VASCULAR RESISTANCE IN SKELETAL MUSCLE AND SMALL INTESTINE DURING HYPERCAPNIA FOLLOWING BLOCKADE OF BULBAR VENTRAL SURFACE STRUCTURES

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The key role of structures of the ventral part of the medulla has been demonstrated not only in maintaining the systemic blood pressure level, but also in controlling vascular resistance and the intravascular capacity of the skeletal muscles and intestine under conditions of normoxia and normocapnia. There is evidence in the literature of the high sensitivity of superficial structures in the ventral medulla to changes in the partial pressure of carbon dioxide in the CSF and in the intercellular fluid of the brain.

The aim of this investigation was to study the importance of the contribution of chemosensitive structures of the brain stem in the formation of changes in vascular resistance in the gastrocnemius muscle and small intestine under conditions of hypercapnia.

EXPERIMENTAL METHOD

Experiments were carried out on 27 cats weighing 1.9-2.8 g, anesthetized with urethane (1.1 g/kg, intravenously), with artificial ventilation of the lungs and an open chest. The frequency of artificial respiration was 20-25 cycles/min; the tidal volume was established in accordance with the arterial blood gas composition (in the femoral artery), which was determined by means of a BMS 3 MK2 gas microanalyzer ("Radiometer"). The medulla was exposed on the ventral aspect by an incision through the layers of soft tissues, the ventral part of the basilar and temporal bones was removed, and the dura and arachnoid mater were divided. The vascular bed of the hemodynamically isolated gastrocnemius muscle and small intestine was perfused with the animal's own blood (37.5°C) by means of a constant delivery pump. The capillary hydrostatic pressure was measured by the method described previously [1]. The perfusion pressure in the regions studied was measured with mechanotron transducers of an electromanometer; the venous outflow was determined as the change in blood level in the extracorporeal venous reservoir. The gas composition of the expired air was determined by the MKh-6202 mass-spectrograph, the capillary tube of which was inserted into the tracheotomy tube. To block central chemosensitive structures [6, 7] filter papers soaked with 2% procaine, with an area of 12 mm^2, were applied bilaterally to the rostral part of the ventral surface of the medulla 1-3 mm caudally to the trapezoid bodies. The significance of mean values was determined by Student's t test.

EXPERIMENTAL RESULTS

The arterial blood gas composition 30-40 min after exposure of the ventral medulla and the beginning of artificial perfusion of the blood vessels of the gastrocnemius muscle and small intestine, was: pO_2 = 102.3 ± 5.1 mm Hg; pCO_2 = 28.2 ± 1.7 mm Hg (pH = 7.360 ± 0.020).

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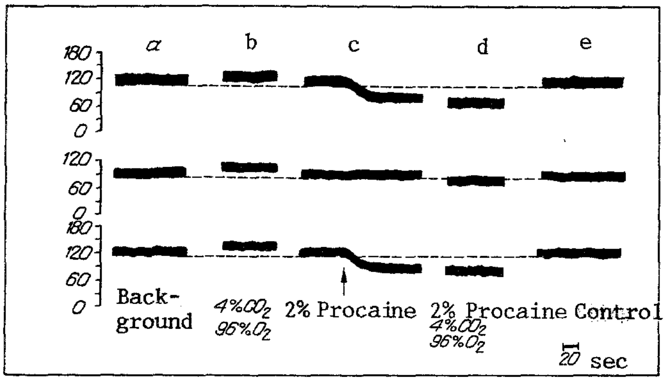

Fig. 1. Changes in systemic BP and perfusion pressure in vessels of gastrocnemius muscle and small intestine under hypercapnic conditions and with application of procaine to rostral part of ventral surface of medulla. From top to bottom: systemic BP (mm Hg); perfusion pressure in vessels of small intestine (mm Hg); perfusion pressure in vessels of gastrocnemius muscle (mm Hg). a) background (40 min after beginning of artificial perfusion of vascular bed); b) 5th minute of inhalation of gas mixture containing 4% CO_2 and 96% O_2 by animal; c) bilateral application of 2% procaine to rostral part of ventral surface of medulla (interval between b and c is 30 min); d) combination of procaine application to ventral surface of medulla with inhalation of gas mixture containing 4% CO_2 and 96% O_2 for 5 min (interval between c and d is 5 min); e) 10 min after removal of procaine from surface of brain stem and returning animals to inhalation of atmospheric air. Arrow indicates beginning of application of 2% procaine to rostral part of ventral surface of medulla.

At the end of the 5th minute of inhalation of a gas mixture containing 4% CO_2 and 96% O_2 (pO_2 = 330 ± 14 mm Hg; pCO_2 = 41.7 ± 1.5 mm Hg; pH = 7.240 ± 0.020), the systemic blood pressure and perfusion pressure in the vessels of the gastrocnemius muscle and small intestine were observed to rise, by 10.4 ± 3.2%, 10.5 ± 2.8%, and 13.2 ± 1.5% respectively. The outflow of venous blood from vessels of the gastrocnemius muscle and small intestine was increased by 3.1 ± 1.6% and 2.5 ± 0.8% of the total blood volume contained in the vascular bed of each preparation respectively. Thus during inhalation of a gas mixture containing 4% CO_2 and 96% O_2 the precapillary resistance in the skeletal muscles and intestine was increased, but the intravascular capacity of the regions studied was reduced, i.e., changes in the parameters of the resistive and capacitive functions of the vessels of the gastrocnemius muscle and small intestine during hypercapnia correspond qualitatively to changes in the analogous parameters during electrical stimulation of structures in the rostral part of the ventral medulla [2, 4]. Under hypercapnic conditions the mean capillary hydrostatic pressure fell in the skeletal muscles and rose in the intestine (Fig. 2b), and this led to an increase in the absorption of fluid from the interstitial space into the microvascular bed in the gastrocnemius muscle and to intensification of the transcapillary filtration of fluid in the intestine.

After bilateral application of procaine to the rostral part of the ventral surface of the medulla (it was noted in [6] that during short-term application of 2% procaine to the brain stem it can penetrate to a depth of not more than 100 μ) a marked fall of the systemic arterial pressure (BP) and perfusion pressure in the skeletal muscles (Fig. 1c) and a small decrease in the capillary hydrostatic pressure in the test preparations (Fig. 2c) were observed, whereas the perfusion pressure in the vessels of the small intestine remained virtually unchanged (Fig. 1c). Five minutes after procaine blocking of chemosensitive structures of the ventral surface of the medulla the animals were transferred to inhalation of a gas mixture containing 4% CO_2 and 96% O_2 and at the end of the 5th minute of inhalation, reduction of the systemic BP and perfusion pressure in vessels of the gastrocnemius muscle and small intestine by 9.2 ± 3.8%, 3.3 ± 3.0%, and 9.0 ± 2.4% was observed compared with the level of the hemodynamic parameters established after procaine application to the brain. No significant differences were found between the capillary hydrostatic pressure in a series of observations illustrated in

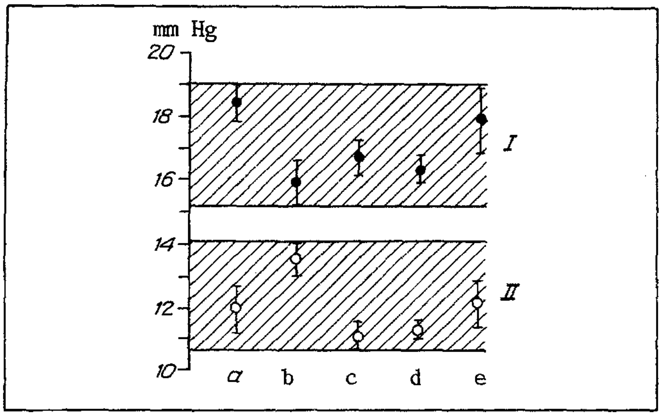

Fig. 2. Changes in mean capillary hydrostatic pressure in gastrocnemius muscle (I) and small intestine (II) under hypercapnic conditions and after application of procaine to rostral part of ventral surface of medulla. Legend as to Fig. 1.

Fig. 2c and d. After combined application of cocaine to the brain and inhalation of the hypercapnic gas mixture, the outflow of blood from the venous bed of the gastrocnemius muscle and small intestine was reduced by 0.6 ± 0.3% and 3.1 ± 0.8% respectively of the total volume of blood contained in each of these preparations.

The results are evidence that before blockade of the chemosensitive structures in the rostral part of the ventral medulla, during inhalation of a gas mixture containing 4% CO_2 and 96% O_2, the resistance of the resistive vessels was increased in the skeletal muscles and intestine, but the intravascular capacity was reduced, i.e., the vasoconstrictor effect was stronger than the vasodilator effect. We know [5] that an excess of oxygen in the inspired gas mixture blocks vascular chemoreceptors. Taking this information into account, and also our observations showing that after procaine application to chemosensitive structures of the ventral surface of the medulla, associated with blockade of vascular chemoreceptors and inhalation of a hypercapnic gas mixture by the animals, a decrease in the resistance of the resistive vessels and an increase in the intravascular capacity are observed in the gastrocnemius muscle and small intestine, it can be tentatively suggested that predominance of the direct vasodilator effect of CO_2 over its reflex vasoconstrictor effect takes place under these conditions. Thus the hemodynamic effect of CO_2 depends on the relationship of its direct action on the wall of arteries and veins and its reflex effect not only through vascular chemoreceptors, but also through the chemosensitive structures of the ventral surface of the medulla.

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