Myogenic Tone and Neurogenic Vasoconstriction in Microcirculatory Bed of Rat Cerebral Cortex

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The reaction of rat parietal cortical vessels to stimulation of the sympathetic ganglion was studied by intravital microscopy. Stimulation induced constriction of cerebral arterioles. Clamping of one or both carotid arteries led to myogenic vasodilatation in the majority of cases. Constrictive response of brain arterioles to ganglion stimulation under conditions carotid occlusion did not differ from that under conditions of baseline cerebral blood supply.

Key Words: *cerebral arterioles; myogenic vasodilatation; neurogenic vasoconstriction; microcirculation*

Stimulation of the superior sympathetic ganglion (SG) in rabbits reduces bloodflow in some brain areas, which is recorded by laser Doppler techniques [4,5]. A similar effect was previously revealed in rats; however under conditions of decreased blood supply caused by ligation of the middle cerebral artery or carotid arteries (CA), this stimulation of SG increased, but not decreased the bloodflow on the surface of the rat cerebral cortex [2].

Presumably reduced myogenic tone of cerebrocortical arterioles in the presence of decreased intravascular pressure modified the vasoconstrictive effect of SG stimulation. This determines new relationship between vascular conduction in the microcirculatory system and distribution of the bloodflow. In this study we verified this hypothesis.

MATERIALS AND METHODS

Experiments were carried out on 12 male albino rats (250-300 g) narcotized with sodium ethaminal (5 mg/100 g intraperitoneally) and then 2-3 mg/100 g sodium ethaminal. Spontaneous respiration was maintained through tracheostoma. The left upper SG was exposed and a bipolar stimulating electrode was implanted into it; the electrode consisted of two 0.2-mm nichrome wires twisted together with bent ends free from insulation on the side adhering to the ganglion. The electrode was fixed on the ganglion and adjacent tissue with MK-7 surgical glue. The ganglion was stimulated with a DISA-multistim stimulator by a series of rectangular current pulses (0.5-0.7 msec, 40 pulse/sec, 8-10 V, 20-30 sec stimulation duration, interval between stimulations at least 3 min). The sympathetic tract below the ganglion was not crossed. For blood pressure (BP) recording, a polyethylene catheter filled with heparin solution was inserted either into the left femoral artery or into external CA. One or both common CA were occluded by tightening the ligature loops under the common CA.

Arterioles of the parietal cortex of the first, second, and third orders of branching were examined under a microscope through a 4×4-mm hole in the skull. The pia mater was removed and the brain was covered with a transparent film preventing its drying and swelling. The brain was illuminated with a DRSh-250-33 mercury lamp. The examination was carried out in reflected light with warmed ×20 and ×10 epiobjectives. The device for measuring the external diameter of microvessels by image-splitting technique [1] was attached to MBI-15 microscope.

The significance of differences was evaluated using Student's paired t test at 0.05 level of significance.

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RESULTS

The mean BP at the beginning of each experiment was 149±25.4 mm Hg (*M*±*SD*). During stimulation of the superior cervical SG BP in the femoral artery was stable or changed negligibly (-1.7±1.1%, $p>0.05$). On the other hand, preliminary experiments showed that stimulation of the ganglion always caused a pressor reaction recorded in the external CA. Arterioles in the cerebral cortex constricted in the majority of cases in response to stimulation of the ganglion. The diameter of arterioles decreased by 10.9±1.1% (*M*±*m*, $n=61$, $SD=9.1$). In one case stimulation induced vasodilatation and in 11 no vascular response was recorded. The latency of reaction was 13.3±5.3 sec (*M*±*SD*).

Standard deviation of the value of vasoconstrictive reaction of arterioles was rather high, which suggests a certain gradient of responses of arterioles of different diameter to the sympathetic stimulus. However analysis of correlations showed no relationship of this kind (Fig. 1).

Reactions of 34 arterioles to SG stimulation were studied in experiments with complete CA occlusion. Fluctuations of BP in the femoral artery during occlusion of one or two CA were irregular, and the changes were insignificant (2.5±3.3 mm Hg, *M*±*m*, $p>0.05$). CA occlusion on one or both sides changed the diameter of arterioles in the majority of cases (Fig. 2). Left-sided or bilateral CA occlusion slowed the capillary bloodflow.

Dilatation of arterioles in response to CA occlusion was analyzed separately. Unilateral ipsi- or contralateral occlusion led to nasodilatation by 6 and 8% of the initial diameter, respectively, while bilateral occlusion led to 18.5±4.2% dilatation.

Sympathetic ganglion was stimulated 1 min after CA occlusion during maximal and stable dilatation of arterioles. Similarly to normal perfusion, stimulation under conditions of decreased blood supply resultant from CA occlusion led to constriction of brain arterioles. The diameters of only 4 vessels did not change in response to stimulation. Bloodflow velocity did not decrease, but even increased, like in previous experiments, in which bloodflow velocity was recorded by a laser Doppler technique [2]. The mean vessel constriction in this group was 9.6±1% of the initial diameter, which did not differ significantly from constriction of the same vessels under conditions of the baseline perfusion pressure (10.4±1.2%, *M*±*m*, $p>0.05$). According to the reaction to CA occlusion, the vessels were divided into 3 groups: dilating arterioles (myogenic reaction to decreased intravascular pressure, $n=16$), constrictive arterioles ($n=10$), and arterioles whose diameter did not change ($n=8$). Paired comparison of the effects of SG stimulation in 3 groups of vessels showed that the reaction to SG stimulation in ischemia did not differ significantly from the reaction of the same vessels during normal blood supply (Fig. 2).

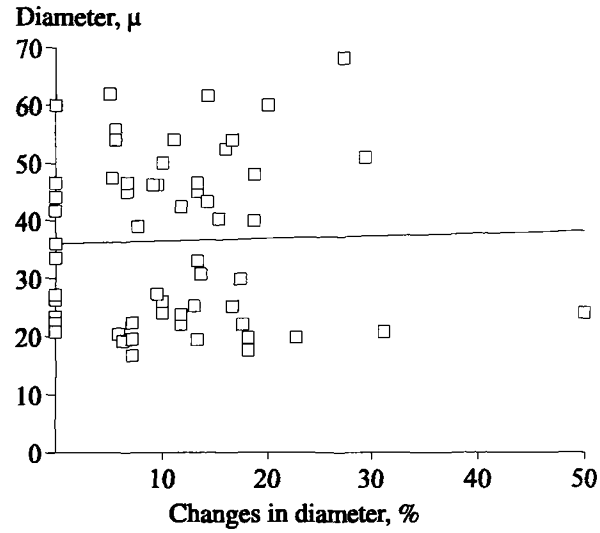

Fig. 1. Correlation between relative changes in the diameter of arterioles in response to stimulation of the ganglion and in the initial diameter ($r=0.016$, $p>0.05$).

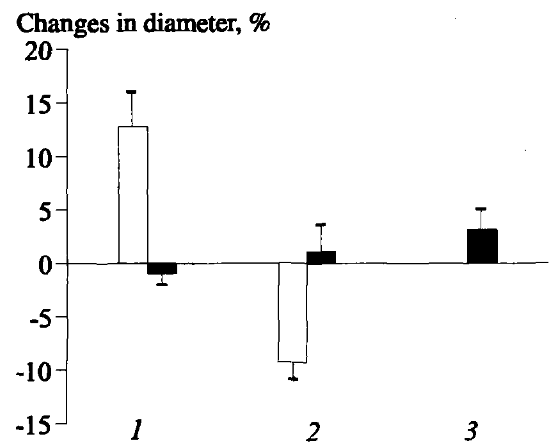

Fig. 2. Relative changes in arteriole diameter in response to occlusion of carotid arteries (light bars) and stimulation of the ganglion under conditions of carotid occlusion (dark bars). *1*) diameter of vessels; *2*) constricting vessels; *3*) vessels with unchanged diameter. Differences in the reactions of arterioles to stimulation during normal perfusion and after carotid occlusion are insignificant.

Hence, in contrast to capillaries of the rat skeletal muscle, studied before in similar experiments [3], the myogenic dilatation of the rat brain arterioles does not block (and does not potentiate) their constrictive response to stimulation of vasoconstrictive pathways.

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