TWO PHASES OF PROTEIN HYPEREMIA OF SKELETAL MUSCLE

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The dynamics of working hyperemia of the gastrocnemius muscle was studied on acute experiments on cats with the aid of an electromagnetic flowmeter. The increase in blood flow was shown to be divided into two phases: Phase I was characterized by a rapid increase in blood flow; during phase II the rate of its increase fell sharply. The duration of phase I was independent both of the frequency of stimulation and of the number of motor units participating in contraction. With a small number of contracting motor units or a low frequency of contraction phase II did not develop. It is postulated that phase I of working hyperemia is connected with opening of the precapillary arterioles and phase II with dilatation of larger arteries.

KEY WORDS: *Circulation; working hyperemia; ascending vasodilatation.*

During working hyperemia in skeletal muscles not only the intramuscular, but also extramuscular arteries dilate, although the processes taking place during contraction of the muscle (biochemical processes, at least) cannot directly influence the extramuscular arteries [5, 8, 9, 10]. Changes in the diameter of the femoral artery are small: The hydraulic resistance falls by only 15-25% [7, 9]. Nevertheless, the decrease in hydraulic resistance of the intramuscular arteries may be more substantial [7, 9]. In that case their dilatation during working hyperemia (WH) could make a noticeable contribution to an increase in blood supply to the working muscle. Dilatation of the femoral artery begins 20-40 sec after the beginning of muscular contraction [7, 9].

If the intramuscular arteries also begin to dilate after dilatation (or opening) of the precapillary arterioles, this delay should be reflected in the dynamics of the increase in blood supply to the muscles. The object of this investigation was to test this hypothesis.

EXPERIMENTAL METHOD

In cats anesthetized with urethane and chloralose (0.5 and 0.3 g/kg) the left hind limb was securely fixed [3]. The gastrocnemius muscle (GM) was dissected and a piece of the calcaneus with Achilles tendon was sawn off and connected to a recording instrument. In this way, knowing the initial length of the muscle, the force of its isometric contraction could be measured or the muscle could be loaded and its shortening against a constant force recorded. The popliteal artery and vein were isolated and all vascular branches divided except those supplying GM. To record the arterial inflow the popliteal artery was divided and connected by means of a cannula to the main trunk transducer (diameter 2 mm) of an RKÉ-1 electromagnetic flowmeter [4]. When the venous outflow was recorded the same transducer was connected to the peripheral end of the popliteal vein. Venous blood was drained from it into the opposite femoral vein. The RKÉ-1 instrument can be used to measure blood flows with an absolute error of not more than 0.06 ml/min. The linear characteristic of the instrument was confirmed by calibration for blood. The blood flow rate signal was recorded with a transmission band of 0.3 Hz. In this way, because the pulse waves were smoothed out, rapid

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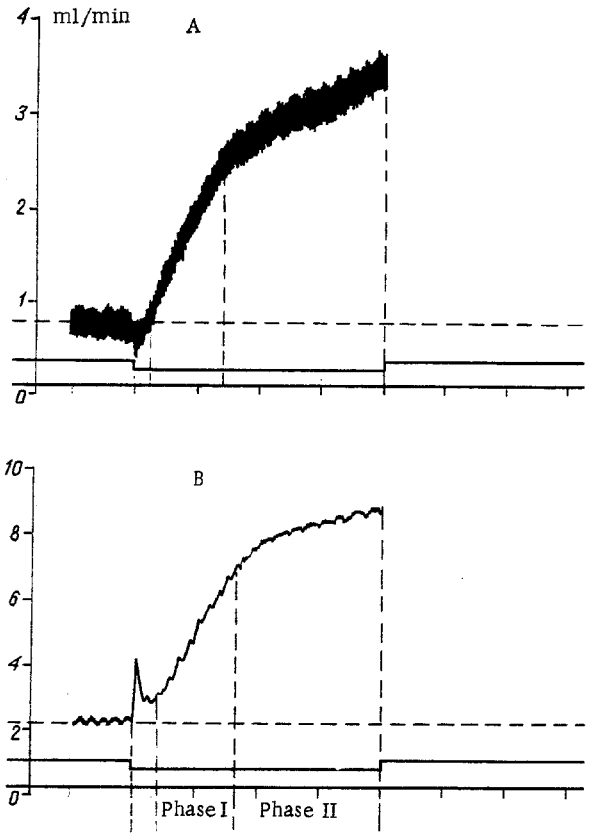

Fig. 1. Measurements of blood flow during contraction of gastrocnemius muscle in cats. A) Arterial inflow; B) venous outflow. In both cases stimulation by pulses of supermaximal strength and with a frequency of 8 sec^{-1}. From top to bottom: blood flow (broken line shows initial level of blood flow), stimulus marker, time marker (10 sec).

changes in blood flow during WH could be observed. Contractions of GM were induced by stimulation of the peripheral end of the sciatic nerve, all branches of which were divided except those supplying GM. In the experiments of series I (19 cats) different numbers of fibers of GM were caused to contract in response to stimuli with a frequency of 8 sec^{-1}. The amplitude of the stimuli (duration 0.2 msec) was chosen so that the strength of short test isometric contractions was 5, 10,..., 100% of the maximal strength. During recording of the blood flow, GM was made to contract against a constant force of 0.5 kg. In the experiments of series II (24 cats) the nerve was stimulated with supermaximal stimuli and their frequency was increased from 4 to 24 sec^{-1}. Under these circumstances the muscle contracted against a constant force of 0.5 kg just as in the experiments of series I. The pressure in the carotid artery was measured with an electromanometer and recorded on a KSP-4 automatic potentiometer. The blood flow was recorded on the second channel of the KSP-4 instrument.

EXPERIMENTAL RESULTS

The process of WH can be divided into two phases: phase I, a period of rapid increase of blood flow (15-20 sec), preceded by a latent period of 1-3 sec; phase II, a period of slow increase of the blood flow. The curves obtained by recording the arterial inflow during WH (Fig. 1A) differed from the venous outflow curves (Fig. 1B) in the absence of the initial discharge of blood from the veins of the contracting muscle. It will be noted that when the outflow of blood from the contracting GM was recorded previously by means of an intervalograph [1, 2], because of the gross nonlinearity of this method of recording, it was impossible to distinguish the phase of change in the velocity of the blood flow during WH clearly.

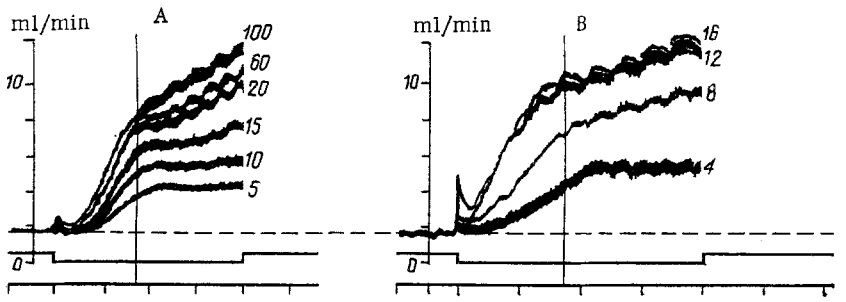

Fig. 2. Outflow of blood from isotonically contracting cat gastrocnemius muscle. A) Frequency of stimulation 8 sec^{-1}. Numbers show strength of isometric contraction (in % of maximal); B) Supermaximal stimulation; numbers show frequency of stimulation (in sec^{-1}). Legend as in Fig. 1.

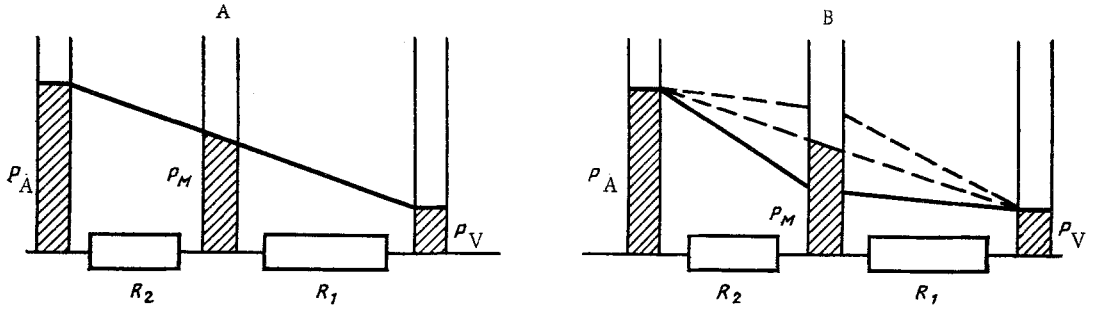

Fig. 3. Diagram of hydraulic resistance of arterial part of vascular system of a skeletal muscle. Hydraulic resistance: R_1) precapillary arterioles; R_2) larger arteries; P_A arterial pressure; P_M pressure at entrance into precapillary arterioles; P_V venous pressure. Distribution of pressures: A) in resting muscle; B) during working hyperemia. Continuous line shows decrease chiefly in R_1. Top broken line shows decrease mainly in R_2. Bottom broken line shows resistances changing so that the ratio between them is constant. Explanation in text.

The duration of phase I was independent both of the strength of contraction (i.e., the number of contracting muscle fibers) and of the frequency of stimulation. In all experiments, irrespective of the parameters of nerve stimulation, the duration of phase I was constant for the given animal (Fig. 2). However, the rate of increase of the blood flow during phase I increased with an increase both in the strength of contraction of GM and in the frequency of stimulation. As a result, the blood flow measured at the end of phase I differed by as much as 10 times for extreme value of the strength of contraction or the frequency of stimulation. With small strengths of contraction (5 and 10% of maximal) or a low frequency of stimulation (4 sec^{-1}) the blood flow did not increase after phase I, although the contraction lasted longer. With stronger contractions or higher frequencies of stimulation, phase II of WH began a few seconds after phase I (Fig. 2). The rate of increase of the blood flow in phase II rose with an increase in the strength of contraction or the frequency of stimulation. In both series of experiments the dynamics of the increase in blood flow was thus similar although the frequency of stimulation and the number of contracting fibers differed, yet, despite this fact, the duration of phase I was the same, namely 15-20 sec.

The hydraulic resistance of the whole network of resistive vessels of the muscle can be represented as the sum of the resistances of the small (precapillaries) and the larger vessels. If the systemic pressure remains unchanged, the pressure at the entrance to the small vessels will depend on the ratio between the resistance of these and the largest vessels and not on the absolute value of the corresponding resistances. If during WH the resistance of the precapillary arterioles and of the larger vessels changed so that the ratio between remained constant, the pressure at the entrance to the former would remain unchanged

also. If, however, during contraction of the muscle the strength of one of the resistances changed, this would lead to a change in pressure at the entrance to the small vessels (Fig. 3). During contraction of GM the pressure in vessels of the order of 100 μ is known to fall initially, but later it recovers as the contraction continues [6]. This fact suggests that during WH the small vessels open first. This is manifested as phase I. The constancy of the duration of this phase, i.e., the independence of its duration of the number of contracting fibers and the frequency of stimulation, is evidently determined by the fact that a time of 15-20 sec from the beginning of contraction of GM is required for the process of opening (dilatation) of the precapillary arterioles to reach completion. The increase in the rate of rise of the blood flow with an increase in the number of contracting fibers or in the frequency of stimulation in phase I is due to the increasing number of precapillary arterioles involved in dilatation.

To correspond with what was said above, phase II can be regarded as a reflection of dilatation of the large vessels. The fact that dilatation of the femoral artery begins a short time after contraction of GM led to the view that this phenomenon is connected with the conduction of the wave of vasodilatation from the arterioles [5, 7, 10]. However, replacement of a segment of the artery by a prosthesis (proximally to the point where the diameter was recorded) does not abolish its dilatation during WH [9]. This shows that the response is local in origin, possibly a response to acceleration of the blood flow [8]. It is interesting to note from this point of view that phase II is absent if the increase in blood flow in phase I is sufficiently slow (Fig. 2). It can accordingly be postulated that dilatation of the large intramuscular vessels is a local response to acceleration of the blood flow (or to a fall in pressure) in those arteries.

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