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EFFECT OF PROLONGED HYPOKINESIA ON RESISTANCE OF RESISTIVE VESSELS IN RATS

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Under the effect of prolonged hypokinesia, the perfusion pressure in resistive vessels, measured under conditions of deep anesthesia and complete denervation, increased by approximately the same degree as arterial pressure in non-anesthetized animals. The increase in arterial, perfusion pressure and the resistance of resistive vessels in animals subjected to prolonged hypokinesia was accompanied by an increase in adrenoreactivity. During prolonged hypokinesia, partial obliteration of the vascular bed of the skeletal muscles plays a significant role in the observed increase in resistance of vessels of the extremities. The increase in adrenoreactivity of the vessels during hypokinesia may be realized as a partial case of an increase in the adrenoreactivity of structures whose innervation is disturbed.
EFFECT OF PROLONGED HYPOKINESIA ON RESISTANCE OF RESISTIVE VESSELS

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It has recently been shown that during considerable hypokinesia partial atrophy of the cardiac muscle and the vascular bed of the skeletal muscles is combined with a reduction in the functional potentialities of the circulatory system as a whole [2, 9, 10]. At the same time, the question of the ratio of the per-minute volume of circulation, resistance of the vascular bed and arterial pressure during hypokinesia is not clear in many respects. The majority of researchers have thus noted an increase in arterial pressure in people and animals exposed to considerable hypokinesia [1, 2, 8]. However, it is not clear whether this depends on the increase in resistance of the resistive vessels or the per-minute volume, since the studies obtained contradictory data [3, 5, 12]. No direct measurement has yet been made of the resistance of the resistive vessels during prolonged hypokinesia. The purpose of this work was correspondingly to measure the resistance of the resistive vessels of the extremity of animals who endured lengthy hypokinesia by recording the perfusion pressure in these vessels under conditions of regulated blood flow.

The experiments were performed on mongrel male rats in two stages. The first stage of the experiment was conducted on 11 rats and provided for the recording of systolic arterial pressure on the tail with the help of the physiograph "Narco Bio-systems" (United States) in the same animals before and after limited motor activity for 3 months. The second stage of the experiment was conducted under urethane anesthesia (1.6 mg/g intraperitoneally) and provide for

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recording of the perfusion pressure with increasing amounts of blood flow in 10 control animals and 11 animals exposed to the effect of hypokinesia for 3 months. The pressor reaction in response to the administration of noradrenaline (0.035 μg/kg/min) was also measured. Noradrenaline was administered by a microinjector directly into the femoral artery at a rate of 0.025 ml/min. with a filling rate of 0.9 ml/min.

Hypokinesia was attained by placing the animals in special chambers of small size that were made according to the body size of the animal. The animals that were exposed to hypokinesia for 3 months weighed 265 ± 4 g, and the control animals weighed 385 ± 4 g.

In order to measure the tone of the resistive vessels, a previously employed technique was used [11]. The hind limb of the rat was perfused through the femoral artery with blood which came from the carotid artery. The perfusion was done with the help of a roller pump with gradual increase in blood flow from 0.4 to 2.3 ml/min. The perfusion pressure in the artery was recorded on a one-channel potentiometer KSP-4 with the help of a pressure gage "Elema-Shonander."

Hydraulic resistance was computed according to the formula: 

\[ R = \frac{P}{Q} \text{ per } 100 \text{ g of weight of the limb}, \]

where \( R \) is the resistance (in units of resistance), \( P \)--perfusion pressure (in mm Hg), \( Q \)--volumetric velocity of blood flow (in ml/min per 100 g of limb weight).

Measurement of the arterial pressure in the nonanesthetized animals indicated that in the control it was 115 ± 2.4 mm Hg, in the animals who had experienced lengthy hypokinesia it was 145 ± 2.6 mm Hg (p < 0.01). Thus, hypokinesia resulted in a reliable increase in the arterial pressure by 26%.

The curves in fig. 1 characterize the dynamics for the perfusion pressure in the resistive vessels of the control animals and the animals who experienced hypokinesia, with a gradual increase in the
filling rate. With even rate of filling the vessels, the perfusion pressure in the animals who underwent hypokinesia increased by 31-36%.

Consequently, under the influence of prolonged hypokinesia, the perfusion pressure in the resistive vessels measured during deep anesthesia and complete denervation increased roughly to the same degree as the arterial pressure in nonanesthetized animals.

The resistance of the resistive vessels, as indicated, was computed by dividing the perfusion pressure by the amount of blood flow in the limb relative to the weight of the limb. Since muscle atrophy of the limbs in the animals exposed to hypokinesia was significant, and the weight of the limb was reduced by 29.6%, the amounts of blood flow which we created through the limb, in calculation for a unit of weight were greater than in the control animals. With equal perfusion pressure, this had to result in the fact that the resistance of the resistive vessels of the animals exposed to hypokinesia was lower than in the control. The curves of fig. 2 show that the resistance of the resistive vessels in the animals exposed to hypokinesia with equal rates of filling was actually 13% greater than in the control.

An increase in arterial, perfusion pressure, and finally the resistance of the resistive vessels in animals exposed to prolonged hypokinesia was naturally accompanied by an increase in the adrenal reactivity. In response to the addition to the blood of an equal
On the whole, the findings show that with an equal volume of blood injected into the vascular bed of the limb, the resistive vessels of the limb of the animals who were exposed to prolonged hypokinesia have greater resistance and increased adrenal activity. In explaining this phenomenon, one should bear in mind at least three possibilities.

The first possibility follows from the fact which has been proven by morphological studies that with the duration of hypokinesia that we used, a considerable reduction is observed in the number of capillaries in the muscles of the limb, as well as degenerative changes in the wall of the arterioles and obliteration of part of them [2]. These data make it possible to assume that during prolonged hypokinesia, partial obliteration of the vascular bed of the skeletal muscles plays a significant role in the increase in the resistive resistance of the limb vessels that we found.

The second possibility follows from the fact that during prolonged hypokinesia, an increased output of calcium from the bones into the blood plasma is observed. According to certain data, the calcium content in the blood is also increased [4]. This may lead to intensified entrance of calcium into the musculature of the vessels and

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**Figure 2.** Hydraulic Resistance of Control Rats (a) and Animals with Hypokinetic Activity for 90 Days (b)

On x-axis: normalized blood flow (in ml/min per 100 g of limb weight);
On y-axis: normalized hydraulic resistance (in mm Hg/ml/min per 100 g of limb weight)
as a consequence of this, an increase in the tone and resistance of the preserved vessels of the limb.

Finally, the third possibility is important for an explanation of the increased adrenal reactivity during hypokinesia. It states that during hypokinesia, atrophic changes develop early in the neuro-muscular synapses [6], and there is a reduction in the activity of the sympathetic-arenal system [9] and the concentration of catecholamines in the skeletal muscle [7]. This allows us to think that an increase in the adrenal reactivity of the vessels during hypokinesia may be realized as a particular case of increased adrenal reactivity of the structures whose innervation has been disrupted.

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