubble flowmeter 4 and was discharged into the right femoral vein, was inserted into the distal end of the left saphenous artery 1. To measure the pressure in the distal end of the saphenous artery, a thin polyethylene catheter 5 was passed through the cannula 2. The pressure in the arch of the aorta was measured by a W 102 RFT electromanometer (East Germany) through a polyethylene catheter introduced into the brachial artery. The pressure drop between the arch of the aorta and the distal end of the saphenous artery was measured by means of a DMI-06 differential pressure transducer, the chambers of which were filled beforehand with PMS-6 polymethylsiloxane. The pressure drop on the cannula 2 was measured by a similar differential pressure transducer as an indicator of the flow rate. It was shown by means of the bubble flowmeter that within the range from 0.1 to 3 ml/min this pressure drop is directly proportional to the volume velocity of the blood flow with an accuracy of not less than 10%. Thus, by measuring the pressure drop on the cannula 2, it is possible to determine the blood flow. To rule out errors connected with possible changes in viscosity of the blood, values of the pressure drop on the cannula were expressed as a ratio of the blood flow measured with the bubble flowmeter every 30 min.

The required changes of blood flow through the saphenous artery were produced by the automatic flow control system. The principle of its work is that the electromechanical throttle 3 (Fig. 1) compresses by a measured amount the elastic polyvinylchloride tube along which blood is discharged into the vein so that the blood flow signals coincide with the demand signal. The demand signals were shaped by means of a G6-31 functional generator so that they gave rise to changes in the blood flow with time, of "stepwise" or "sawtooth" shape and of varied duration.

EXPERIMENTAL RESULTS

A characteristic trace of the change in pressure drop between the arch of the aorta and the distal end of the saphenous artery in response to a stepwise increase of blood flow in the saphenous artery from 0.6 to 1.2 ml/min, followed by the return to its initial value, is shown in Fig. 2. During the first few seconds after a sharp increase in the blood flow the pressure drop increased just as sharply. However, it then began to fall sharply, to reach a new equilibrium value, which exceeded the original pressure drop by only 10-15%, although the blood flow still remained twice as high as initially. With a decrease in flow to the original value the pressure drop returned after the transition process to its initial value. These changes in pressure drop are evidently due to the fact that in response to an increase or decrease in the blood flow, comparatively slow dilatation or constriction of the arteries takes place respectively. After the increase in blood flow the pressure drop returned to a new steady-state value within about 20 sec, whereas after reduction of the blood flow it did so after 40 sec.

If the blood flow was increased from 0.1 to 1 ml/min quickly (in 3 sec) the pressure drop changed directly proportionally to the change in blood flow. This means that the response of dilatation of the arteries is comparatively slow, and during 3 sec the diameter of the vessels is unable to increase significantly. It is this increase in the pressure drop, proportional to blood flow, that is characteristic of a hydraulic system consisting of tubes with constant diameter. Dependence of the pressure drop on blood flow in the arteries would also be the same if they did not increase their diameter in response to the increase in blood flow. For each test with a rapid increase of flow the tangent of the angle of slope of the relationship between pressure drop and blood flow was determined. This value under the experimental conditions used ($M \pm m$, $n = 14$) was 36 ± 10 mm Hg/(ml·min).

If the blood flow was increased at a constant rate from 0.1 to 1.5 ml/min slowly (in the course of 600 sec) the reaction of dilatation of the arteries was able to develop, and the dependence of the pressure drop on blood flow became significantly nonlinear (the averaged curve of this dependence in Fig. 3). It will be clear from Fig. 3 that during a change in blood flow from 0.5 to 1.5 ml/min the change in the pressure drop was so small that it amounted virtually to stabilization. A straight line drawn with a gradient of 36 mm Hg(ml·min), corresponding to changes in the pressure drop during rapid changes in blood flow when the dilator response of the conducting arteries did not have a change to develop, is shown in Fig. 3 for comparison.

Thus, in rats, stabilization of the pressure drop from the arch of the aorta to the distal end of the saphenous artery takes place during a more than threefold change in blood flow. This stabilization is evidently based on the ability of the conducting arteries of rats to increase their diameter in response to an increase in blood flow.

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