BULK RIGIDITY OF CAPACITIVE VESSELS OF RATS IN LOCAL ARTERIAL HYPOTENSION

S. M. Shenderov and V. A. Saltykova

UDC 661.12-008.331.1-092.9-07:616.147.3-072.7

Experiments on rats showed that lowering of the arterial pressure in the vessels of the hind limb for up to three months is accompanied by a decrease in the bulk rigidity of the capacitive vessels of this region.

KEY WORDS: Local arterial hypotension; bulk rigidity of veins.

Previous investigations showed [1, 3, 4, 6] that a prolonged fall of arterial pressure in the vessels of the posterior part of the body in rats, caused by constriction of the abdominal aorta, leads to a progressive decrease in the hydraulic resistance of the resistive vessels of the limb. The state of the capacitive vessels has not so far been studied in chronic arterial hypotension.

The object of this investigation was to determine the bulk rigidity of the limb veins in rats after the creation of regional arterial hypotension for periods of 30 and 90 days.

EXPERIMENTAL METHOD

Regional arterial hypotension was induced by applying a nichrome coil to the abdominal aorta below the orifices of the renal arteries [2], as a result of which the pressure in the distal part of the aorta was lowered by 30-45%.

Experiments were carried out on 38 male albino rats weighing 250-480 g. Group 1 (control) consisted of 9 rats, group 2 included 10 rats with regional hypotension lasting 30-35 days, group 3 contains 10 rats with regional hypotension lasting 90 days, and group 4 consisted of 9 rats undergoing a mock operation (application of the nichrome coil to the abdominal aorta but with the turns sufficiently wide so as not to reduce the lumen of the vessel).

Acute experiments were performed under urethane anesthesia (0.7 ± 0.2 g/kg, intravenously) 30-35 and 90 days after constriction of the aorta or 90 days after the mock operation.

The bulk rigidity of the veins was characterized by the index E_{15} [8], the ratio between the increase in pressure (ΔP) and the increase in volume of the veins (ΔV) at a venous pressure of 15 mm Hg, and it was expressed in cm water/ml/100 g weight of the limb. A catheter was introduced into the left femoral artery in the distal direction, and by means of a controlled output roller pump the limb vessels were perfused with blood entering the pump from the carotid artery. The skin and all the muscles were burned through with the thermocautery at the level of the boundary between the upper and middle thirds of the thigh and the femoral and sciatic nerves were divided. The limb temperature was kept at 36°C. Periodically (at intervals of 5-10 min), the outflow of blood via the femoral vein was blocked and the increase in pressure (ΔP) distally to the site of occlusion was measured with an electromanometer. Knowing the output of the pump (which could be altered in steps from 0.5 to 2.1 ml/min) the increase in volume of the capacitive vessels (ΔV) was calculated for time steps of 0.5-1 sec.

Laboratory of Regulation and Biophysics of the Circulation, Institute of General Pathology and Pathophysiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. M. Chernukh.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 80, No. 9, pp. 18-21, September, 1975. Original article submitted November 1, 1974.

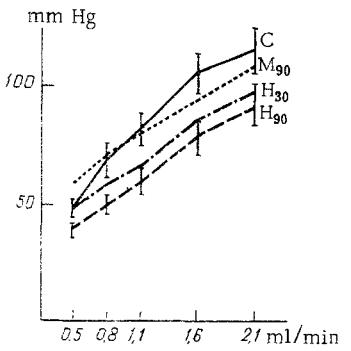

Fig. 1

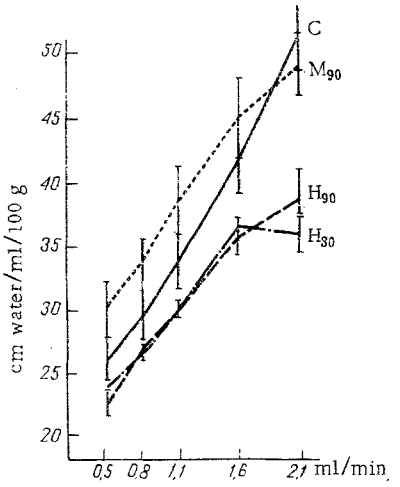

Fig. 2

Fig. 1. Perfusion pressure in control rats (C), rats undergoing mock operation (M_{90}), and rats with regional hypotension lasting 30 (H_{30}) and 90 (H_{90}) days. Abscissa, volume velocity of perfusion (in ml/min); ordinate, perfusion pressure (in mm Hg). Values of M ± m are given.

Fig. 2. Bulk rigidity of veins in control rats (C) rats undergoing mock operation (M_{90}), and rats with regional hypotension lasting 30 (H_{30}) and 90 (H_{90}) days. Abscissa, volume velocity of perfusion (in ml/min); ordinate, bulk rigidity of veins (in cm water/ml/100 g weight of limb). Values of M ± m are given.

EXPERIMENTAL RESULTS AND DISCUSSION

In the control rats and rats undergoing the mock operation the pressure in the femoral artery was virtually indistinguishable from that in the corotid artery. In rats on whose aorta a constricting coil was applied the pressure in the carotid artery 30 days after the operation was 101 ± 2.5 mm Hg, and after 90 days it was 98 ± 2 mm Hg. The pressure in the femoral artery of these animals was 70 ± 3.50 and 54 ± 3 mm Hg respectively, i.e., it was lowered by 30 and 45% respectively.

The results illustrated in Fig. 1 show that prolonged regional hypotension was accompanied by a progressive decrease in the hydraulic resistance of the resistive vessels, which was particularly clearly marked 90 days after the fall of arterial pressure in the vessels in the hind part of the body. These results are in agreement with those obtained previously in the writers' laboratory [1, 4].

As the results given in Fig. 2 show, the bulk rigidity of the veins rises substantially with an increase in the rate of their filling with blood, as was observed previously in investigations on man [5, 7] and experiments on animals [3]. The increase in E_{15} was particularly marked in the control animals when the rate of filling of the veins with blood was high, i.e., the rate of deformation of their walls was high.

It also follows from the results in Fig. 2 that the bulk rigidity of veins in the limb in which the arterial pressure was kept low for a long time was clearly less than in animals with a normal arterial pressure in the limb vessels. This difference was particularly great when the rate of filling of the veins with blood was high. In the control animals an increase in output from 1.6 to 2.1 ml/min was accompanied by a sharp increase in the rigidity of the veins, whereas in the experimental rats the value of E_{15} was virtually unchanged under these circumstances.

That the decrease in bulk rigidity of the veins was in fact caused by the arterial hypotension is clear from the results of measurement of E_{15} in the animals undergoing the mock operation. In these animals this index not only was not reduced, but it actually showed a tendency to rise, although the differences from values obtained in experiments on the control animals were not statistically significant ($P > 0.05$).

It is difficult at present to reach definite conclusions regarding the causes of the fall in bulk rigidity of the veins in a region of arterial hypotension. By analogy with the increase in extensibility of resistive

vessels during a prolonged fall of arterial pressure it might be postulated that the decrease in bulk rigidity (and, correspondingly, the increase in extensibility) of the capacitive vessels in the region of arterial hypotension was due to a lowering of the venous pressure and a decrease in the stretching forces acting on the walls of the veins. Measurement of the lateral pressure in the femoral vein of rats with regional hypotension in fact yielded somewhat lower values than in the control animals. However, these differences were small. It is also evident that the level of the venous pressure in the main veins does not necessarily reflect the hydrodynamic conditions in the smaller capacitive vessels of the limb, the state of which mainly determines the value of E_{15}. A more definite conclusion regarding the cause of this phenomenon could therefore be proposed if the pressure could be measured in the small veins of the limb. The writers intend to carry out such an investigation in the near future.

LITERATURE CITED

1. O. Ya. Kaufman, A. G. Zakharov, A. N. Rogoza, et al., Byull. Éksperim. Biol. i Med., No. 12, 35 (1972).
2. A. Kh. Kogan, Byull. Éksperim. Biol. i Med., No. 1, 112 (1961).
3. V. A. Saltykova and S. M. Shenderov, Byull. Éksperim. Biol. i Med., No. 10, 7 (1974).
4. S. M. Shenderov, A. G. Zakharov, A. N. Rogoza, et al., in: Proceedings of the First All-Union Conference on the Microcirculation [in Russian], Moscow (1972), p. 143.
5. M. Echt, L. Lange, and H. L. Thron, Z. Kreisl.-Forsch., 61, 842 (1972).
6. B. Folkow, M. Gurevich, et al., Acta Physiol. Scand., 83, 532 (1971).
7. N. K. Hollenberg and L. O. Breuss, Canad. J. Physiol. Pharmacol., 50, 310 (1972).
8. L. Lange, M. Echt, K. Kirsch., et al., Pflüg. Arch. ges. Physiol., 330, 111 (1971).