PHYSIOLOGY

Resistance and Capacitance Functions of Veins in the Gastrocnemius Muscle During Electrical Stimulation of Ventral Subdivisions of Medulla Oblongata

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Perfusion of blood vessels in the gastrocnemius muscle with autoblood under local electrostimulation of pressure zones of ventral part of the medulla oblongata revealed increments in resistance to blood flow in the muscle veins and arteries, which were respectively, 83 and 81% of the constrictor reactions of these vessels in response to a supramaximum electrostimulation of the sympathetic chains, while decrease in the venous blood filling was 33%. In response to electrostimulation of VMO depressor zones, the venous blood filling did not increase, while in the muscle arteries and veins the resistance to blood flow decreased by 60 and 130% in comparison with the dilator reactions of these vessels after dissection of sciatic nerve in the muscle preparation. Both quantitative and qualitative aspects of resistance and capacitance function of venous and resistive arterial vessels are shown to depend on individual features in the regulation of veins and arteries at the central and peripheral level.

Key Words: *ventral surface of medulla oblongata; venous resistance and capacitance function; gastrocnemius muscle*

According to modern views [4,7,9], ventral subdivisions of the medulla oblongata (VMO) contain populations of neurons, electrostimulation (ES) of which induces pressor or depressor shifts in systemic arterial pressure (AP) and the corresponding alterations in regional hemodynamics. There are no data in the literature on the effect of local ES of VMO on venous resistance to blood flow, which characterizes the resistance function of peripheral veins [6].

Our aim was to compare dynamic changes of venous resistance to blood flow (venous resistance function) with blood filling (capacitance function of veins) in feline gastrocnemius muscle under local ES of VMO pressor and depressor zones.

MATERIALS AND METHODS

Experiments were carried out under Urethane anesthesia (1 g/kg intravenously) on 14 heparinized (2000 u/kg intravenously) cats weighing 3-4 kg. The gastrocnemius muscle was isolated hemodynamically (shank preparation [2]). Its vessels were perfused with a constant volume of autoblood. Alterations in resistance to blood flow in the arterial bed were determined by the method of arterial resistography [8] and that in venous bed with the help of original

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method of venous resistography [3,11]. Changes in the venous blood filling were measured by the method of accumulography [6]. Operative access to VMO was performed from the ventral cervical surface. Electrical stimulation was carried out in a rectangular area of VMO with the boundaries located 8 mm rostral, 4 mm caudal, and 3 mm lateral to the site of the basilar artery intersection with the level of exit of the roots of hypoglossal nerves) from medulla oblongata [7]. The electrode was inserted to the depth of 1000-3000 μm. Electrical stimulation of pressor and depressor VMO points (in respect to alterations of AP) was carried out with the help of a glass-insulated Nichrome electrode of 100 μm in diameter using current-stabilized unipolar rectangular pulses (50-200 μA, 0.5 msec, 50 Hz) for the periods of 30 to 100 sec. It was discontinued as soon as the maximum change in systemic AP had been recorded. At the end of experiment, electrical stimulation of lumbar sympathetic chains was performed at the level of L_{IV}-L_V by rectangular pulses (6 V, 5 msec, 30 Hz), then the muscle preparation was decentralized by cutting the sciatic nerve to discontinue the vascular neurogenic tone. The results were statistically analyzed using Student's *t* test.

RESULTS

Electrical stimulation of VMO pressor and depressor points evoked, respectively, constriction and dilation of veins and arteries in the gastrocnemius muscle. They were observed as an increment or decrement of venous and arterial resistance to blood flow in the gastrocnemius muscle (Table 1). Stimulation of VMO pressor points led to a decrease in the filling of the muscle veins by 0.06±0.03 ml, while ES of the depressor points did not affect this parameter. The lack of changes in blood filling of veins in the gastrocnemius muscle during ES of VMO depressor points was an unexpected phenomenon, because ES was accompanied by a decrease in venous resistance to the constant blood flow (venous resistance function), which under the conditions of resistography method [6] can be explained by dilation of intramuscular veins due to relaxation of their smooth muscle (SM) contractile elements. The data show that no increment in blood filling of the muscle veins against the background of a decrease in venous resistance to blood flow could be attributed to relaxation of SM elements of the vascular walls. Presumably, this fact is a consequence of the differences in resistance and capacity functions of regional veins at the peripheral level. The distribution of SM-elements in the walls of veins in the feline hind limbs is characterized by a pronounced heterogeneity [1,6]. The uninterrupted layer of smooth muscle begins in the skeletal muscle veins greater than 200-300 μm in diameter [1], while in the veins of middle and large caliber there are "clusters" of SM-elements which look like a kind of "venous sphincters" located at the place of introitus of small veins into the greater ones and around venous valves [10]. On this basis one can suppose that the decrease in blood filling of the gastrocnemius muscle veins evoked by ES of VMO pressor zones was smaller due to accumulation of blood in the muscle-free (venules of less than 200 μm in diameter) and muscle-deficient fragments of venous vascular bed as a result of increment in the resistance to blood flow due to constriction of greater veins and "venous sphincters". Evidently, it may explain the absence of the expected increment in the muscle vein blood filling during ES of VMO depressor zones, be-

TABLE 1. Neurogenic Alterations of Arterial and Venous Resistance to Blood Flow and of Blood Filling in Feline Gastrocnemius Muscle (*M±m*)

Index	Constrictor reactions		Dilator reactions	
	electrostimulation of sympathetic chains	electrostimulation of VMO pressor points	cutting of sciatic nerve	electrostimulation of VMO depressor points
Changes in perfusion pressor, mm Hg				
in arteries	30.0±0.2	28.1±0.1 (81.4±14.3)	20.0±1.0	26.6±2.2 (124.3±12.1)
in veins	2.6±0.4	2.0±0.2 (83.2±6.1)	2.6±0.4	1.5±0.1 (60.2±17.3)
Changes in venous filling, ml	0.18±0.06	0.06±0.03 (33.1±6.4)	—	—

Note. Values in parenthesis are the percent of constrictor and dilator vascular reactions evoked from VMO, relative to similar reactions induced by electrical stimulation of sympathetic chains or by cutting the sciatic nerve, which were taken for 100%.

cause dilation of great veins and "venous sphincters" led to a decrease in venous resistance to blood flow, which caused the "passive" income of accumulated blood from the muscle-free fragments of venous vascular bed into the venous blood flow.

Therefore, alterations in blood filling in the veins of a skeletal muscle evoked by pressor or depressor ES depend on blood volume ratio in the muscle-free and muscle-deficient fragments of venous bed ("passive mechanism"), as well as on the modifications of blood volume in large veins with pronounced SM-layer (active mechanism). An important factor that affects the corresponding alterations in venous resistance to blood flow is the active mechanism of constriction or dilation of muscle veins with SM-elements in their walls. To evaluate the degree of alterations in resistance and blood filling of the muscle veins during ES of VMO, we compared the neurogenic constrictor reactions in veins and arteries evoked by local ES of VMO pressor zones with vascular reactions evoked by supramaximum [6] ES of lumbar sympathetic chains. The values of these reactions were taken for 100%.

Increments in the blood flow resistance caused by ES of VMO pressor zones in veins and arteries of the gastrocnemius muscle were, respectively, 83 and 81% of the constrictor reactions of these vessels evoked by ES of sympathetic chains. At the same time, the decrease in blood filling of veins in the muscle preparation in response to ES of VMO pressor zones was much lesser: about 33% relative to similar venous reactions evoked by ES of sympathetic chains. This fact is explained by the differences in central regulation of the resistance and capacitance function of veins in the gastrocnemius muscle, because this study compares decrements in venous blood filling evoked by both neurogenic effects at the same conditions of combined action of "passive" and "active" mechanism of regulation of venous capacitance function at the peripheral (effector) level.

The dilator responses of the gastrocnemius muscle veins and arteries evoked by local ES of VMO depressor zones were compared with analogous reactions according to the sign of reactions evoked after dissection of the sciatic nerve (neurogenic muscle decentralization); the values of these reactions were taken for 100%. In these experiments, electrical stimulation of VMO depressor zones provoked a greater decrease in arterial resistance to blood flow in the muscle that the decrease caused by dissection of sciatic nerve. Thus, relative to the arterial dilator reactions evoked by decentralization of the muscle preparation, the dilator arterial reactions caused by ES of VMO depressor zones were 130%. Similar value (the decrease in venous resistance to blood flow) for venous dilator reaction in the gastrocnemius muscle was 60%. The degree of arterial dilator responses evoked from VMO depressor zones was greater than that elicited after decentralization of the muscle vessels (i.e., after discontinuation of neurogenic tone) by 30%, being greater than analogous venous dilator responses by 2 times. It seems that electrical stimulation of VMO depressor zones caused active cholinergic relaxation of SM in the gastrocnemius muscle arteries in addition to inhibition of sympathetic pulse traffic from medulla oblongata [7]. This conclusion is based on the evidence [4,5] that the cholinergic nervous pathways originating in the hypothalamic "defensive zone" travel via the neuronal structures of VMO, so electrical stimulation of VMO depressor zones can elicit similar reactions in the skeletal muscle arteries as ES of the "defensive zones" in hypothalamus or amigdala [4]. The absence of cholinergic innervation in the skeletal muscle veins [5] explains the smaller values of venous dilator responses evoked by ES of VMO depressor zones in comparison with arterial reactions.

From these data one can conclude that quantitative differences in the resistance function of arterial and venous vessels as well as quantitative and qualitative differences of the resistance and capacitance functions of venous vessels observed in the skeletal muscle in response to electrical stimulation of neuronal structures of VMO result from combined effect of peripheral (effector) and central mechanisms.

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