INDEPENDENCE OF BLOOD SUPPLY OF A CONTRACTING MUSCLE OF THE FORCE DEVELOPED AND PREVENTION OF ITS SHORTENING

L. R. Manvelyan, V. M. Khayutin, and V. A. Khorunzhii

UDC 612.741.61

Experiments on cats have shown that the intensity of dilatation of blood vessels in the gastrocnemius muscle and the duration of recovery of their tone after contraction are independent of the degree of shortening of the muscle, of the force developed by it, and of the amount of physical work performed, and are determined entirely by the frequency of impulses in the motor fibers. The critical frequency of impulses at which the contracting muscle compresses its vessels is the same for isotonic and auxotonic contractions, and in most experiments it is about 16 pulses/sec.

The writers have previously shown that two patterns of blood supply are characteristic of auxotonic contractions of the cat gastrocnemius muscle, in which the muscle shortens initially by overcoming the slight mechanical resistance, but then, encountering an obstruction to further shortening, develops an isometric contraction [2, 4]. The first of these patterns consists of an increase in the blood flow during contractions, while the second consists of a decrease. Unhindered production of working hyperemia in the course of contraction can take place only if the frequency of the impulses does not exceed 8-16 pulses/sec. At a higher frequency the contracting fibers compress the muscle vessels. The muscle then works itself "into debt." This debt is "paid off" after contraction, in the period of postcontraction hyperemia, when the initial tone of the resistive vessels is restored.

Experiments have shown [2, 4] that the higher the frequency of muscle contraction, the slower this process takes place. Such a relationship can be regarded as a type of "memory," ensuring that the extent of the "debt" is commensurate with the time required to pay it off. From the standpoint of the classical metabolic theory of working hyperemia [6], the mechanism of such a "memory" must be concentration of the muscle. In fact, with an increase in the frequency of impulses the strength of contraction and the amount of physical work done by the muscle increase. This is made possible by increased breakdown of various compounds.

At the same time, when the number of contracting fibers and the frequency of the impulses remain constant, the force developed by the muscle is determined by the response of its contractile system to whatever prevents its shortening. If dilator metabolites are products of compounds whose breakdown enables the development of force, in the absence of resistance to shortening of the muscle the formation of such metabolites will be diminished. In that case, during isotonic contraction of the muscle, dilatation of its vessels in response to impulses of the same frequency will be less marked than during the development of force. If, however, the dilator metabolites are formed at earlier stages of the process (for example, in biochemical cycles of excitation or of the linking of excitation with contraction), the responses of the vessels during isotonic contractions will remain the same as during auxotonic contractions, and in both cases they will be determined entirely by the frequency of the impulses.

Laboratory of Biophysics of the Cadriovascular System, Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 69, No. 6, pp. 6-9, June, 1970. Original article submitted September 8, 1969.

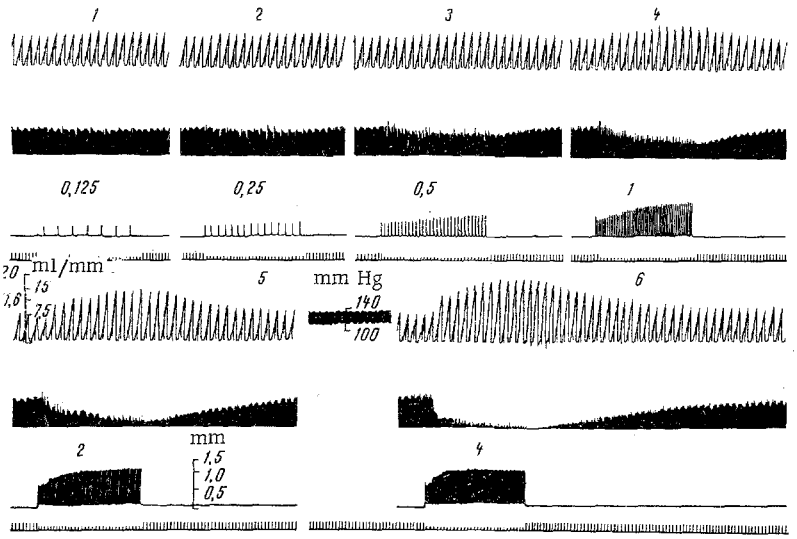

Fig. 1. Relationship between increase in outflow of blood and frequency of pulses during isotonic contraction of muscle. From top to bottom: blood flow recorded by drop counter and intervalograph, myogram, time marker (2 sec) and marker of duration of period of stimulation; curve between 5 and 6 represents arterial pressure; numbers above myogram give frequency of stimulation (pulses/sec).

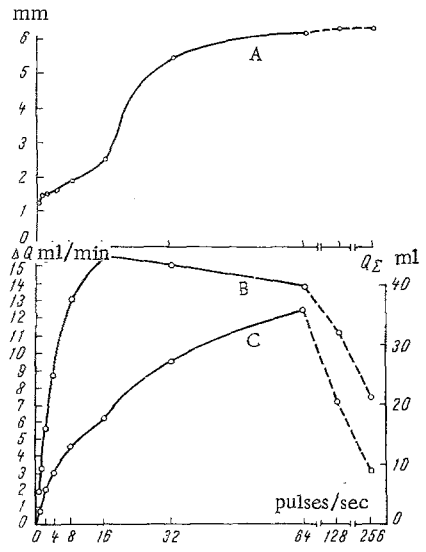

Fig. 2. Shortening of muscle (A), peak blood flow (B), and additional blood volume (C) as functions of frequency of pulses. Mean results of 7 experiments.

EXPERIMENTAL METHOD

In cats anesthetized with urethane and chloralose (0.5 and 0.05 g/kg), the outflow of blood from the veins of the left gastrocnemius muscle was measured by means of a photoelectric drop counter [3] in response to stimulation of the peripheral end of the divided sciatic nerve with square pulses (0.2 msec, not exceeding 5 V). Clotting of the blood was prevented by 5% heparin solution (0.3 ml/kg body weight). From the hermetically sealed drop counter the blood flowed into the femoral vein of the opposite limb. In the experiments of series I (4 animals), the force of auxotonic contractions of the muscle was measured by means of a tensometric sensor element [1, 5]. After each contraction the tendo Achillis was disconnected from the sensor element and stimulation of the nerve was repeated with pulses of the same frequency, evoking an isotonic contraction of the muscle. The frequency of the pulses for each pair of stimuli was doubled. The duration of stimulation was 60 sec for pulses of a frequency of 0.125-1/sec and 30 sec for pulses with a frequency of 2-256/sec. Intervals between successive stimuli were 5 or 10-15 min for the corresponding frequency ranges. The limb was fixed in the position of extension by means of steel pins inserted into the distal ends of the femur and tibia and secured by metal holders [5].

In the experiments of series II (7 animals), only isotonic contractions were produced, the amplitude of which was measured by means of a rheostatic detector. Its mechanical resistance was compensated by a weak spring (20-30 g resistance). The conditions of stimulation were the same as in the experiments of series I, except for the duration which was 60 sec for pulses of any frequency.

The velocity of the blood flow was measured by an electronic intervalograph, and in the experiments of series II it was measured simultaneously by the drop counter (every 5 sec). The pressure in the carotid artery was measured by an electromanometer. The values were recorded on an ink-writing polygraph and

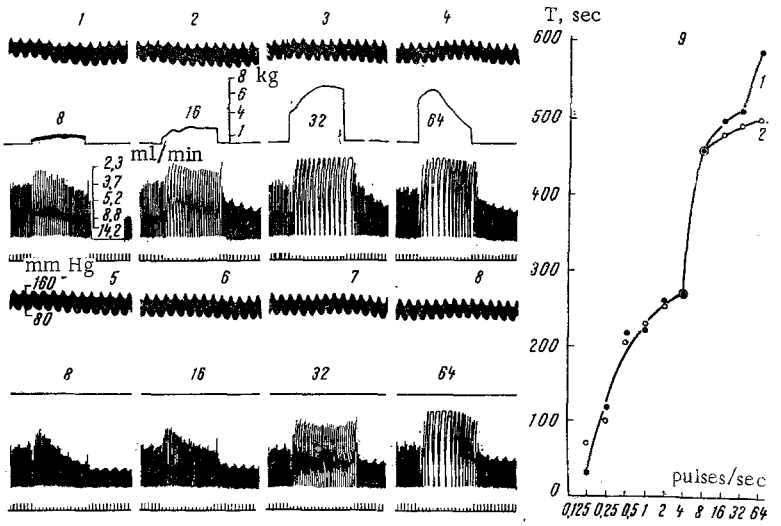

Fig. 3. Degree of compression of vessels during auxotonic (1-4) and isotonic (5-8) contractions at a frequency of 8-64/sec and duration of postcontraction hyperemia (9) as functions of pulse frequency. From top to bottom: arterial pressure, myograms (1-4), outflow of blood recorded by intervalograph, time marker (2 sec), and marker of duration of period of stimulation. On curve 9, auxotonic contractions are shown by filled circles, isotonic by empty circles.

the following indices were calculated: 1) the maximal outflow of blood after contraction of the muscle (the peak blood flow); 2) the duration of the period for recovery of tone by the blood vessels after the end of contraction and until restoration of the original blood flow, i.e., the duration of postcontraction hyperemia; 3) additional blood supply during this period (the sum of the readings of the drop counter); 4) the force; or 5) amplitude of contraction of the muscle.

EXPERIMENTAL RESULTS AND DISCUSSION

The threshold vasodilator reaction during isotonic contractions was observed at the same pulse frequency as during auxotonic contractions, namely one pulse every 8 sec (Fig. 1, 1). With an increase in the pulse frequency the rate of dilatation of the vessels and the degree to which they could reduce their resistance both increased (Fig. 1, 2-6). The intensity of vasodilatation continued to rise up to a frequency of 8-16/sec (Fig. 2B), the same as during auxotonic contractions. Values of the peak blood flow for the same frequencies of stimulation were practically identical and independent of the type of contractions.

The mechanism responsible for working hyperemia during contraction of the muscles thus acts independently of whether the contractile system is compelled to overcome resistance to shortening.

During contractions of all fibers of the muscle at a frequency of 4/sec under both isotonic and auxotonic conditions the degree of vasodilatation reached more than half its possible maximum, while at a frequency of 8/sec degree of vasodilatation was close to the possible maximum. This frequency band is the "starting line" for work of motoneurons under natural conditions [2]. Yet even at a frequency of 16/sec, shortening of the muscle did not exceed 40% of the maximum (Fig. 2A). The steepest part of the curve of muscle shortening as a function of pulse frequency lay between 16 and 32/sec. It is in this region that contraction of the muscle in all experiments led to compression of the vessels. A slight decrease in the blood supply in three experiments took place at a frequency of 8/sec (Fig. 3), and in five experiments at a frequency of 16/sec.

It will be clear from Fig. 3 that with an increase in frequency the intensity of compression of the vessels also increases. The duration of postcontraction hyperemia is correspondingly lengthened. The sharp, stepwise growth of this index, starting at a frequency at which in this experiment compression of the vessels also began (see Fig. 3, 9), is highly characteristic. This phenomenon could be explained by delay in the flushing of dilator metabolites during contraction or by hypoxia as the result of the deficient blood supply. Results of experiments to test this hypothesis will be described in subsequent articles. However,

it may be noted that during contraction of all fibers of the muscle, the duration of postcontraction hyperemia is determined entirely by the frequency of the pulses. Statistical analysis shows that it is independent of whether the muscle contracts isotonically or auxotonically.

Hill [7] has directed attention to the fact that no contraction is in fact absolutely isotonic, because there is always some small initial force applied to parallel elastic components. For displacement of a muscle during "isotonic" contraction, a force not less than the weight of the muscle (30-40 g) is necessary. For contraction at a frequency of 32-64/sec, the shortening amounts to 0.6-0.7 cm, so that the work done under these circumstances is 19.5-26 g · cm. The force of auxotonic contraction (at the same frequency of stimulation) averaged 7500 g · cm. The muscle shortened by 0.2-0.3 cm under these circumstances, so that the work done was 1500-2250 g · cm. An approximate calculation thus shows that the external physical work in the latter case is 50-100 times greater than in the first case. The degree of vasodilatation (Fig. 2) and the duration of postcontraction hyperemia (Fig. 3), however, are the same for both conditions.

The degree of vasodilatation in a working muscle and the duration of postcontraction hyperemia are thus independent of the quantity of physical work done by the muscle.

LITERATURE CITED

1. L. R. Manvelyan and V. M. Khayutin, Byull. Éksperim. Biol. i Med., No. 4, 21 (1970).
2. L. R. Manvelyan, V. M. Khayutin, and V. A. Khorunzhii, Byull. Éksperim. Biol. i Med., No. 5, 14 (1970).
3. V. M. Khayutin and P. I. Yarygin,Byull. Éksperim. Biol. i Med., No. 1, 105 (1958).
4. V. M Khayutin, in: O. Hudlicka (Editor), Circulation in Skeletal Muscle, Oxford (1968), p. 145.
5. V. A. Khorunzhkii, Byull. Éksperim. Biol. i Med., No. 11, 119 (1966).
6. W. H. Gaskell, J. Physiol. (London), 3, 48 (1880).
7. A. V. Hill, Trails and Trials in Physiology, Baltimore (1965).