IMPORTANCE OF CAROTID SINUS REFLEX REGULATION DURING THE ACTION OF CROSS ACCELERATION FORCES

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E. B. Shul'zhenko

Laboratory of General and Experimental Cardiology (Head, Active Member AMN SSSR, Professor V. V. Parin), Institute of Normal and Pathological Physiology (Director, Active Member AMN SSSR, Professor V. V. Parin) of the AMN SSSR, Moscow
(Presented by Active Member AMN SSSR, V. V. Parin)
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During the action of the physiologically most convenient transverse forces of inertial acceleration, certain characteristic changes are observed in the cardiovascular and respiratory systems. These changes are the result of the fact that the increased hydrostatic pressure of the blood stimulates the pressure receptors of the cardiovascular system with an intensity which depends on the magnitude of the overload. The minute volume of blood falls, while the peripheral resistance remains essentially unchanged. The mean pressure in the aorta is lowered [2-5]. The changes which develop are probably based on complex interrelationships between the sympathetic and parasympathetic mechanisms of regulation of the cardiovascular system.

The object of the present investigation was to elucidate the role of the pressure receptors of the carotid sinuses in the regulation of the hemodynamic changes arising during the action of cross acceleration forces.

EXPERIMENTAL METHOD

Experiments were carried out on ten dogs of both sexes weighing 10-15 kg. Under general anesthesia (morphine 0.75 mg/kg, chloralose 50 mg/kg, Nembutal 10 mg/kg), a catheter was introduced into the left ventricle and aorta of the dog. The pressure was recorded by means of tensometric pick-ups placed at the level of the heart and firmly fixed to the chair of the centrifuge. Respiration was maintained by means of a special apparatus. The animals were exposed to the action of acceleration forces of increasing strength (3-6-9 g), for a period of 1 min for each strength. Bilateral denervation of the carotid sinuses was then carried out and the experiment was repeated. To determine the minute volume of blood and the peripheral resistance, Maxwell's method was used [6].

EXPERIMENTAL RESULTS

During exposure to overloads of increasing magnitude, the pulse rate of the dogs with intact carotid sinuses rose and reached its maximum at 9 g. It regained its initial level 3 min after stopping the centrifuge (Fig. 1A).

The values of the systolic pressure, the mean pressure during systole, and the mean intra-aortic pressure during the cycle fell slightly during the action of overloads of 3 g and at the beginning of the application of an overload of 6 g, after which they rose and reached their maximum at an overload of 9 g. The diastolic pressure rose at the end of the action of an overload of 6 g. At 9 g its value was significantly higher than originally. The pulse pressure, which fell initially, rose slightly at the end of exposure to all overloads, although it still remained below the initial value. It rose during the first minute after stopping the centrifuge and returned to its initial level after 3 min (Fig. 2A).

The minute volume fell at the beginning of overloading and then stayed at this level until the end of exposure to an overload of 9 g, when it rose. This rise was especially marked during the first minute after stopping the centrifuge (Fig. 1B).

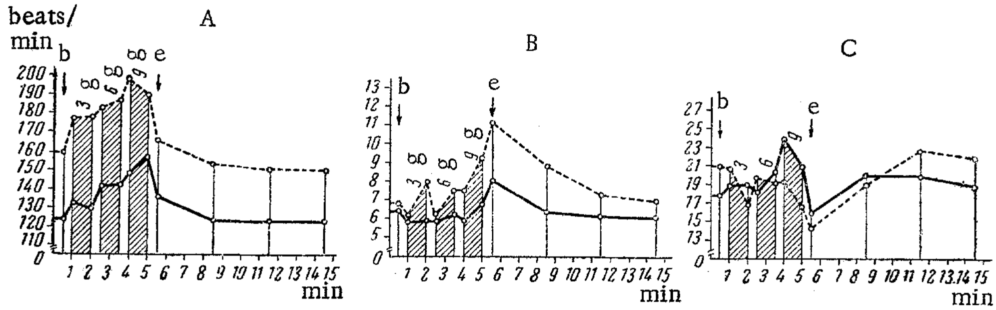

Fig. 1. Changes in the rhythm (A) and minute volume of the heart (B) and in the peripheral resistance (C) during exposure of dogs with intact (continuous line) and denervated (broken line) carotid sinuses to overloads of 3, 6, and 9 g; b) beginning of rotation; e) end of rotation of centrifuge.

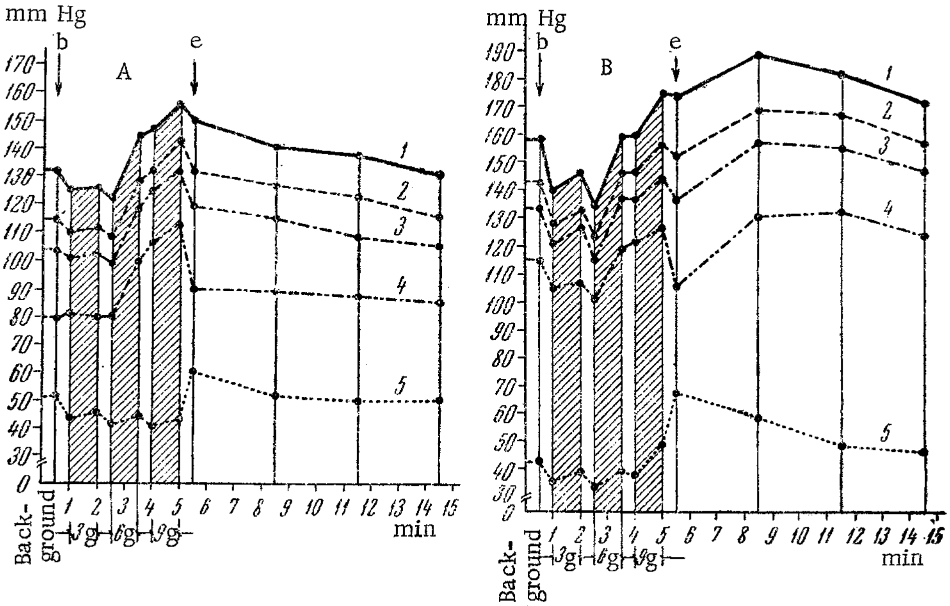

Fig. 2. Changes in intra-aortic pressure during the action of overloads of 3, 6, and 9 g on dogs with intact (A) and denervated (B) carotid sinuses: 1) systolic pressure; 2) mean pressure throughout systole; 3) mean pressure throughout cycle; 4) diastolic pressure; 5) pulse pressure. Rest of legend the same as in Fig. 1.

The peripheral resistance rose during overloading and reached its maximum at the beginning of the action of an overload of 9 g. It fell slightly during the 1st minute after stopping the centrifuge, but during the 3rd and 7th minutes its value once again exceeded the original level (Fig. 1C).

The animal responded to presentation of the overload first by an increase in the pulse rate and peripheral resistance. If overloads of 3 g and 9 g are regarded as preliminary or preparatory, after increasing the overload to 9 g the development of hyperfunction of the heart, directed towards the compensation of the disturbances arising in the body during exposure to acceleration, was clearly observed. The compensatory reaction began at the end of exposure to 9 g. After stopping the centrifuge, the functional state of the cardiovascular system gradually returned to normal.

After bilateral denervation of the carotid sinuses against the background of a pressor reaction of the animal ($P < 0.001$) to overloading, a more marked increase in the rhythm was observed than in the intact dogs. The increase in the heart rate reached its maximum at the beginning of exposure to an overload of 9 g. During the 3rd-7th min after stopping the centrifuge, the rhythm remained below its initial level (see Fig. 1A).

At the beginning of overloading by 3 g and 6 g, the dogs with denervated carotid sinuses showed a more marked fall of pressure in the aorta than the intact animals. At the end of exposure to an overload of 6 g and at the beginning of exposure to 9 g, the arterial pressure fluctuated within the limits of the original level, and exceeded it at the end of overloading. The degree of this excess in the dogs with denervated carotid sinuses was 17 mm Hg or 11%, while in animals with intact carotid sinuses it was 24 mm Hg, or 18% ($P < 0.05$). After stopping the centrifuge, the arterial pressure continued to rise and reached its maximum in the 3rd minute. The diastolic pressure, which fell at the beginning of overloading, remained below the initial level until the end of exposure to overloading of 6 g, after which it rose again and exceeded the initial value. The same reaction was observed during exposure to 9 g; the pulse pressure fell, but the changes differed from those observed in the dogs with intact carotid sinuses by the greater variations of the absolute values during the period of exposure; after stopping the centrifuge, the pulse pressure rose to 58% of its initial value (Fig. 2B).

The changes described in the animals with denervated carotid sinuses took place against the background of a lowered peripheral resistance throughout the period of overloading. The peripheral resistance fell considerably during the 1st minute after stopping the centrifuge and then rose during the 3rd-7th min (Fig. 1C).

The value of the minute volume, which fell at the beginning of all the overloads, rose and exceeded the initial value. It reached its maximum during the 1st min after stopping the centrifuge and remained above its initial level until the 3rd-7th minute (see Fig. 1B).

Hence, after denervation of the carotid sinuses, a sharper fall was observed in all the indices of the arterial pressure at the beginning of exposure to overloads of 3 g and 6 g. Mobilization of the compensatory mechanisms in these circumstances was inadequate, for the increase of pressure in the period of overloading was less marked than in the dogs with intact carotid sinuses. This was especially obvious at the beginning of exposure to an overload of 9 g. From 1 to 3 min after stopping the centrifuge, the arterial pressure rose significantly against the background of a sharp rise of the minute volume. Evidently, in the absence of the pressure receptors of the carotid sinuses, the body is unable to compensate for the hemodynamic disturbances produced by overloading, and not until the overloading ends does a reaction develop aimed at correcting the observed changes.

In the light of ideas of the adaptive significance of the vascular tone [1], it is important to note the changes in the arterial pressure during overloading took place in the animals with intact carotid sinuses against the background of an increased heart rate, an increased vascular tone, and insignificant fluctuations in the minute volume, while in the animals with denervated carotid sinuses they took place against the background of an increased pulse rate, an increased minute volume, and a lowered peripheral resistance. Consequently, in the first case, the adaptive changes in the rhythm and vascular tone were adequate to maintain the arterial pressure at the level essential for the animal during overloading. In the second case, despite the pressor effect after denervation of the sinuses, the vascular tone was lowered during overloading, and the increased rhythm and minute volume were inadequate to compensate for the hemodynamic changes arising during overloading. The development of a maximal phase of compensation in the second case took place only after the centrifuge had stopped.

It is possible, therefore, that during the action of increasing overloads, the adaptive changes in vascular tone play a leading role in the compensatory changes in the cardiovascular system. The action of acceleration forces evidently produces its effect on the changes in the peripheral vascular resistance through a complex chain of reflex acts. When the pressure receptor system of the carotid sinuses is intact, adaptive changes of vascular tone develop actively, whereas, after denervation of the carotid sinuses, the action of overloading lowers the peripheral resistance of the vessels, for this important link in the neuroreflex regulation of vascular tone is absent.

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