CAPACITIVE FUNCTION OF THE SPLEEN DURING ELECTRICAL STIMULATION OF THE VENTRAL MEDULLA OBLONGATA

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The spleen is one of the organs performing the role of blood depot in the body [5]. In the case both of reflex influences on the spleen and direct stimulation of its sympathetic nerves, the spleen has been shown to have a significant effect on the circulating blood volume [2, 5, 7]. The leading role in the maintenance of vasomotor tone and also in the manifestation of baroreceptor and chemoreceptor vascular reflexes has been shown [4] to be played by structures of the ventral portion of the medulla oblongata (VM). However, no information can be found in the literature on the effect of structures of VM on the capacitive function of organs involved in regulation of the venous return of blood to the heart.

The aim of this investigation was to study the character, magnitude, and rate of development of responses of the acccumulative vessels of the spleen to electrical stimulation of structures of VM.

EXPERIMENTAL METHOD

Experiments were carried out on 20 male and female cats weighing 1.8-3.6 kg, anesthetized with urethane (1 g/kg body weight, intravenously), subjected to thoracotomy and artificial respiration with the "Vita" apparatus, and receiving heparin (1000 U/kg). The parameters of respiration were chosen by means of a BMs3, Mark 2 gas analyzer ("Radiometer," Denmark). The animal's head was fixed in a stereotaxic apparatus. Access to the test structures was obtained from the vental surface of the medulla. The middle of the roots of the hypoglossal nerves (the XII pair of cranial nerves) was taken as the reference level. Electrodes were inserted into the brain rostrally and caudally to this level by 2 mm (for brevity of description these points will be called "+2" and "−2" respectively) with a step manipulator (MSh-8) at a depth of 1500 μ from the ventral surface [3]. The electrodes were made of nichrome wire 100 μ in diameter, coated with fluorine plastic, and with an area of their active surface of 300 μ^2. Nerve tissue structures were stimulated with square pulses with a constant frequency of 50 Hz and duration of 1 msec, with two values of current strength: threshold and twice the threshold level. The threshold strength of current was defined as the minimal current causing a shift of perfusion pressure in the vascular region tested, and it varied in different animals from 5 to 50 μA, in agreement with data in the literature [3]. Perfusion of the hemodynamically isolated spleen was carried out in the experiments with the aid of a constant delivery pump. The resistance and capacity of the vessels were studied by methods of resistography and accumulography. All the parameters studied (arterial, perfusion, and venous pressure, venous outflow) were recorded by means of manometers on mechanotron transducers, and recorded on an N30131 automatic writer. Venous drainage took place at a pressure of 10 mm Hg in the splenic vein, for it has been shown [1] that the possibility of development of passive-elastic rebound of the veins is reduced at this venous pressure level. Preservation of the integrity of innervation of the splenic vessels was verified by the response of the perfusion pressure and venous drainage to the pressor carotid sinus reflex. The results were subjected to statistical analysis by Student's t test.

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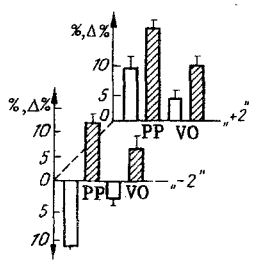

Fig. 1

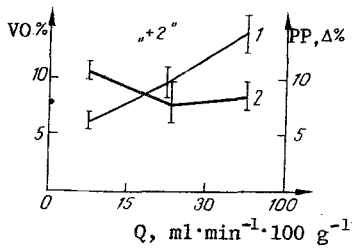

Fig. 2

Fig. 1. Changes in perfusion pressure (PP) and venous outflow (VO) in splenic vessels in response to stimulation of VM. Unshaded columns — increase in responses to stimulation of neuronal zones by current of threshold strength, shaded columns — increase of responses to stimulation of these structures by current of twice the threshold strength. Numbers on right: +2) rostral pressor zone of VM, −2) caudal depressor zone of same portion of brian stem. Ordinate, mean values of perfusion pressure (in % of initial level) and of venous outflow (in % of volume of blood contained in splenic vessels) to stimulation of above brain structures.

Fig. 2. Dependence of venous outflow (VO) and perfusion pressure (PP) on blood flow in spleen. 1) Venous outflow (in % of blood volume retained in splenic vessels), 2) change in perfusion pressure (in % of initial level). +2) Rostral pressure zone of VM.

EXPERIMENTAL RESULTS

The blood flow in the splenic vessels in these experiments was 23.7 ± 4.2 ml · min^{-1}/100 g tissue, with a mean weight of the spleen of 16.2 ± 1.9 g, in agreement with data in the literature [6]. The initial arterial blood pressure of the animals was 124 ± 3.4 mm Hg. The perfusion pressure in the region studied in these experiments averaged 121 ± 1.3 mm Hg.

Stimulation of brain structures in the rostral pressor "+2" zone by current of threshold strength caused an increase of venous outflow by 3.6 ± 0.8% of the volume of blood retained in the splenic vessels (Fig. 1). The latent period and time of recording of the peak values of these responses were 6.3 ± 1.0 and 32.8 ± 2.7 sec respectively. With an increase in current strength to twice the threshold level there was a greater increase in the venous outflow, to 10.1 ± 1.1% of the volume of blood retained in the splenic vessels. An increase in the rate of development of the response of the accumulation vessels under these circumstances also was noted: the latent period of changes in the venous outflow was reduced to 4.3 ± 0.4 sec and the time of the peak response was virtually unchanged at 27.9 ± 3.1 sec. Stimulation of structures of this "+2" pressor zone by currents of threshold and above threshold strength led to an increase of perfusion pressure in the splenic vessels by 9.1 ± 1.3 and 16.1 ± 1.7% of the initial value respectively. The latent periods of changes in perfusion pressure during stimulation of the medullary structures by currents of two different strengths were 3.1 ± 0.4 and 2.2 ± 0.1 sec, and the time of appearance of peak vasomotor responses was 14.1 ± 0.3 and 12.9 ± 0.2 sec respectively. Thus the experiments revealed a direct relationship between the increase in the venous return and the perfusion pressure in the splenic vessels, on the one hand, and the strength of electrical stimulation of brain structures at the "+2" point on the other hand. The greatest increase in venous outflow was found when the strength of the current was twice the threshold value, and it amounted on average to 10.1% of the total blood volume (32 ml/100 g [1]) in the spleen.

Since considerable differences in the initial blood flow in the splenic vessels were found in these experiments an attempt was made to determine how the vasomotor responses depended on the initial blood flow in the spleen. The results of the investigations were divided into three groups, in which the blood flow ranged from 0 to 15, from 15 to 30, and from 30 to 100 ml · min^{-1}/100 g tissue. The increase in the venous outflow in the splenic vessels in response to stimulation of the "+2" pressor

zone of VM under these circumstances amounted to 6.1 ± 0.6, 9.6 ± 1.3, and 13.9 ± 1.6% respectively ($p < 0.05$). The increase in perfusion pressure in the splenic vessels was 10.6 ± 0.7, 7.8 ± 1.7, and 8.5 ± 1.2% respectively. These results indicate that the degree of filling of the spleen with blood has a marked influence on its release in response to electrical stimulation of the rostral zone of VM. No significant difference was observed in the change in perfusion pressure under these circumstances (Fig. 2).

In response to stimulation of the "−2" caudal zone of VM with a current of threshold strength reduction of the venous outflow by 3.6 ± 0.8 compared with the volume of blood retained in the splenic vessels was observed (Fig. 1). The latent period and the time taken to reach the peak of the responses were 8.0 ± 0.7 and 35.0 ± 2.0 sec respectively. An increase in the strength of the stimulating current to twice the threshold value led in 85% of cases to an increase in the venous outflow by 5.5 ± 1.8%, in 5% it was unchanged, and in 10% it was reduced. The latent period and time of the peak of the responses in cases when the venous outflow was increased were 5.6 ± 0.3 and 32.0 ± 0.8 sec respectively. The perfusion pressure in the splenic vessels fell in response to stimulation of the caudal zone of VM at threshold strength on average by 12.8 ± 1.5% of the initial level, whereas if the strength of the current was increased to twice the threshold level, its character was reversed and the increase in the parameter amounted to 10.1 ± 1.3%. The temporal characteristics of the change in perfusion pressure in the splenic vessels in response to stimulation of brain structures at the "−2" point were virtually equal to the values of the corresponding parameters during stimulation of the "+2" pressor zone of VM. The investigations thus showed a decrease in values of the venous outflow and perfusion pressure in the splenic vessels during stimulation of the caudal zone by a current of threshold strength and their increase by stimulation at twice the threshold strength. Comparison of dependence of the venous outflow and perfusion pressure values on the initial blood flow in the spleen revealed no significant differences in experiments with stimulation of brain structures in the "−2" depressor zone of VM.

The results of these investigations are evidence that electrical stimulation of the structures of VM affects both the capacitive and the resistive functions of the spleen. The experiments showed that the venous outflow in this organ during stimulation of the "+2" rostral zone of VM, in the region of which the paragigantocellular nucleus lies, is directly dependent on the strength of the current used to stimulate the brain structures and on the blood flow in the spleen. The rate of development of responses of the accumulative vessels in this case also depended directly on the strength of the stimulating current. The more rapid development of responses of the accumulative vessels to stimulation of the pressor zone of VM compared with responses obtained to stimulation of the depressor zone can be explained by the existence of direct connections between this region and the sympathetic preganglionic neurons, whereas the caudal "−2" zone of VM is known to have no direct connections with the above-mentioned structures. Thus is can be concluded from the results of these investigations that structures of VM affect regulation of the vasomotor tone of the spleen and, in particular, they exert an action on the capacitive function of the spleen.

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