MUSCLE RECEPTOR EXCITABILITY IN RELATION TO CARDIAC ACTIVITY AND ARTERIAL PRESSURE

V. P. Kolychev

From the Department of Normal Physiology (Head - Professor M. R. Mogendovich), Perm Medical Institute (Director - Doctor of Medical Sciences I. I. Kositsyn) (Received January 20, 1959. Presented by Active Member of the AMN SSSR V. V. Parin)

A study of pressor reactions during muscular work due to alterations in the excitability of proprioceptors as induced by drugs, is of both theoretical and practical importance. M. F. Golovkina [2] has used this method in coldblooded animals. She decerebrated frogs by I. M. Sechenov's method, and observed the fall in blood pressure on cooling the gastrocnemius muscle or immersing it in a 0.5 novocaine solution. Changes in the cardiovascular system caused by cooling a muscle or by its treatment with novocaine may be due to the effective elimination of the proprioceptors. However, in order to obtain a complete proof, it is necessary to exclude the direct effect of cooling, which may involve the heart, and which by itself might produce a depressor effect [3, 10]. In working on coldblooded animals, we have therefore attempted to use the most delicate method of temporarily eliminating proprioceptors (depressing their excitability) by using anelectrotonus. Our starting point has been the fact established by us previously [5], that the excitability of muscle receptors may be either increased or decreased by being brought into contact with an anode or a cathode.

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In warmblooded animals, which have a constant temperature and more highly developed thermoregulatory mechanisms than coldblooded species, proprioception was depressed by cooling the muscle or by the application of novocaine. At the same time, the general body temperature was controlled.

The object of the present investigation was to reduce the excitability of receptors in resting muscle by treatment with drugs, and to observe the effect on the cardiovascular system.

METHOD

The experiments were carried out on 60 autumn frogs (Rana temporaria) and on 20 adult rabbits. The first experiment was carried out on bulbospinal frogs in which the heart and gastrocnemius muscles were exposed. A silver choride needle electrode was inserted in one of the gastrocnemius muscles, and the indifferent electrode plate placed on the belly. One to one-and-a-half hours after making the preparation, the initial e.c.g. was recorded; the constant current was then applied and the effect of anelectrotonus or catelectrotonus on cardiac activity found. The current was then switched off, and the recording continued until the heart returned to its initial state. The current applied to the gastrocnemius ranged in strength from 3 to 30 ma.

The next experiment was carried out on acute preparations of unanesthetized rabbits. The effect on the arterial pressure of effectively eliminating the receptors of the triceps femoris was found. This effect was obtained by cooling the muscle in frozen physiological saline, or by applying a 2% novocaine solution; the latter was either applied to the surface of the muscle, or 2-3 ml were injected intramuscularly. Blood pressure was recorded from the common carotid by an aneriod or a mercury manometer. The rectal temperature was measured.

TABLE 1

Changes in Cardiac Activity Following "Unipolar" Application of an Anode or Cathode to the Gastroenemius Muscle

| Action on muscle | Change in cardiac activity | | | | | | | | |
|---------------------|----------------------------|-----------|-----------|-----------|----------|-----------|--|--|--|
| | | amplitude | | frequency | | | | | |
| | increase | decrease | no change | increase | decrease | no change | | | |
| Anode (120 tests) | | 55 | 65 | _ | 59 | 61 | | | |
| Cathode (130 tests) | 88 | 12 | 30 | 91 | 12 | 27 | | | |

TABLE 2

Experiment No. 19 Without Anesthetic. Female Rabbit, weight 3.5 kg (16/II 1957)

| Time at which exp. | 77 | Site of action (paw) | B1 (in | Rabbit | | |
|------------------------------------|------------------------|----------------------|---------------------|-----------------------------|--------------------|-------|
| carried out (in hours and minutes) | Treatment of muscle | | before treatment | during treatmen r | after treatment | toman |
| 14.00-14.05 | Coil separation, 10 cm | Left | 120 | 130 | 119 | 37,6 |
| 14.10—14.15 the same | | Right | 120 | 127 | 121 | 37.6 |
| 14.25—14.30 Cooling | | Right | 120 | 112 | 117 | 37.5 |
| 14.40—14.45 Coil separation 10 cm | | Lef t | 116 | 124 | 118 | 37.5 |
| 14.55—15.00 the same | | Right | 117 | 118 | 116 | 37.4 |
| 15.15—15.20 Coil separation 7 cm | | Left | 116 | 132 | 120 | 37.4 |
| 15.30—15.35 the same | | Right | 120 | 120 | 120 | 37.3 |
| 15.55—16.00 | Novocaine injection | Left | 121 | 115 | 115 | 37.3 |
| .16.15—16.20 | Coil separation, 7 cm | Left | 115 | 116 | 116 | 37.3 |
| | | 1 | | | | - |

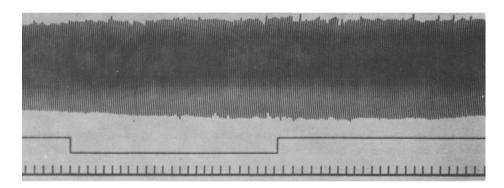


Fig. 1. Heart changes following application of a cathode to the gastrocnemius muscle. Curves, from above downwards; heart contractions, stimulation of gastrocnemius muscle, time marker (5 seconds)

RESULTS

Table 1 summarizes the results obtained on frogs.

We have seen that applying the cathode to a skeletal muscle caused an increase in amplitude and frequency of the heart beat in 88 out of 130 cases, and that only in an insignificant number of the tests was an inhibitory effect produced.

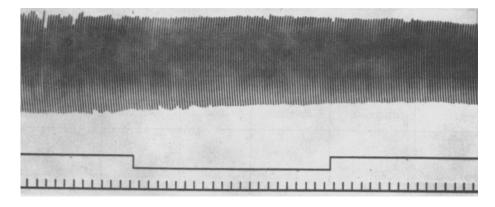


Fig. 2. Change in cardiac activity following treatment of the gastrocnemius muscle with an anode. Curves as in Fig. 1.

On the other hand, the effect of the anode almost always exerted an inhibitory influence, and a stimulating effect on the heart was never obtained.

Both direct observation and the e.c.g. records showed that the effect of applying the cathode to the gastroc-nemius muscle was to stimulate the heart, the effect being greater on the amplitude of the contractions than on their rate (Fig. 1). The increase in the contraction amplitude was due to a stronger stystole and a greater relaxation during diastole. The depression in activity following treatment of the muscle with the anode was also due to changes in the amplitude of contraction (Fig. 2).

It must be noted that the changes described above were not very marked when a weak galvanic current was used; but evidently the mechanism plays an important part in regulating the cardiovascular system, particularly in the case of pathological proprioceptive disorders and central regulatory cardiac reflexes.

When the direct current was applied to the muscle, single contractions occurred, and the question naturally arises as to whether the cardiac changes resulted from these contractions. In some experiments, therefore, the current was established as follows: The switch was closed at a subthreshold value of the current, insufficient to induce muscular contraction; then gradually, by means of a rheostat, the strength of the current was increased to the required value. It was found that change in the performance of the heart was precisely the same whichever way the current was applied.

Thus, change in the excitability of resting skeletal muscle receptors induced electronically may have the same effect as muscular work [1, 5] in reflexly increasing cardiac output. There is reason to suppose that the stimulating effect caused by cathodic treatment is due to an increase in proprioceptive afferent impulses, while reduction in cardiac output following anodic treatment of the muscle results from a fall (elimination) of proprioceptor impulses.

This view has been confirmed by Edwards [11], who studied the effect of catelectrotonus and anelectrotonus on a single afferent axon innervating the muscle. According to him catelectrotonus caused an increase in the rate of impulses from a muscle spindle, while anelectrotonus had the opposite effect.

The next stage was to study the effect on the heart of temporary proprioceptor suppression in warmblooded animals. The experiments showed that even the temporary elimination of the receptors of a single muscle produces marked effects on the blood pressure. As an example, the results of a typical experiment on a rabbit are shown in Table 2.

As can be seen from Table 2, elimination of skeletal muscle reception by cooling or by novocaine injections causes a rapid fall in arterial pressure of 6-8 mm mercury on average, while stimulating the unesthetized muscle with current from an induction coil causes an increase in pressure. Current from an induction coil applied to a skeletal muscle which has been previously cooled or anesthetized with novocaine has practically no effect on the blood pressure, although the muscle itself contracts strongly. No appreciable body temperature change resulted from the cooling. Similar results were obtained in experiments on other rabbits.

Comparing the results of the experiments on frog and rabbit, it can be seen that different factors acting on resting skeletal muscle and altering its excitability cause reflex cardiovascular changes. The actions of anelectrotonus, novocaine, and cooling are similar. If the muscle receptors are temporarily eliminated by suppressing their excitability, reflex changes occur in the circulatory system and possibly also in other internal organs.

This leads to the question of in what way reduction or complete elimination of the muscle receptors affects the cardiovascular system. It has been suggested that the continuous flow of impulses from skeletal muscle receptors which maintain a certain tone but which are not actually contracted, has a continuous influence on the nervous centers and so regulates the circulatory system. In other words, the level of activity of the cardiovascular system, even at rest, appears to be maintained by a constant flow of impulses originating in skeletal muscle receptors which are at rest, but which have a tone. M. R. Mogendovich, A. K. Chuvaev, and G. Z. Chuvaeva have demonstrated a relationship between the tone of the arm muscles and arterial pressure, under clinical conditions [8].

The cardiovascular system reacts therefore not only to contraction or relaxation of skeletal muscle [4, 6, 8, 12], but also to an increase or decrease (elimination) of the excitability of the receptors of muscles which may be in a condition of comparative rest. These effects must be mediated by visceromotor reflexes [7].

SUMMARY

Experiments were performed on frogs and rabbits. A study was made of the dependence of the cardiovascular system on changes in proprioceptor excitability induced by anesthesia. It was shown that application of a cathode to a frog gastrocnemium causes an increase in the heart rate; on the other hand, application of an anode to the muscle depresses cardiac activity. Cooling the triceps femoris of a rabbit, or injecting novocaine into it, causes a rapid decrease in arterial pressure of 6-8 mm Hg. Consequently, not only muscular activity, but also change of excitability of the skeletal muscle proprioceptors is accompanied by reflex circulatory changes. These are visceromotor effects.

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