INVESTIGATIONOF THE WORKING HYPEREMIA OF SKELETAL MUSCLE: PARAMETERS OF ELECTRICAL STIMULATION OF A MIXED NERVE

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Experiments on cats have shown that square pulses, 0.2 msec in duration and up to 3 V in voltage, produce supermaximal stimulation of motor fibers of the divided sciatic nerve without exciting vasomotor fibers, and they thus enable selective stimulation of these fibers to be carried out during the investigation of mechanisms of working hyperemia.

Injection of curare into an animal blocks muscular contraction in response to stimulation of the peripheral end of a divided muscular nerve and prevents the development of changes in the blood supply to the muscle [1, 5, 7, 8]. This fact shows that working hyperemia of the skeletal muscle, produced by electrical stimulation of its nerve, is due to excitation of its motor fibers only. To avoid simultaneous excitation of vasomotor fibers, Keller and co-workers [9] stimulated the nerve with an alternating current of 100-600 Hz. However, this frequency produces Wedensky inhibition.

If short square pulses are used, the amplitude at which they excite low-threshold (medullated) motor fibers without exciting high-threshold vasomotor (nonmedullated) fibers can be determined.

EXPERIMENTAL METHOD AND RESULTS

To determine the necessary pulse parameters, the strength of contraction of the gastrocnemius muscle and the flow of blood from its veins were measured in cats anesthetized with urethane and chloralose (0.5 and 0.05 g/kg, respectively). The rate of blood flow was measured by means of an airtight drop counter [2] and recorded on an electronic intervalograph, working with an ink-writing galvanometer [3]. The gastrocnemius muscle was lifted, and the small vessels and all branches of the popliteal artery and vein lying beneath it, except branches to the heads of the gastrocnemius muscle, were ligated. The heads of the muscle were left attached to the condyles of the knee joint. The sciatic nerve was divided in the popliteal fossa and the peripheral end placed on electrodes (platinum plates 1.5 mm in width). All branches of this nerve to other muscles were also divided. The popliteal fossa was filled with mineral oil.

The strength of maximal isometric concentration of the gastocnemius muscle in cats may reach 25-30 kg [6]. To obtain strictly isometric contractions, fixation of the limb both at the knee joint and relative to the sensor element measuring the strength of contraction must therefore be as rigid as possible. For this purpose, steel pins were passed through the distal ends of the femur and tibia and fixed to an apparatus designed for fixation of the limbs in the horizontal plane. Stronger contractions of the muscle required firm attachment of the tendo Achillis to the sensor recording this strength. A piece of the calcaneus to which the tendo Achillis is attached was removed with a saw [10]. Two half-rings were fitted to the tendon in front of the bond, placed in a U-shaped holder, and fixed to it with two screws. Holes are present in each of the half-rings. The half-rings were brought together by means of the screws, thereby preventing sliding of the bond during a strong isometric contraction. The U-shaped tendon holder was connected by a steel wire with the

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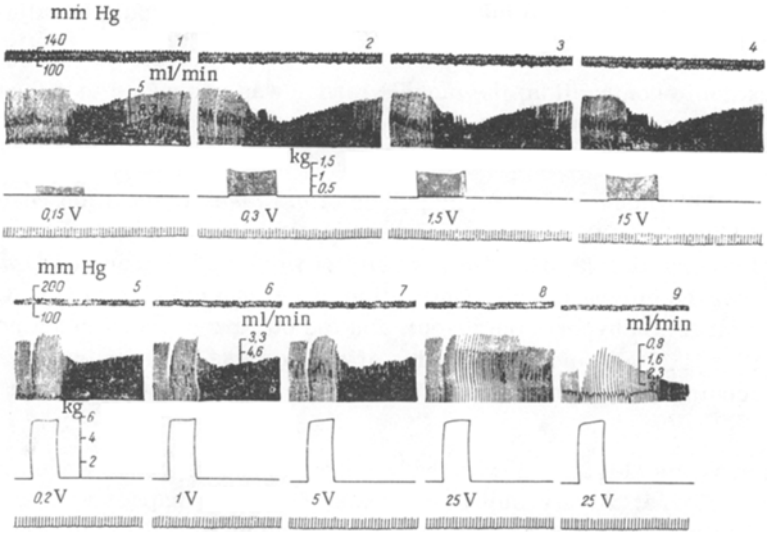

Fig. 1. Effect of pulse amplitude (duration 0.2 msec, frequency 20/sec) during stimulation of peripheral end of divided sciatic nerve on strength of auxotonic contractions and outflow of blood from gastrocnemius muscle of cat. From top to bottom: pressure in carotid artery, blood flow recorded by intervalograph, myogram, time marker (2 sec). Numbers above marker give amplitude of pulses (V), frequency of stimuli 2/sec (1-4) and 20/sec (5-9).

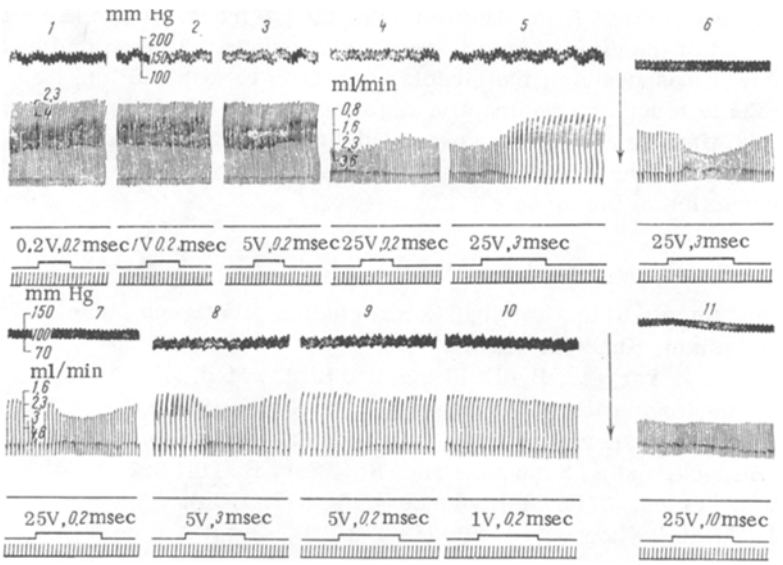

Fig. 2. Response of vessels of gastrocnemius muscle of a cat immobilized with succinylcholine iodide (150 mg/kg/min) to change in amplitude and duration of pulses stimulating nerve. Significance of curves as in Fig. 1. Numbers above marker of stimulation show amplitude (V) and duration (msec) of pulses, frequency 20/sec in every case. Injection of dihydroergotamine between 5 and 6; of atropine (intravenously in doses of 0.5 mg/kg) between 10 and 11.

sensor of the strength of contraction. The initial tension in the muscle could be adjusted by displacement of the sensor in the apparatus [4].

To produce an auxotonic contraction, the muscle tendon was connected to a tensometric ring through a U-shaped spring plate. During its contraction the free ends separated by 5-6 mm. The rigidity of the plate then increased sharply, and after preliminary shortening the muscle developed a force of up to 10 kg.

Single contractions of the muscle evoked by pulses of 0.2 msec in duration reached maximal strength at an amplitude of 0.3 V (Fig. 1: 1, 2). An increase in pulse amplitude by 5, and then by 50 times did not increase the strength of contraction [3, 4]. The intensity of working hyperemia likewise was unchanged. The same result was observed during tetanic contraction of the muscle (Fig. 1: 5). Only when the voltage reached 25 V was postcontraction hyperemia absent, and the outflow of blood remained slow even after contraction of the muscle (Fig. 1: 8). A more clear idea of the process is given by trace 9 of Fig. 1, in which all conditions of the preceding trace were repeated except that the rate of movement of the intervalograph pen was slower.

Pharmacological analysis (Fig. 2) showed that changes in the response were due to excitation of high-threshold vasoconstrictor fibers. As a result of blockade of motor synapses with succinylcholine iodide, stimulation of the nerve with pulses of 0.2 msec in duration and up to 5 V in amplitude caused neither contraction of the muscle nor reactions of its vessels (Fig. 2: 1-3). A weak constrictor reaction appeared only when the amplitude was 25 V (Fig. 2: 4). The reaction was strengthened if the pulse duration was increased to 3 msec (Fig. 1: 5). This response was replaced by vasodilatation after administration of dihydroergotamine, specifically blocking adrenergic receptors, to the animal (Fig. 2: 6).

Since dilatation of the vessels of the curarized muscle was abolished by atropine (Fig. 2: 11), it must have arisen through excitation of cholinergic dilators [11]. The threshold of excitation of these fibers of the sciatic nerve during stimulation with pulses 0.2 msec in duration was about 5 V (Fig. 2: 9). It is important to stress that during stimulation of the nerve with pulses of the same duration, but with an amplitude of 1 V (supramaximal for excitation of motor fibers) the blood flow was unchanged (Figs. 1: 2 and 2: 10).

Changes in the outflow of blood from the vessels of the gastrocnemius muscle during electrical stimulation of the peripheral end of the divided sciatic nerve with pulses 0.2 msec in duration and up to 3-4 V in amplitude, were thus due to responses of the muscle vessels to its contraction, i.e., to stimulation of motor fibers exclusively. In the absence of a contractile reaction stimulation of these fibers did not affect the outflow of blood either at the time or after stimulation of the nerve. Excitation of sympathetic vasomotor fibers took place during stimulation of the sciatic nerve by pulses of much greater amplitude than is necessary for producing maximal contraction of the muscle.

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