in the cerebrum, cerebellum, optic lobes and medulla oblongata on in vivo administration of alloxan (Table). Likewise, the levels of ribonucleic acid in different regions of rat brain studied also decreased considerably on alloxanization (Table). The protein content was higher in the cerebral region compared with other regions of the brain in both normal and diabetic rats. However, decrease in the protein content was highest in the brain stem of diabetic rats (Table). On the other hand, the level of ribonucleic acid was higher in the medulla compared to the other regions of the brain. Paralleling the change in protein content, medulla showed marked response for the changes in the levels of ribonucleic acid during diabetes (Table). This differential response of the different regions of the brain is in relation to the differential functional status of the broad compartements of the brain.

Discussion. The decrease in the levels of protein and ribonucleic acid in different regions of the brain of alloxan-diabetic rats appears to be the direct effect of insulin deficiency caused by alloxanization. It has also been shown earlier that insulin deficiency during diabetes inhibits protein synthesis and accelerates the catabolism of amino acids[15]. It has been suggested that the deceleration in the incorporation of amino acids into proteins is one of the major reasons for the decreased protein synthesis observed during diabetes[15]. Supporting this, the free amino acid content in the brain of diabetic rats has been found to be considerably high (JAYASHREE, unpublished observations). However, PETERSON et al.[7] report an increase in the rate of protein synthesis during diabetes in rats.

The decrease in the levels of ribonucleic acid in different regions of the brain of rat during alloxan-diabetes is in accordance with the decrease in total protein content of the different regions on alloxanization. This, therefore, indicates that the entire protein synthetic machinery is disrupted during diabetes.

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Cardiac Pressoreceptors and Peripheral Resistance

A. MARTÍNEZ SEEBER

Orientación Fisiologia Humana, Departamento de Ciencias Biológicas, Facultad de Farmacia y Bioquimica – U. B. A., Junin 956, Buenos Aires (Argentina), 17 May 1976.

Summary. The results obtained show that the pressoreceptors, probably ubicated in the left ventricle of the rat, respond to the distention with vasodilatation. The afferent tract of this reflex is in the vagus nerve and the efferent one is in the sympathetic nervous system. The probable function of this reflex is discussed.

Previously we have described the effect of the cardiac pressoreceptors on the regulation of the cardiac rate in the rat[1]. To produce the activation of the pressoreceptors, the ascending aorta was occluded and this produces cardiac distention. We have also observed that it is necessary to excise the aortic and carotid pressoreceptor nerves to obtain the reflex.

Effects of the ascending aortic occlusion on the aortic perfusion pressure

	Control pressure (mm Hg)	δ (mm Hg)
Control rats ($n = 11$)	73±10	+0.7±0.4
Rats without aortic and carotid nerves ($n = 14$)	123±12	− 8.2±2.4[a]
Atropine treated rats ($n = 10$)	83±10	− 5.3±1.4[b]
Vagotomized rats ($n = 9$)	127±13	0 ±0.2
Phentolamine treated rats ($n = 14$)	48± 6	+0.1±0.1

n, number of experiments. [a] $p < 0.005$. [b] $p < 0.001$.

The purpose of this work was to study the effect of the cardiac pressoreceptors on the peripheral resistance and to describe the different pathway of the reflex.

Methods. Wistar rats weighing about 200 g were used. The animals were anesthetized with 1 g/kg urethane and kept with controlled breathing by means of a Harvard breathing pump (Model 680). The abdominal aorta was exposed by a midline incission and cannulated in a peripheral direction below the renal arteries, and the carotid artery also was cannulated in a central direction. Blood was pumped from the carotid cannula to the hind quarters by a pump with a flow of 0.7 ml/min. Blood obtained from donor animals was used to fill the tubing. Perfusion pressure was recorded from T tube on a poligraph via a Statham P23AA pressure transducer. The modifications in the perfusion pressure were considered representative of changes in the peripheral resistance.

The ascending aorta was identified and occluded about 10 sec and the perfusion pressure was recorded continuously. 5 groups of rats were used: a) control rats; b) rats with carotid and aortic nerves excised; c) vagotomized

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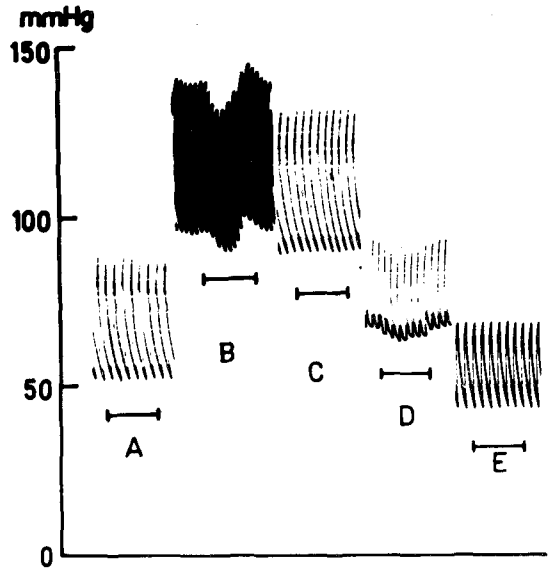
rats; d) atropine-treated rats; e) phentolamine-treated rats. Except the control animals, all the other groups had the carotid and aortic nerves excised. The drugs were given through the jugular vein in the following doses: 1.5 mg/kg atropine and 5 mg/kg phentolamine, 15 min prior to aortic occlusion. Results are stated as mean ± SE and compared with those obtained in control animals.



A) Control animal. B) Animal without aortic and carotid nerves. C) Vagotomized animal. D) Atropine treated animal. E) Phentolamine-treated animal. Segment: period of aortic occlusion.

Results. The results obtained are to be found in the Table and one experiment of each group is shown in the Figure. The cardiac distention in 11 tests carried out on 6 control animals does not show modifications in the perfusion pressure of the aorta. In 14 experiments on 7 animals without carotid and aortic pressoreceptor nerves, the occlusion of the ascending aorta produced a significant decrease in the perfusion pressure.

Similar results were obtained in 10 experiments carried out in 6 atropine-treated rats. Incision of the vagus nerve inhibited the vasodilatation obtained in 9 tests done in 6 rats and the same inhibition was reached in 14 experiments made in 6 animals treated with phentolamine.

Discussion. The decrease in the perfussion pressure obtained through aortic occlusion indicates that there are receptors in the heart, probably in the left ventricle[1,2], that respond to the distention with vasodilatation. The persistence of the reflex in atropine-treated animals, and the inhibition obtained by means of the vagotomy, would indicate that the afferent tract is to be found in the vagus nerve. The suppression of the vasodilatation obtained with phentolamine makes it possible to think that the efferent tract is the sympathetic nervous system.

The absence of this reflex in the presence of the aortic and/or carotid pressoreceptor nerves would indicate that its function, under normal conditions, is less important than the carotid or aortic reflexes. Similar conclusions were reached by OBERG and THORÉN in the cat[3], although it is possible that this reflex has a more important function in the regulation of blood pressure in the hypertensive state[4].

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Ox-Spleen Erythropoietic Factor: Chromatographic Investigations and Dosages

P. DE FRANCISCIS, A. M. GRECO, S. BERTUGLIA and G. CASTALDO TUCCILLO

Istituto di Fisiologia Umana dell'Università, Facolta di Medicina e Chirurgia, Via Sergio Pansini, I-80131 Napoli (Italy), 12 April 1976.

Summary. The constant proteic fraction, obtained from ox-spleen homogenates by chromatographic elutions, is the main seat of erythropoietic activity in our assay with polycythemic mice.

In a preceding work[1], we reported a preliminary purification method for the erythropoietic factor from ox-spleen, while in this study we report the results of further investigations of the incoagulable proteins present in the filtrate 'D', obtained with our purification method[1], and their probable correlations with the erythropoietic effect elicited after injection in polycythemic mice.

Materials and methods. The preliminary purification method reported[1] was used for spleens removed from 3 calves (about 12 months old) immediately after death of the animal. The obtained filtrates D_1, D_2, D_3 were tested after dialysis against tap-water at 4 °C and lyophilization for their UV-absorption (proteic concentration 100 µg/ml in 0.033 *M* phosphate buffer pH 6.3) and then analyzed by elution on Sephadex DEAE-A 50 using 0.033 *M* phosphate buffer pH 6.3 in continuous gradient salt to 1.0 *M* NaCl. The swollen-equilibrated Sephadex was packed in a column of 1.5 × 50 cm at 4 °C under an operating pressure of about 1.0 cm H_2O/cm height of bed. The materials, before and after chromatographic elution, were assayed for their erythropoietic activity employing CF/1 strain female mice, 8–10 weeks of age, made polycythemic by discontinuous hypoxia according to FISHER's method[2]. The s.c. injections of the substances were made on the 4th and 5th day following the hypobaric chamber treatment and the erythropoietic increase was determined, whether using ^{59}Fe or by reticulocyte calculation. In the first case, 0.5 µCi of ^{59}Fe was injected i.v. 24 h after the material administration; 48 h later each mouse was bled via cardiac puncture, microhematocrits and haemoglobin percentage were determined (microhematocrits = 74 ± 1.38; haemoglobin = 19%) and 1.0 ml of blood counted for calculation of percent RBC ^{59}Fe incorporation in red cells. We treated 8 groups of polycythemic mice (10 mice

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