REFLEX CHANGES IN ARTERIAL PRESSURE TO CONTRACTION OF THE GASTROCNEMIUS MUSCLE IN CATS

T. I. Sirota

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In cats anesthetized with chloralose and urethane either pressor (up to 12 mm Hg) or depressor (up to 24 mm Hg) reflexes or the change from depressor into pressor reflexes was observed with an increase in the frequency of stimulation of motor nerves. Reflex changes in arterial pressure increased with an increase in the number of motor units involved in contraction and in the frequency of stimulating pulses. Reflex changes in blood pressure in response to contraction of the muscle are evidently evoked by activation of group III mechanoreceptors and, perhaps, of group IV receptors also.

An important aspect of the mechanism of the changes in circulation during muscular activity is the role of reflexes arising in response to contraction of skeletal muscles [2, 6]. During passive stretching of the muscles, the arterial pressure rises [1] or falls [17]. During tetanic isometric contraction of the leg muscles evoked by stimulation of the sciatic nerve in spinal cats, pressor reflexes of up to 20 mm develop [11]. During tetanic contraction of the lower limb muscles of cats induced by stimulation of the ventral roots of L6, L7, and S1, pressor reflexes of up to 50 mm are observed [7].

The object of the investigation described below was to discover how reflex changes in arterial pressure in response to contraction of the gastrocnemius muscle depend on the number of motor units involved in contraction and on the frequency of the impulses reaching them.

EXPERIMENTAL METHOD

Cats were anesthetized (chloralose 30 mg/kg and urethane 0.5 g/kg in 22 experiments, chloralose 50 mg/kg in three experiments), the left hind limb was securely fixed, and the gastrocnemius muscle attached by the calcaneus to a strain gage as described previously [4]. All nerves running from motor roots L7 and S1 to the limb, except branches to the left gastrocnemius muscle were divided. After laminectomy the ventral and dorsal roots of L7 and S1 were divided on the right. The brain was flooded with mineral oil (38°C), and the body temperature was maintained at 37.5-38°C and the muscle temperature at 36-37°C. To measure the strength of the maximum possible isometric contraction, segments of the left motor roots of L7 and S1 were stimulated with supermaximal pulses 0.5 msec in duration and with frequency $f = 64$ sec^{-1} for 2.5 sec. The amplitude of the pulses was then reduced to values at which the strength of contraction was approximately 25 to 75% of the maximal. The response of the arterial pressure to contraction of the muscle was measured by an electromanometer in the carotid artery. To avoid injury to the muscle through prolonged isometric contraction with the joints rigidly fixed [9], the reflex changes in arterial pressure were investigated while the muscle was shortened. During these investigations the muscle carried a load of 350 g. The duration of the contractions was 15-20 sec and the intervals between them 10-15 min. The pulse frequency varied from 4 to 64 sec^{-1}. Usually the first series of contractions was evoked by pulses of supermaximal amplitude, and the next series by submaximal pulses. The amplitudes of the pulses corresponded to previously determined strengths of isometric contractions.

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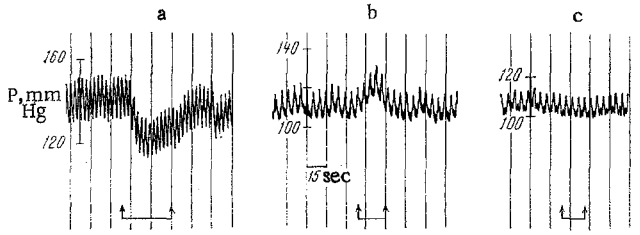

Fig. 1. Changes in arterial pressure during contraction of gastrocnemius muscle induced by stimulation of motor roots L7 and S1. Parameters of stimulation: 1 V, 32 sec^{-1}, 0.05 msec. a) Experiment No. 8; b) experiment No. 18; c) the same experiment later division of the corresponding dorsal roots. Arrows denote period of stimulation.

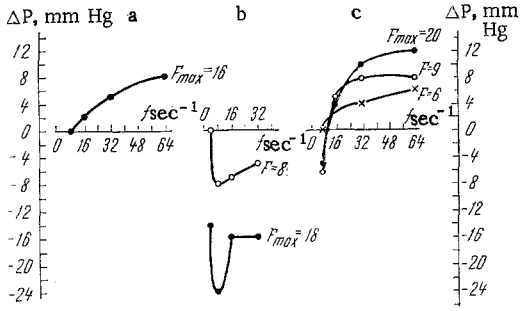

Fig. 2. Reflex changes in arterial pressure as a function of frequency of stimulation. Amplitudes of pulses correspond to strengths of isometric contractions indicated on graphs, τ = 0.05 msec: a) experiment No. 20; b) No. 15; c) No. 5. F in kg.

EXPERIMENTAL RESULTS

Records of changes in arterial pressure (ΔP) arising during contraction of the gastrocnemius muscle are shown in Fig. 1. After division of the corresponding dorsal roots the responses of the arterial pressure disappeared (Fig. 1c). It thus follows that they were reflex in origin. Pressor, depressor or combined depressor and pressor reflexes arose in different animals.

Curves showing ΔP as a function of the frequency of stimulation for different amplitudes of the pulses are shown in Fig. 2.

1. In nine animals only pressor reflexes, not exceeding 10-12 mm Hg, appeared in response to stimulation of the motor roots. With an increase in f the pressor reflex also increased (Fig. 2a). In four experiments there was no reflex in response to frequencies of 4 or 8 sec^{-1}.

2. In nine animals only depressor reflexes were observed throughout the range of pulse frequencies (Fig. 2b). At 4 sec^{-1} the pressure fell on the average by 16 mm, and at 32 sec^{-1} it fell by 11 mm.

3. In seven animals, in response to stimulation of motor fibers by pulses at frequencies of 8-16 sec^{-1} the pressure fell, but at higher pressures it rose (Fig. 2c). This change from depressor to pressor took place at different frequencies in different experiments. In this group of experiments ΔP ranged from −10 to + 18 mm Hg.

In each of the experiments of the three groups described above the overall characteristics corresponded to one of the types shown in Fig. 2.

In ten experiments a family of characteristics of $\Delta P(f)$ could be obtained for parameters of stimulation corresponding both to 100% strength of contraction and to submaximal contraction. The absolute value of ΔP rose with an increase in the number of muscle units involved (which corresponds to an increase in the strength of isometric muscle contraction).

In response to contraction of the gastrocnemius muscle induced by stimulation of its motor fibers in cats anesthetized with chloralose and urethane, reflexes of both pressor and depressor types can thus develop. The changes in pressure increase with an increase in the frequency of stimulating pulses and in the number of fibers.

Investigations [10, 12, 13] have shown that electrical stimulation of afferent fibers of groups I and II of muscular nerves does not induce vasomotor reflexes. According to some observations [10, 12, 17] electrical stimulation of group III afferent fibers of muscular nerves in animals lightly anesthetized with urethane leads to elevation of the arterial pressure. However, with deepening of anesthesia the reflex becomes depressor. Under these conditions the inhibitory component of the response of vasoconstrictor neurons to stimulation of the group III fibers is evidently increased, and this causes the appearance of depressor reflexes [3]. Only the reflexes evoked by selective stimulation of group IV fibers were always pressor in character [12].

Endings of group III and IV fibers are activated during contraction [5, 12, 14]. During tetanic contraction of the gastrocnemius muscle a low-frequency discharge appears in almost half of the local pressure

receptors, impulses from which are conducted along group III fibers [14]. The results of the present experiments and those of the investigations cited above suggest that reflex changes of arterial pressure in response to muscle contraction are evoked by activation of mechanoreceptors of group III and also, perhaps, by group IV receptors.

The results of the experiments with electrical stimulation of muscle afferents help to explain the reasons why adequate stimulation of muscle receptors during contraction of the muscle evokes reflex changes of arterial pressure of different character in different animals. The appearance of depressor reflexes in some of the present experiments performed on animals under mixed urethane and chloralose anesthesia could be attributed to central blocking of the transmission of impulses by group IV fibers [15], which are less resistant to anesthesia than central transmission of group III impulses. Under these conditions the pressor reflexes may be changed into depressor, a phenomenon which is characteristic not only of electrical stimulation of afferent fibers [8]. This view is also supported by the fact that in three experiments when chloralose anesthesia was used, only pressor reflexes (of the order of 10 mm) were recorded. Also under chloralose anesthesia, during massive contraction of the hind limb muscles [7] pressor reflexes of up to 50 mm were observed. This is understandable if it is remembered that the number of motor units participating in such contraction is definitely greater than in the present experiments. In spinal animals, in which the inhibitory component of the response of the vasoconstrictor neurons is absent [11], only pressor reflexes were observed. In the experiments of group 3, the change from depressor into pressor reflexes was observed with an increase in the frequency of the stimulating pulses. This change was probably due to restoration of the central effects of impulses transmitted along group IV fibers as a result of the increase in frequency of their discharges with increased strength of muscle contraction. In fact, the formation of responses to electrical stimulation of group IV afferent fibers of muscular nerves requires temporal summation, and the higher the frequency of stimulation, the more strongly this summation is manifested [16].

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