Cardiac Pressoreceptors in Rat

The existence of cardiac pressoreceptors has been described by numerous authors in different animal species[1]. LEDSOME and LINDEN[2,3] described their existence at the pulmonary vein and left atrium junction, stating that they responded to distention with tachycardia and higher blood pressure. KARIM et al.[4] observed that this effect occurred through an increase of the sympathetic activity of the cardiac nerves.

On the other hand, the existence of receptors in the left ventricle responding to distention with bradycardia[5,6] has also been postulated, the vagus nerve being the afferent tract and the sympathetic nerve the efferent one[7].

Cardiac distention was carried out to the effect of studying this reflex, through the occlusion of the ascending aorta in different groups of rats; heart rate modifications were measured.

Material and methods. Wistar rats weighing about 250 g were used. The animals were anesthetized with 35 mg/kg pentobarbital and kept with controlled breathing by means of a Harvard breathing pump (Model 680). Ventilation took place through room temperature air. Once the aortic and carotid pressoreceptor nerves were incised, the ascending aorta was identified and occluded from 5 to 7 sec. Heart rate was obtained through electrocardiographic tracing prior to and during the occlusion.

Five groups of 10 animals each were used: a) normal rats; b) vagotomized rats; c) rats with destroyed medulla oblongata; d) atropine-treated rats; e) alprenolol-treated rats. The drugs were given through endovenous injection in the following doses: 1.5 mg/kg atropine and 1 mg/kg alprenolol, 15 min prior to aortic occlusion.

In some animals, left intraventricular pressure was measured by means of a catheter placed through the ventricular wall, using a mercury manometer, and central venous pressure with a catheter placed in the superior vena cava, through the jugular vein, with a water manometer. Results are stated as mean $\pm$ E. S.

Results. The results obtained are to be found in the Table. In 24 experiments on 10 animals, the occlusion of the ascending aorta significantly decreased the heart rate ($p < 0.005$). Incision of the vagus nerves inhibited the bradycardia obtained in 28 tests carried out on 10 animals. Destruction of the medulla oblongata prevented the modifications in the heart rate obtained through aortic occlusion in 38 experiments. In 44 tests carried out with 10 atropine-treated rats, aortic occlusion significantly decreased the heart rate ($p < 0.015$) while in 36 experiments with alprenolol-treated animals there were no heart rate modifications.

Intraventricular pressure increased 87% $\pm$ 10 by means of aortic occlusion, whereas there were no pressure modifications in the superior vena cava. Integrity of the aortic and/or carotid pressoreceptor nerves prevented the heart rate modifications that took place during aortic occlusion.

Effects of the ascending aortic occlusion on the heart rate

	Control	Occlusion	Δ
Normal rats N:24	339 $\pm$ 6	304 $\pm$ 8	35[a]
Vagotomized rats N:28	329 $\pm$ 5	332 $\pm$ 5	3
Rats with destroyed medulla N:38	309 $\pm$ 8	304 $\pm$ 7	5
Atropine treated rats N:44	322 $\pm$ 5	304 $\pm$ 5	18[b]
Alprenolol treated rats N:36	242 $\pm$ 6	242 $\pm$ 6	0

N, number of experiments. [a] $p < 0.005$. [b] $p < 0.015$.

Discussion. The bradycardia obtained through aortic occlusion indicates that there are receptors in the heart or in the pulmonary circuit responding to a distention increase with a heart rate decrease. The fact that this reflex disappears by means of vagotomy but is not modified when atropine is given, would indicate that the afferent tract is to be found in the vagus nerve. Annulment of this reflex in animals with destroyed medulla oblongata would indicate that the center or some neuronal connection is to be found at that level. Suppression of the bradycardia obtained by means of aortic occlusion, through the use of alprenolol (β-blocking agent), would prove that the efferent tract would work via the sympathetic nervous system.

Most probably receptors are to be found in the left ventricle, since central venous pressure is not modified and receptors placed in the left atrium respond to distention with tachycardia [2,3]; this would agree with the findings of AVIADO and SCHMIDT[1] who think that the most feasible localization of cardiac receptors would be in the left ventricle and in the right atrium, although they may also be found in coronaries and pulmonaries arteries or in the right ventricle[8,9]. The fact that this reflex is not present in some of the experimental groups excludes the possibility of its being due to ischemic alterations of nervous centers or to modifications of the gas contents in blood. The contradictory results found by KLUSSMAN et al.[10] might arise from the fact that in those experiments the pressoreceptor nerves had not been incised.

The function of this reflex under normal conditions seems to be slight since it does not appear in the presence of the aortic and/or carotid pressoreceptor nerves, although it is not possible to exclude that they perform a more important function under certain pathological conditions such as high blood pressure or cardiac distention.

Resumen. Se describe la probable existencia de presorreceptores en el ventriculo izquierdo de la rata. La vía aferente de este reflejo sería el vago y la eferente el simpático. El centro o alguna conexión neuronal estaría a nivel del bulbo raquídeo.

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