CHANGES IN SENSITIVITY OF AORTIC BARORECEPTORS UNDER THE INFLUENCE OF THE SYMPATHETIC NERVOUS SYSTEM

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The dependence of activity of the aortic baroreceptors on the pressure in the aorta during stimulation of the branch from the stellate ganglion innervating the aortic baroreceptor zone was studied in experiments on cats. Stimulation of the sympathetic branch was shown not to change the threshold of activity of the baroreceptors (80 mm Hg) or their activity within the range from 80 to 140 mm Hg. With higher pressures in the aorta, stimulation of the sympathetic branch reduced baroreceptor activity, as a result of which the curve of baroreceptor activity as a function of pressure became flatter and reached maximal activity at a higher intraaortic pressure (on the average 32 mm Hg higher). The mechanisms of the action of the sympathetic nerves on baroreceptor sensitivity and the physiological significance of this phenomenon are discussed.

KEY WORDS: aortic baroreceptors; afferent impulsation; nervous regulation of the circulation.

Physical exertion, emotions, and stress are accompanied by excitation of the sympathetic nervous system, as a result of which changes take place in the basic parameters of the circulation: The arterial pressure rises, the cardiac output and frequency of the cardiac contractions are increased, and so on. The mechanoreceptor zones of the cardiovascular system play a role in these changes in the regime of the circulation. Morphological studies have shown that the principal baroreceptor vascular zones (the aorta and carotid sinus) are abundantly supplied with efferent sympathetic fibers [1, 13, 14]. This fact has served as a basis for the suggestion that the sensitivity of the baroreceptors is tuned by the sympathetic nervous system. This hypothesis has been tested experimentally in only a few investigations. Their results have proved conflicting: During stimulation of the sympathetic branch innervating the carotid sinus baroreceptor zone increased baroreceptor activity was observed in some experiments [8, 15] but no effect in others [6, 11]. A decrease in baroreceptor activity in the right aortic nerve during stimulation of the sympathetic branch was obtained by Keith et al. [7].

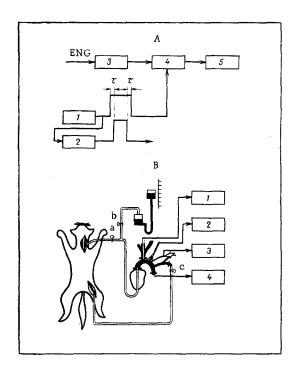
In the investigation described below the effect of the sympathetic branch innervating the aortic baroreceptor zone on the sensitivity of the baroreceptors was studied at different levels of pressure in the aorta.

EXPERIMENTAL METHOD

Cats were anesthetized with chloralose and urethane. In the experiments of series I the blood pressure in the right common carotid artery and the activity of the whole trunk of the left aortic nerve were recorded. The integral of this activity, determined over a constant period of time, was chosen as the quantitative characteristic of activity in the nerve. The IÉ-I integrator, producing voltage pulses at the output, the amplitude of which was proportional to the integral activity of the input signal over the chosen integration period, was used for this purpose. After thoractomy the left stellate ganglion was dissected. The branch from it innervating the aortic baroreceptor zone was left intact and all the other branches were divided. The ganglion for the intact branch from it was stimulated with square pulses (1 msec, 10 Hz, 6-10 V). To avoid inductance from stimulation of the electrodes recording activity in the aortic nerve, the scheme shown in Fig. 1A was set up. Activity in the aortic nerve, amplified by the amplifier 3, was led through an electronic key 4 to the input of the inte-

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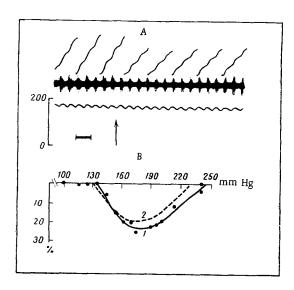


Fig. 1 Fig. 2

Fig. 1. Scheme showing assembly of apparatus for preventing inductance from stimulation (A) and block diagram of experiments on isolated preparation of arch of aorta (B). In A: 1 and 2) stimulators; 3) amplifier; 4) electronic key; 5) integrator. In B: 1) electromanometer; 2) amplifier; 3) stimulator; 4) electrothermometer; a, b, c) clamps.

Fig. 2. Effect of stimulation of sympathetic branch on activity in aortic nerve (A) and dependence of decrease in activity of aortic nerve (in % of initial activity) during stimulation of sympathetic branch on intra-aortic pressure (B). In A: Curves from top to bottom show integral activity of aortic nerve; electroneurogram of aortic nerve; mean intra-aortic pressure (in mm Hg). Time marker 1 sec. Arrow indicates beginning of stimulation of sympathetic branch. In B: 1) results obtained in experiments on intact animals; 2) difference between curves shown in Fig. 3.

grator 5. The pulse produced by the stimulator 1 was led to the electronic key, which closed the integrator input for a period equal to the duration of the pulse (1.2 msec). The leading edge of the pulse triggered the stimulator 2, which formed the stimulating pulse after a delay $\tau=0.1$ msec. At the moment of application of the stimulating pulse to the sympathetic nerve, the signal from the aortic nerve had thus not reached the input of the integrator. The stimulator 1 worked throughout the experiment, whereas stimulator 2 worked during the period of stimulation. Consequently, the time during which the integrator accumulated aortic nerve activity in each integration period was the same during or without stimulation of the sympathetic branch, so that the effect of excitation of the sympathetic nerve on activity in the aortic nerve could be determined quantitatively. The pressure in the aorta was raised by gradual occlusion.

The experiments of series II were carried out on an isolated preparation of the aortic arch of cats with an intact aortic nerve and stellate ganglion (Fig. 1B). The preparation was perfused with blood from a donor with the blood supply to the stellate ganglion intact. The experimental method was such that it was possible to disconnect the preparation from the donor and to create various levels of constant pressure in it by altering the vertical height of a reservoir with mercury. Activity of the aortic nerve was recorded and the branch from the stellate ganglion stimulated just as in the experiments of series I (Fig. 1A). The conditions of this method were such that the aortic baroreceptors and stellate ganglion were preserved intact for a long time and the effect of stimulation of the sympathetic branch on the activity of the aortic baroreceptors could be studied over a wide range of intra-aortic pressures. All the indices studied were recorded on the Mingograph-1600 apparatus.

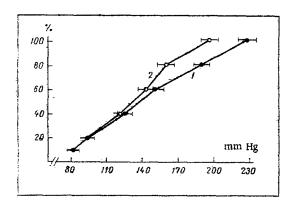


Fig. 3. Activity $(M \pm m)$ of aortic nerve (in % of maximal) as a function of pressure in aortic preparation during (1) and without (2) stimulation of sympathetic branch. Abscissa, pressure in aortic preparation (in mm Hg); ordinate, activity in aortic nerve (in % of maximal).

EXPERIMENTAL RESULTS

A typical record obtained in the experiments of series I is shown in Fig. 2A. Clearly stimulation of the sympathetic branch appreciably reduced baroreceptor activity. The arterial pressure remained unchanged. The results of ten experiments of this series are shown in Fig. 2B. In each experiment the effect of stimulation of the sympathetic branch on baroreceptor activity was determined at the arterial pressure which existed in that animal. In cases when a constant increase in pressure in the region of the arch of the aorta could be obtained by partial occlusion of the descending aorta, the sympathetic branch was stimulated at this new level of pressure also. As Fig. 2B shows, the decrease in activity of the aortic nerve depended on the level of the arterial pressure. With a pressure below 140 mm Hg or above 240 mm Hg, stimulation of the sympathetic branch had no effect on baroreceptor activity. The maximal effect was observed when the pressure was 170-180 mm Hg.

To determine the effect of the sympathetic nervous system on baroreceptor activity at all levels of intraaortic pressure, experiments were carried out on an isolated preparation of the aortic arch. Activity in the
aortic nerve was recorded as the pressure in the preparation was raised from 0 to 280 mm Hg without and
during stimulation of the sympathetic branch. The mean results of ten experiments of this series are illustrated in Fig. 3. Clearly stimulation of the sympathetic branch did not change the threshold pressure for excitation of the baroreceptors (80 mm Hg) or their activity within the range from 80 to 140 mm Hg. With a higher
intra-aortic pressure stimulation of the sympathetic branch reduced baroreceptor activity, as a result of which
the curve of baroreceptor activity as a function of intra-aortic pressure was more sloping in character and
reached maximal activity of the aortic baroreceptors at a higher intra-aortic pressure (on average, 32 mm Hg
higher). The results of the experiments on the aortic arch preparation thus coincided with those of experiments on the intact animals (Fig. 2B).

By what mechanism do sympathetic efferent endings act on baroreceptor sensitivity? Morphological and electron-microscopic investigations [1, 13, 14] have shown that sympathetic nerve endings are distributed mainly on the smooth muscle cells of the baroreceptor zones. Bagshaw and Peterson [4] found that stimulation of the sympathetic branch innervating the carotid sinus reduces its diameter, i.e., reduces the extensibility of the carotid sinus. This effect was most marked when the arterial pressure was high. Evidently in the region of low and average pressure the extensibility of the baroreceptor zones is determined mainly by the elastic properties of its elastic cells, but at a high pressure the tone of the smooth muscle cells also plays a substantial role. In that case contraction of the muscle cells of the zone in response to excitation of sympathetic nerve fibers reduces its extensibility, chiefly at a high pressure. Since the baroreceptors are stretch receptors, a decrease in the extensibility of the baroreceptor zone leads to a decrease in baroreceptor activity and an increase in the intra-aortic pressure at which baroreceptor activity is maximal. This is what was found in the present experiments also. However, in some investigations [8, 15], some increase in baroreceptor activity of the carotid sinus was observed during stimulation of sympathetic efferent fibers, which does not agree with the results of the present experiments. How can this difference be explained? Analysis of the works cited above shows that their authors observed an increase mainly in low-amplitude activity, i.e., activity in unmyelinated fibers. Activity of receptors with unmyelinated fibers located in the heart and on the blood vessels as a rule is

known to be irregular, and it increases substantially and is grouped into volleys synchronous with the cardiac rhythm only if the pressure (degree of stretching) is considerable [12]. In that case, a certain decrease in the extensibility of the baroreceptor zone under the influence of stimulation of the sympathetic branch could have virtually no effect on activity in the unmyelinated fibers. Meanwhile, catecholamines liberated in sympathetic nerve endings can sensitize receptors with unmyelinated fibers. It has in fact been shown that the action of adrenalin and noradrenalin on the baroreceptor zone causes an increase of activity mainly in unmyelinated fibers from baroreceptors [2, 9, 10]. In the present experiments the activity of the whole trunk of the aortic nerve was recorded, i.e., chiefly high-amplitude activity in myelinated fibers, and a change in the activity in the unmyelinated fibers could not be detected under those conditions.

What is the physiological role of the influence of sympathetic efferent fibers on baroreceptor activity? The experimental evidence suggests that the role of vascular baroreceptors in myelinated and unmyelinated fibers is different. For instance, in experiments with separate electrical stimulation of myelinated and unmyelinated fibers of the aortic nerve it was shown that excitation of the unmyelinated fibers causes a far stronger and longer decrease in the arterial pressure [5]. On the other hand, reflex responses of the circulation to small but rapid fluctuations in the arterial pressure level are caused chiefly by excitation of baroreceptors with myelinated fibers [3]. These results and those obtained in the present experiments suggest that in all states of the body accompanied by excitation of the sympathetic nervous system (physical exertion, emotion, stress), under its influence the baroreceptors with unmyelinated fibers are sensitized, thus restraining a rapid rise in the arterial pressure level. At the same time, baroreceptors with myelinated fibers are tuned to a higher blood pressure level and the dynamic range of their function is broadened.

LITERATURE CITED

- 1. E. B. Khaisman, Aortic Baroreceptors [in Russian], Moscow (1966).
- 2. H. Aars, Acta Physiol. Scand., 83, 335 (1971).
- 3. H. Aars and L. Myhre, Acta Physiol. Scand., 91, 43A (1974).
- 4. R. J. Bagshaw and L. H. Peterson, Am. J. Physiol., 212, 1462 (1967).
- 5. W. W. Douglas, J. M. Richie, and W. Schaumann, J. Physiol. (London), 132, 187 (1956).
- 6. W. Floyd and E. Neil, Arch. Int. Pharmacodyn., 91, 230 (1952).
- 7. I. C. Keith, C. Kidd, C. M. Malpus, et al., J. Physiol. (London), 238, 61P (1974).
- 8. K. Koizumi and A. Sato, Am. J. Physiol., 216, 321 (1969).
- 9. S. Landgren, Acta Physiol. Scand., 26, 35 (1952).
- 10. S. Landgren, E. Neil, and Y. Zotterman, Acta Physiol. Scand., 25, 24 (1954).
- 11. A. Moncada and A. M. Scher, Circulation, 28, 771 (1963).
- 12. B. Oberg and P. Thoren, Acta Physiol. Scand., 85, 145 (1972).
- 13. P. M. Rees, J. Physiol. (London), 193, 245 (1967).
- 14. D. J. Rees and K. Fuxe, Am. J. Physiol., 215, 1054 (1968).
- 15. S. R. Sampson and E. Mills, Am. J. Physiol., 218, 1650 (1970).