0.01), which fell 1 h after decompression by 46.4 ± 0.9 mm Hg ($P < 0.01$) from its initial level. All animals developed ARF, as shown by a sudden and progressive reduction of glomerular filtration, the renal plasma flow, maximal tubular secretion, minute diuresis, and tubular reabsorption. All this was accompanied by oliguria or anuria. All the control animals died from progressive ARF: five dogs after 2 days, three after 3 days, and two after 4 days. Fundamentally different results were obtained in the experimental animals. Of 10 experimental dogs three died 2-3 h after decompression from acute cardiovascular failure. Glomerular filtration and minute diuresis in the surviving animals exceeded the initial values, tubular reabsorption was within normal limits and the remal plasma flow and maximal tubular secretion were completely restored. All these phenomena were accompanied by rapid recovery of the 24-hourly diuresis. Of seven dogs in which ARF was successfully abolished, one died on the 4th day from thromboembolism of the pulmonary artery and one on the 6th day from hemorrhage caused by erosion of the femoral artery due to wound suppuration.

All these data provide experimental confirmation of the value of PGE_2 (prostenon) for clinical treatment of patients with ARF.

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REACTIVITY OF MESENTERIC MICROVESSELS IN RATS WITH MESENTERIC SHOCK

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In the opinion of many authorities, a breakdown of circulatory mechanisms plays an essential role in the pathogenesis of shock of varied etiology [2, 3, 5]. In particular, disturbance of regulation of vascular tone is one cause of the appearance of resistance of hypotension in the late stages of development of shock. The nature of this resistance has not been finally settled. Resistance of hypotension to injection of exogenous adrenalin and of other vasopressor agents likewise is not understood. Some light on the relations of hemorrhagic shock and posthemorrhagic hypotension has been shed by the investigations of Matvienko [1], who showed that in the late stages of acute blood loss decreased reactivity of the mesenteric microvessels to adrenalin is observed.

The object of this investigation was to study changes in sensitivity of the mesenteric microvessels to adrenalin on a model of mesenteric shock.

EXPERIMENTAL METHOD

Experiments were carried out on noninbred male rats weighing 250 ± 10 g, anesthesized with pentobarbital in a dose of 3.5 mg/100 g. The microcirculation was observed by the method in [4]. Reactivity of the microvessels was estimated by determining threshold concentrations of adrenalin applied locally in a volume of 0.1 ml [6]. The minimal concentration of adrenalin, application of which caused distinct changes in the velocity of the blood flow or contraction of metarterioles was taken as the threshold. Mesenteric shock was produced through a mid-

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line incision in the abdominal wall with application of a ligature of elastic material to the superior mesenteric artery. A catheter connected to a mercury manometer was introduced into the carotid artery to record systemic arterial pressure. Before the ligature was tied the initial state of the microcirculation was studied and background sensitivity of the microvessels to adrenalin determined. The ligature was removed after 2 h. All the animals developed mesenteric shock between 30 min and 1 h after removal of the ligature, and their blood pressure fell to 50-40 mm Hg. Reactivity of the microvessels was subsequently determined 10-15 min and 2 h after removal of the ligature ($M \pm m$).

EXPERIMENTAL RESULTS

Changes in sensitivity of the microvessels to adrenalin during mesenteric shock induced in rats by acute occlusion of the superior mesenteric artery were biphasic in character.

Immediately after removal of the ligature a considerable increase in reactivity of the microvessels was observed, as shown by lowering of the threshold adrenalin concentration in the solution for application from 3.5 ± 0.3 μg/liter in the initial state to 2.0 ± 0.2 μg/liter 10 min after removal of the ligatures ($P < 0.001$). It can thus be tentatively suggested that an important role in the development of initial vasoconstriction associated with hypotension is played by increased sensitivity of the microvessels, whose functional state was hitherto undisturbed, to adrenalin.

The first phase of change of reactivity of the microvessels was followed by a second phase, observed 60-120 min after removal of the ligature, and accompanied by a decrease in reactivity of the microvessels. The threshold concentration of adrenalin in the solution was increased under these circumstances to 7.5 ± 0.4 μg/liter ($P < 0.001$). This phenomenon is evidence that the persistent vasodilatation arising in the late stages of development of shock is due not only to a fall in body levels of adrenalin, but also to reduced sensitivity of the microvessels to it.

These results are in good agreement with those obtained by Matvienko [1] on a model of acute blood loss; it can therefore be postulated that changes in sensitivity of the microvessels to adrenalin (and also, perhaps, to other vasoactive substances) are a universal step in the disturbance of regulation of vascular tone in shock of varied etiology. The increase in sensitivity of the vessels to adrenalin at the beginning of shock plays a compensatory role in maintenance of the arterial pressure at a subnormal level. However, besides its beneficial effect, the widespread vascular spasm also has its negative consequences: Predominance of the extracapillary blood flow leads to the development of tissue hypoxia and acidosis, and in turn, this contributes to accumulation of metabolic products and to a decrease in sensitivity of the microvessels to vasoactive substances. The latter is also one cause of the development of decompensation and the transition from vasoconstriction to persistent vasodilatation.

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