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New Relationship between Electrical Characteristics of Evoked Contractions in Skeletal Muscles during Necrobiosis

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Evoked muscle contractions at different stages of necrobiosis were investigated by stimulating impedance myography in patients with vascular pathology and acute or chronic ischemic syndrome of the lower extremities. The threshold and maximum amplitudes and current strength for contraction responses were determined, and the dependence of evoked contractions on stimulating current was evaluated. Approximation of the obtained curves with the well-known force—time excitation curves demonstrated the relationship between excitability and contractility parameters depending on the stage of ischemic necrobiosis. This regularity is a more universal interrelation between muscle electrical characteristics. Electrical characteristics of evoked contractions depend on various factors influencing muscle structure and function.

Key words: *electrodiagnosis; force—time curve; electrical characteristics of evoked contractions; ischemic necrobiosis in muscles*

The force—time curve relating the amplitude and duration of the threshold electric pulse for stimulation of excitable structures is well described by a hyperbolic function [7]. In damaged muscles (in particular, in denervation-induced muscle degeneration) this curve is biased in the upper-right direction [4,5]. There is no evidence on other mechanisms of this phenomenon and their influence on contraction parameters in damaged muscles because of the absence of precise methods for *in vivo* recording of muscle contractions. In particular, the correlation between the force—time excitability parameters and characteristics of muscle contractions were not studied. Thus, it is interesting to compare muscle excitability and electrophysiological parameters of evoked contractions (EPEC) determined by stimulating impedance myography [1-3]. Our aim was to study the correlation between EPEC and force—time excitability parameters at different stages of muscle necrobiosis.

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MATERIALS AND METHODS

The study included 58 patients (49 males and 9 females) with acute and chronic ischemia syndromes associated with atherosclerotic lesions in the aorta and major arteries of the lower extremities, hospitalized for examination, therapeutic treatment, or angioplasty of occluded vessels, and urgent patients with acute ischemia of the lower extremities provoked by thrombosis and embolism in arteries or endovascular prosthesis. The degree of muscle injury corresponded to the 2nd-4th degree chronic muscle ischemia [7] and 3rd-4th degree acute ischemia of the lower extremities [8]. The control group included 5 male patients without vascular diseases. To evaluate the state of ischemic muscles we assessed general characteristics of evoked contractions (EC) in all patients irrespective of the degree of occlusion and the stage of the disease. Muscle excitability was determined in 13 points on the leg according to the standard electrodiagnostic scheme [6]. The baseline impedance, stimulating current, EC

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amplitude (deviation from the baseline impedance) in response to threshold and above-threshold pulses, and the maximum contraction responses (MCR) were recorded at each point. EC were recorded with an originally designed device for stimulating impedance myography. Electrical stimuli were delivered to the muscle via a needle electrode, and evoked contractions were evaluated by changes in muscle impedance. EC were recorded with an electrocardiograph (paper speed 5-50 mm/sec). The reference electrode was positioned under the knee with a rubber band and a gauze pad soaked with a conducting gel.

The calibration was 1 Ω per 10 mm vertical scale. The baseline impedance was measured on a special scale. The stimulating current was set manually or automatically. The pulse duration was 1 msec.

After mathematical analysis of the data, EC amplitude were plotted as a function of stimulating current strength. The slope of the obtained curves varied depending on the degree of muscle damage. We grouped the results according to the severity of 2nd-4th chronic ischemic syndrome (types 2-4 muscles, respectively). Patients with acute ischemic syndrome, in whom EC were completely absent comprised a group with the 4th degree ischemic syndrome. In most cases, these patients were subjected to amputation of the leg. Type 1 muscles were intact muscles of control individuals.

The data were statistically analyzed by Student's t test, χ^2 test, and correlation analysis.

RESULTS

A close relationship was revealed between the electrical characteristics of MCR (Table 1). A direct correlation was found between CR amplitude, the slope of the force—time curve, EC duration, the amplitude/duration ratio, and baseline muscle impedance. These parameters were inversely proportional to the stimulating current (hyperbolic dependence).

A significant decrease in the baseline impedance with increasing the severity of muscle necrobiosis (Fig. 1) attests to disturbed molecular structure of

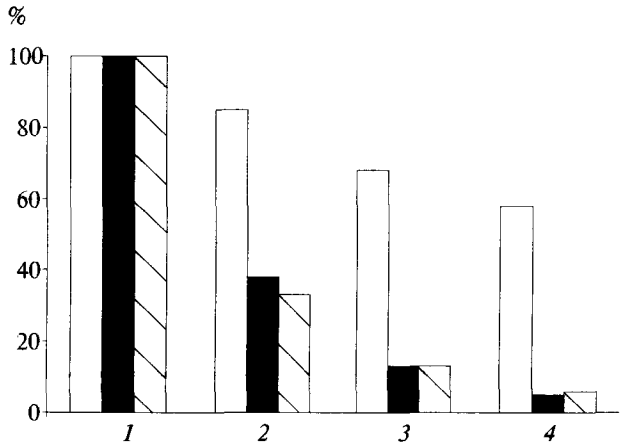

Fig. 1. Baseline impedance (open bars) and amplitudes of threshold (dark bars) and maximum (shaded bars) contraction responses in skeletal muscle during ischemic necrobiosis. *1*) control group, *2-4*) various stages of muscle necrobiosis.

electroexcitable membranes in muscle fibers (sarcolemmal and sarcotubular) which determine the muscle resistance at high-frequency current (2 kHz). A low input resistance impairs regenerative properties of muscle fibers, reduces their conductivity, and disturbs recruitment of new muscle fiber in muscle response to stronger stimuli. This is confirmed by the difference in EPEC between types 1 and 4 muscles. Thus, the slope of characteristic curve reflecting the number of recruited units per current unit decreased, which determined lower amplitude of MCR. Significant decrease in MCR amplitude and the amplitude/duration ratio, and shortening of MCR imply transformation of auxotonic EC into the local contractions.

The amplitude of MCR strictly depended on the threshold EC (Fig. 1). Microscopy showed that the weakest EC in frog muscles recorded by stimulating impedance myography corresponded to local contractions near the tip of the needle electrode.

The quality of local excitation process depended on the state of the muscle tissue. In severe damage (type 4 muscles), EC did not increase with increasing the strength of stimulating current and did not differ from minimum threshold contractions, which indicates

TABLE 1. Parameters of Maximum Evoked Contractions Depending on the Severity of Muscle Damage

Parameters	Muscle type			
	1	2	3	4
Stimulating current, mA	1.27	4.50	7.80	27.0
Amplitude (a), Ω	3.20	1.20	0.41	0.15
Slope of characteristic curve, °	85	61	31	4
Contraction duration (l), sec	0.416	0.380	0.311	0.271
a/l	7.70	3.16	1.32	0.55
Baseline R, W	360	300	243	209

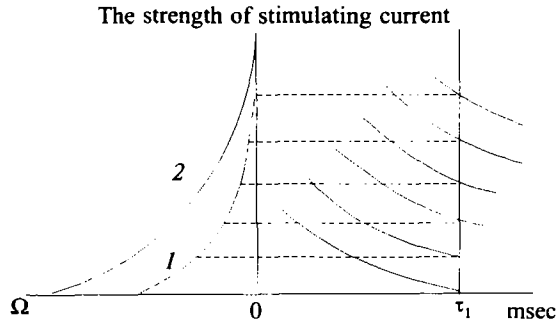

Fig. 2. Threshold and maximum contraction responses and force—time curves in the damaged muscle. Abscissa: left: contraction amplitude; right: duration of stimulating current (τ_{ST}). On the right: the upper-right bias of force—time curves in denervation-induced muscle degeneration. On the left, decrease in threshold (*1*) and maximum (*2*) contraction responses during ischemic muscle damage. Horizontal dashed lines point to the amplitudes of threshold contractions recorded by the stimulating impedance myography method (on the left) and by excitability studies at constant stimulus duration τ_1 (on the right).

impairment of local response transformation into propagating action potential and/or disturbances in electromechanical coupling.

Thus, a new relationship between excitability, recruitment of new muscle units, threshold EC and MCR amplitudes, the corresponding stimulating currents, and the degree of muscle necrobiosis was established.

The Weiss—Lapicque strength-duration model, which establish a relationship between the threshold stimulating current and stimulus duration, is the most close prototype of the new relationship established for EC. The force—time curve is well described by a hyperbolic function, which shifts in the upper-right direction during muscle necrosis [4,5].

The new relationship differs from the Weiss—Lapicque strength-duration model because it relates the amplitude of threshold EC to stimulating current at a constant stimulus duration. Although the corresponding curves are similar (hyperbolas), they differ in their origin and occupy different areas on the plot. The standard curve reflects the dependence of the excitability threshold on the strength of stimulating current and stimulus duration, while the new curves represent the dependence of EC amplitude on stimulating current at a constant stimulus duration. Both types of curves are the functions of stimulating current, and, therefore, have a common axis (Fig. 2). Comparison between the two fields on the plot indicate that the upper-right bias of the force—time curves in damaged muscles corresponds to a decrease in EC amplitudes recorded by stimulating impedance myography. Thus, the new relationship gives a more universal description of the muscle functional properties including contraction and excitation characteristics.

Many pathogenic factors affect the local excitation processes, recruitment of muscle units, the amplitude of threshold EC and MCR, and the value of threshold stimulating current. Ischemia, edema, denervation, and other factors, which affect muscle contractility, lead to EPEC deterioration. Our findings confirmed by the morphological studies suggest that decreased EPEC in muscle transplants can result from neuromuscular degeneration after denervation.

It should be noted that the new relationship is of practical importance. Stimulating impedance myography can be used for the diagnosis and prognosis of muscle diseases and assessment of muscle functional state after trauma of the extremity with pronounced edema, after neural and vascular trauma, in ischemic syndrome of the lower extremities, in muscle transplants, and in other cases, when conventional methods including classical EMG are ineffective.

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