ROLE OF THE CAROTID SINUS PRESSURE RECEPTORS IN REFLEX REGULATION OF HEMODYNAMIC CHANGES IN UNANESTHETIZED ANIMALS EXPOSED TO TRANSVERSE ACCELERATIONS

E. B. Shul'zhenko

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During transverse acceleration, with increasing compression of the thoracic and abdominal organs, changes are observed to develop in both systemic [1, 5, 9, 11, 12] and pulmonary circulations [3, 9, 11, 12]. The continuous functional unity of the hemodynamic changes observed during the action of acceleration suggests that a complex chain of interconnected reflex actions is involved.

A series of experiments was carried out on unanesthetized animals to discover the role of the carotid sinus pressure receptors in the responses of the cardiovascular system to acceleration.

EXPERIMENTAL METHOD

Experiments were carried out on two groups of animals (each consisting of 6 dogs): in the animals of group 1 the carotid sinuses were intact, while in those of group 2 they were denervated. On the 3rd, 4th, and 6th days after the operation the animals were exposed to an acceleration of 9g in the direction from sternum to spine for a period of 1.5 min. The pressure inside the left ventricle and aorta was recorded during the experiments. The pressure was measured by tensometric detectors and recorded on a "Physiograph" apparatus (Elema). The minute blood volume and peripheral resistance were calculated by Maxwell's method [10]. This method cannot give quantitative estimates of changes in these parameters, but merely indicates their trend. Because of this, to visualize the changes in minute volume of the blood more clearly, they were expressed as percentages of the initial value. Changes in the peripheral resistance are shown as the calculated values. For catheterization of the left ventricle and aorta in long-term experiments standard thin catheters were used and were introduced through the left carotid artery and fixed in the vessel by means of the connecting sleeve. After measurement of the blood pressure, physiological saline (2-4 ml) with heparin (50 i.u./ml solution) was injected into the catheter. At the end of injection of the solution, a clamp was applied to the polyethylene portion of the catheter. After disconnection of the detector, the distal end of the catheter was warmed until it softened and it was then compressed by a second clamp, thereby sealing it. In this way the lumen of the catheter was hermetically closed and after removal of the clamps no blood entered it. Catheterization was carried out under the control of an oscilloscope. Thrombus formation was prevented by rinsing the catheters by Turanski's method [7].

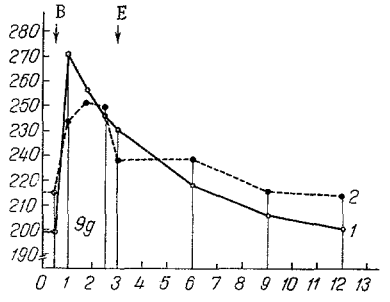

Fig. 1. Changes in systolic pressure in left ventricle during acceleration of 9 g lasting for 1.5 min. In unanesthetized animals with intact (1) and denervated (2) carotid sinuses. B) Beginning; E) end of rotation of centrifuge. Abscissa) time (in min), ordinate) pressure (in mm Hg).

EXPERIMENTAL RESULTS AND DISCUSSION

In the unanesthetized animals subjected to transverse acceleration in the sternum-spine direction the pulse rate and minute volume of the blood were increased, the peripheral resistance was reduced, and the arterial pressure was elevated. Although these changes took

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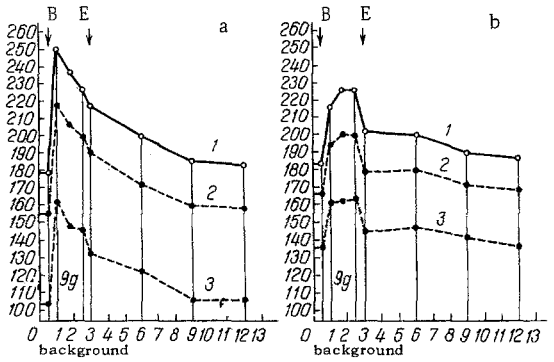

Fig. 2. Changes in pressure within the aorta during exposure to acceleration of 9 g for 1.5 min in unanesthetized animals with intact (a) and denervated (b) carotid sinuses. 1) Systolic pressure; 2) mean during systole; 3) diastolic. Remainder of legend as in Fig. 1.

place in the same direct in animals with intact and denervated carotid sinuses, they were much more marked in the second case.

In the animals of group 1 (intact carotid sinuses) a maximal increase of pressure in the left ventricle and aorta was observed from the very beginning of exposure to acceleration of 9 g (Figs. 1 and 2). This state of transient hypertension developed against the background of a lowering of vascular tone and was due to a maximal increase in the minute volume of the blood (Fig. 3). This change of pressure in unanesthetized animals exposed to acceleration is one of the principal mechanisms of adaptive reactions based on compensatory hyperfunction of the heart. When blood is stored in the lungs and too little flows into the left ventricle, hyperfunctional of the heart takes place probably by a more complete ejection of blood from the left ventricle.

It may be postulated that the mechanism of the observed changes in the hemodynamics was one of redistribution of blood in the system of the pulmonary artery, leading to a continuous increase in the blood volume in the lung and in the resistance of the vessels. Increased stimulation of the receptors of the pulmonary vessels causes a lowering of tone of the systemic vessels, increasing the load on the right ventricle, and thereby lowering the tone of the vagal innervation and raising the tone of the cardiac accelerator nerves.

After denervation of the carotid sinuses, evoking a pressor reaction, the changes at the beginning of acceleration took the form of a small increase in pulse rate, a small increase of the minute volume of the blood, and a decrease in the peripheral resistance throughout the period of acceleration (Fig. 3). Although the arterial pressure exceeded its initial level, its reaction took place after a longer interval, was torpid in character, and differed considerably in magnitude from that observed when the carotid sinuses were intact (Figs. 1 and 2). This was because after denervation of the carotid sinuses, modifications to the functional state of the cardiovascular system take place with the participation of other interoceptive zones, so that the adaptive powers of the cardiovascular system are reduced. Despite the absolute increase in heart rate during the period of acceleration in the animals of group 2, compensatory hyperfunction of the heart was less marked as a result of a decrease in the contractile power of the myocardium. The degree of this decrease may be judged from the level of the pressure in the left ventricle. The smaller increase of pressure in the left ventricle of animals with denervated carotid sinuses suggests that functional exclusion of the carotid sinus pressure receptors reduces the contractile power of the myocardium, as a result of which the minute volume of blood and the pressure rise slightly.

The maximal increase of arterial pressure at the beginning of acceleration and the apparently increased stimulation of the carotid sinus pressure receptors ought to slow the heart. However, this was not observed: the heart rate remained high throughout the exposure to acceleration. Although the nature of

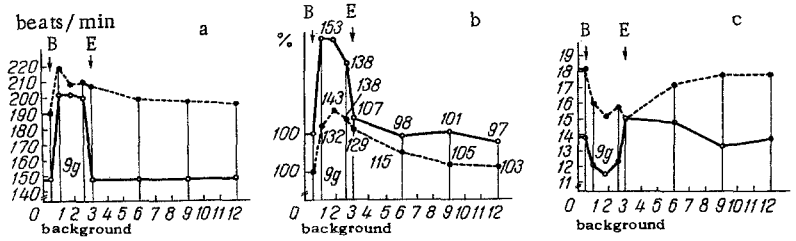

Fig. 3. Changes in heart rate (a), minute volume of blood (b), and peripheral resistance (c) of unanesthetized animals exposed to acceleration of 9 g for 1.5 min.

this phenomenon is unknown, it may be postulated that in unanesthetized animals a purposive reorganization of the afferent impulsation takes place under the influence of the higher levels of cortical regulation.

During the action of acceleration the tone of the vagal center is probably lowered and that of the sympathetic accelerator nerves raised on account of the increased afferent impulsation from different interoceptors of the blood vessels, heart, groups of muscles, and so on. Nevertheless, the chief ("trigger") mechanism in this phenomenon is that connected with the pressure receptors of the carotid sinuses. This is because their high reactivity and their constant afferent activity, observed even with normal fluctuations of arterial pressure, enable the functional state of the cardiovascular system to adapt itself to acceleration without a short time interval after its new level has been assigned. Against this background, other reflex mechanisms of regulation are brought into play, and their synergism ensures an adequate level of compensatory and adaptive changes in response to the action of acceleration.

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