RELATIONSHIP BETWEEN BLOOD FLOW IN AUXOTONICALLY CONTRACTING GASTROCNEMIUS MUSCLE AND FREQUENCY OF STIMULATION

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

UDC 612.741.14:612.741.62]:612.741.61

The relationship between working hyperemia of the gastrocnemius muscle in cats (during supermaximal stimulation of its motor fibers while contracting auxotonically) and the frequency of stimulation within the range from 0.125 to 256/sec was investigated. Maximal vasodilatation occurred at a frequency of about 16/sec, with a further increase in the frequency of stimulation, vasoconstriction began to develop and the duration of postcontraction hyperemia increased stepwise.

With an increase in intensity of motor reflexes, the frequency of impulses in the motor fibers rises from 10 to 90/sec [5]. However, the relationship between the blood supply to the contracting muscles and the frequency of impulses in their motor fibers has been investigated over a much narrower frequency band: 10-20/sec [8, 15, 16].

The object of the present investigation was to study the principles governing the blood supply of a muscle over a whole possible range of frequencies (from the threshold for development of working hyperemia to excessively high frequencies), resulting in a falling off of the strength of muscular contraction, and to establish how the blood supply to the muscle changes within the range of frequencies characteristic of natural working of motoneurons.

EXPERIMENTAL METHOD

The left gastrocnemius muscle was isolated in 9 cats anesthetized with urethane and chloralose (0.5 and 0.05 g/kg, respectively), and all blood vessels entering it except the popliteal artery and vein were ligated. The sciatic nerve and all its branches except those supplying the gastrocnemius muscle were divided. The limb was fixed in a position of extension, by introducing steel pins into the distal end of the femur and tibia and fixing them in holders. The strength of contraction developed by the muscle was recorded by a tensometric pick-up connected to the tendo Achillis. The outflow of blood from the vein was measured by means of a hermetically sealed photoelectric drop counter [4], and recorded by an electronic intervalograph [5]. The pressure in the carotid artery was measured by an electromanometer. The peripheral end of the divided sciatic nerve was stimulated with square pulses (0.2 msec, not exceeding 5 V) [2]. The frequency of the pulses was doubled at each stimulation, starting from 1/8 sec and continuing up to 256/sec. Stimulation of the nerve at the rate of 1/sec continued for 60 sec, and at the higher frequency for 30 sec. The interval between periods of stimulation was 5 min for pulses of 2/sec and 10-15 min for higher frequencies. The nerve and electrodes were covered with cotton wool soaked in mineral oil. The skin over the muscle was sutured and its temperature checked by means of an electrothermometer. Clotting of the blood was prevented by injection of 5% heparin solution (0.3 ml/kg).

Laboratory of Biophysics of the Cardiovascular 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. 5, pp. 14-18, May, 1970. Original article submitted July 4, 1969.

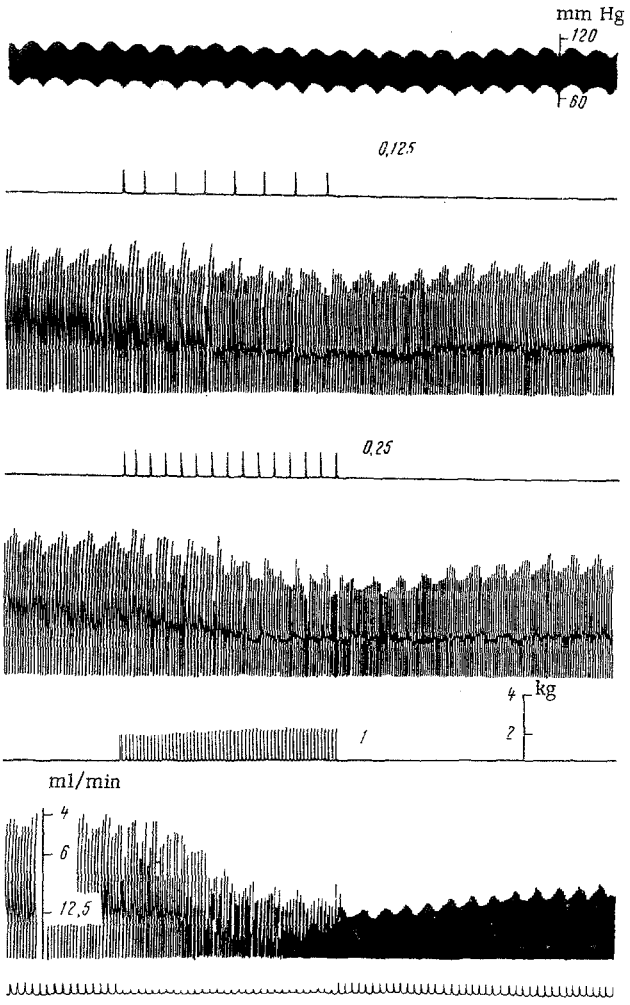

Fig. 1. Responses of vessels of gastrocnemius muscle to single auxotonic contractions separated by intervals of 8, 4, and 1 sec. From top to bottom: arterial pressure, myogram, blood outflow, time marker (2 sec), and period of stimulation (60 sec).

EXPERIMENTAL RESULTS AND DISCUSSION

The strength of isometric contractions of the cat gastrocnemius muscle may reach 25-28 kg [11]. However, in the present experiments, even at frequencies of stimulation of 64-128/sec, it did not exceed 10 kg. The reason for this is that the fixation of the limb at the knee was not sufficiently secure. For this reason, the muscle initially shortened, and equilibrium between the force developed by it and the elastic resistance of the fixing pins and the tensometric ring was reached only later. Contraction under these conditions is described as auxotonic.

After the first two or three single contractions, separated by an interval of 8 sec, an increase in the blood flow took place (Fig. 1). Evidence that this increase was not due entirely to the expression of blood from the small veins and capillaries of the muscle was given by the presence of an aftereffect: postcontraction hyperemia. With an increase in the frequency of stimulation the rate and degree of vasodilatation increased. The duration of postcontraction hyperemia also increased (Fig. 1).

Vasodilatation was accelerated to a particularly marked degree by stimulation at 2/sec and above, when the muscle could not completely reflex in the intervals between stimulating pulses and a small, but constant force was maintained throughout the period of stimulation. With an increase in frequency, this residual force increased (Fig. 2), and at 8/sec it equaled 25-50% of the total amplitude of the contractions.

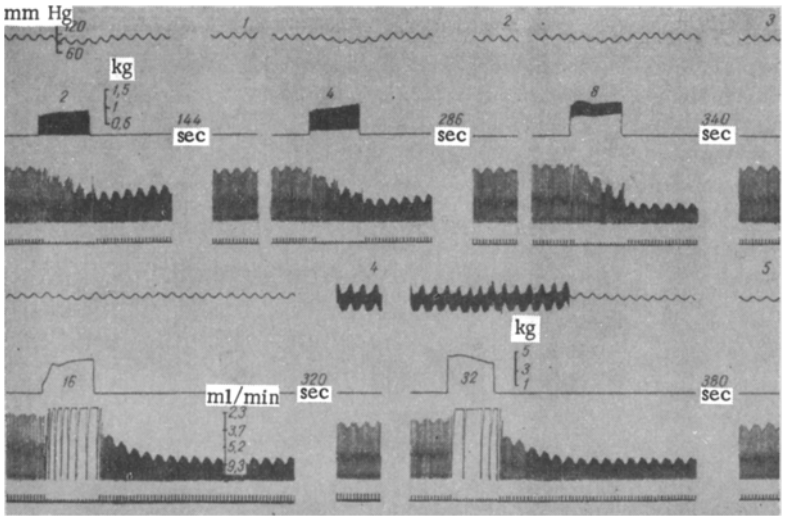

Fig. 2. Dependence of strength of auxotonic muscular contractions and changes in blood flow on frequency of stimulation. Order of curves as in Fig. 1. Numbers above myograms indicate frequency of stimulation (pulses/sec); in intervals, duration (in sec) of segments of traces of postcontraction hyperemia omitted during preparation of the figure. The circuit of the electromanometer averaging pulse fluctuations of pressure was disconnected in part of traces of 4 and 5.

at a frequency of 16/sec, the constant component of the muscle effort increased to such an extent that with a tensometric recording system of normal sensitivity, the myogram recorded was one of smooth tetanus.

In two experiments with stimulation at 8/sec, and in three at 16/sec, the blood flow increased during contraction, but its maximum was reached only after relaxation of the muscle (Fig. 2). This limitation, due to compression of the vessels, occurred in all experiments at frequencies exceeding 16/sec. The contracting muscle compressed the vessels so strongly under these conditions that the blood flow became less than initially. After relaxation of the muscle the blood flow rose considerably (Fig. 2: 4 and 5).

However, as Fig. 3 shows, the blood flow reached its maximum during stimulation at 16/sec. By contrast, the duration of postcontraction hyperemia continued to increase up to a frequency of 64/sec.

The strength of contraction reached its maximum during stimulation at 128/sec. However, even at 64/sec the contractions had become unstable: the effort developed by the muscle fell away sharply. At 256/sec this phenomenon developed so rapidly that the initial strength of contraction was actually smaller than in response to stimulation at 64-128/sec. As a result, the maximal blood flow during contractions at 256/sec did not, on the average, exceed its value during contractions at 2/sec, and usually the postcontraction hyperemia did not continue longer than after contractions at 1-2/sec. The same result, but expressed to a somewhat lesser degree, was also characteristic of contractions in response to stimuli at 128/sec.

Contraction of the muscle thus sets into operation two mechanisms which affect the resistance of its blood vessels, and hence the blood supply, in different ways. The first mechanism is the mechanism of working hyperemia proper, reducing vascular resistance, while the second is compression of the blood vessels, increasing this resistance.

It is highly significant that the first mechanism can reduce vascular resistance down to the limit within only a narrow range of frequencies: not more than 16/sec. What is the lower limit of this range for natural conditions of muscle contraction? Even during the smallest efforts of contraction, the frequency of impulses to the large muscles of the limbs does not fall below 4-5/sec [10, 17], and to the muscles of the fingers below 8-10/sec [8]. Motoneurons maintain moderate postural efforts by impulses at 7-14/sec [1] or 10-20/sec [10]. It can thus be assumed that under natural conditions, the "starting" frequency is 4-8/sec.

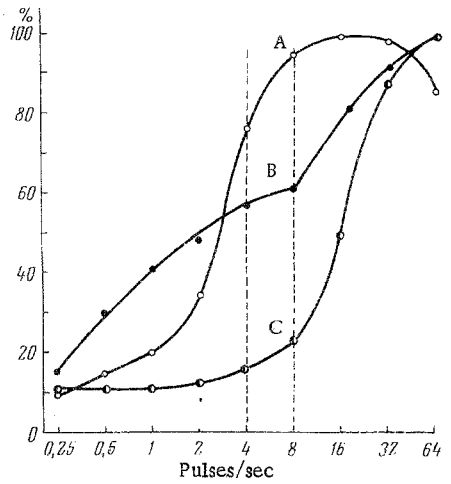

Fig. 3. Relationship between maximal blood flow (a), duration of postcontraction hyperemia (b), and strength of auxotonic contraction (c) (in percent of maximal values, along axis of ordinates) and frequency of impulses (logarithmic scale along axis of abscissas). Mean results of 7 experiments (for curves a and b), in which compression of vessels was observed at frequencies of 16/sec and above. Curve c shows mean results of 9 experiments. Vertical broken lines mark boundaries of region of "starting" frequency of motoneurons during natural contraction of muscles.

The sensitivity of the mechanism controlling dilatation of resistive vessels is very high. Working hyperemia appears during contractions produced by impulses of very low frequency, and even by single impulses (Fig. 1), which are never generated by motoneurons. Contraction of muscle fibers at a frequency corresponding to the "starting" frequency for only 30 sec was able to reduce the resistance of the vessels on the average to 70-90% of the possible maximum (Fig. 3). However, during supramaximal stimulation of the nerve at this frequency the strength of contraction reached only 15-20% of the possible maximum (Fig. 3). It is evident that recruiting of motor units to maintain weak postural efforts can result in an almost maximal dilatation of the blood vessels in the region of these units.

During voluntary efforts exceeding 25% of the possible maximum, motoneurons begin to generate impulses at a frequency of 25-35/sec [8]. During contractions of this type mechanisms leading to compression of the vessels become dominant [7, 15]. In the present experiment, at a frequency of 32/sec, the blood supply to the muscle actually became smaller than initially. The muscle was contracting itself "into debt." Since the "debt" could be "paid off" only after the contraction, so that the necessary time for "payment" could be obtained, it was essential that the frequency of impulses during contraction be proportional to the duration of the postcontraction hyperemia (Fig. 3).

On the assumption that the classical, metabolic theory of working hyperemia [12, 13] holds good, it might be considered that the duration of postcontraction hyperemia is determined by the concentration of metabolites, which in turn, rises in proportion to the frequency of stimulation. From this point of view, it is not surprising that at frequencies exceeding 8-16/sec, at which no further vasodilatation can possibly take place, the duration of postcontraction hyperemia increases. However, this explanation cannot be regarded as satisfactory, for it does not take into account the irregularity of the change in duration of postcontraction hyperemia as a function of pulse frequency. Within the region 4-8/sec, this duration virtually shows no increase, and the curve enters the region of saturation (Fig. 3). The state of saturation comes to an end when compression of the vessels develops with an increase in the frequency of stimulation. Obviously under these circumstances some additional factor comes into play and retards the course of processes restoring the tone of the resistive vessels. In subsequent articles the writers will show that this factor is compression of resistive vessels by contracting fibers of the muscle, and that the intensity of their compression is determined by the pulse frequency and is practically independent of the degree of mechanical resistance to shortening of the muscle and, consequently, of the force developed by the muscle.

LITERATURE CITED

1. V. S. Gurfinkel', Ya. M. Kots, and M. L. Shik, Regulation of Posture in Man [in Russian], Moscow (1965).
2. L. R. Manvelyan and V. M. Khayutin, Byull. Éksperim. Biol. i Med., No. 4, 21 (1970).
3. V. I. Tkhorevskii, Fiziol. Zh. SSSR, No. 2, 199 (1968).
4. V. M. Khayutin and P. I. Yarygin, Byull. Éksperim. Biol. i Med., No. 1, 105 (1958).
5. V. A. Khorunzhii, in: Regulation of the Regional Circulation [in Russian], Leningrad (1955), p. 53.
6. E. D. Adrian and D. W. Bronk, J. Physiol. (London), 67, 119 (1929).
7. H. Barcroft and J. L. E. Millen, J. Physiol. (London), 97, 17 (1939).
8. B. Bigland and O. C. J. Lippold, J. Physiol. (London), 125, 322 (1954).
9. W. C. Bowman, J. Pharm. (London), 11, 641 (1959).

10. F. Bracci, M. Decandia, and T. Gualtierotti, Am. J. Physiol., 210, 1170 (1966).
11. S. Cooper and J. C. Eccles, J. Physiol. (London), 69, 377 (1930).
12. W. H. Gaskell, J. Physiol. (London), 3, 48 (1880).
13. S. M. Hilton, Physiol. Rev., 42, Suppl. 5, 265 (1962).
14. I. Kjellmer, Acta Physiol. Scand., 62, 18 (1964).
15. A. R. Lind and G. W. McNicol, J. Physiol. (London), 192, 595 (1967).
16. H. Merker, H. Ochwald, and W. Schoedel, Pflüg. Arch. Ges. Physiol., 251, 73 (1949).
17. F. H. Norris and E. Z. Gasteiger, Electroenceph. Clin. Neurophysiol., 7, 115 (1955).