Oxygen consumption was determined on individual rats on several occasions following an overnight fast. A closed circuit technique similar to that described by MACLAGEN and SHEAHAN[6,7] was used and the data are expressed in terms of body weight and of surface area (body weight)$^{0.7}$.

Results. Oxygen consumption was measured in 2 experiments (Table). In the first, the genetically obese rats weighed significantly more than their lean littermates but the lean littermates consumed significantly more oxygen per g of body weight. When these uptakes of oxygen were corrected for surface area (body weight)$^{0.7}$ the difference was not statistically significant ($t = 1.8$). In the second experiment the oxygen consumption of 3 genetically obese rats was compared with that of the 3 littermates which became fat following hypothalamic lesions. In this experiment the body weight of the genetically obese rats was not significantly different than their obese lesioned littermates. However, oxygen consumption per g of body weight, or per unit surface area, was significantly higher in the lesioned obese rats.

Comment. The genetic obese rats and their lean littermates consumed nearly identical quantities of oxygen per unit of surface area. However, when the lean littermates became obese following hypothalamic lesions, they consumed significantly more oxygen than their genetically obese siblings. In this respect the Zucker rats are similar to the obese yellow (A^y) and the obese hyperglycemic mice (obob) in which hypometabolism has been noted[1,2] but they differ from the New Zealand obese mice[8] which consume normal or increased quantities of oxygen when compared to lean littermates[9,10].

Zusammenfassung. Der Sauerstoffverbrauch genetisch fettleibiger Ratten ist auffallend niedriger als derjenige von Normalratten (littermates), die nach Hypothalamusverletzung fettleibig wurden.

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[11] The author thanks Dr. E. B. ASTWOOD for his continuing support and encouragement.

Hemodynamic Changes in Rats Without Adrenal Medula Submitted to Hypovolemic Shock[1]

As reported previously, rats without adrenal medulla had a longer survival time than normal controls, when submitted to irreversible hypovolemic shock[2]. Furthermore, continuous i.v. injection of epinephrine decreased survival time while norepinephrine continuous injection increased survival time in rats with irreversible hypovolemic shock[2]. It is well known that the autonomic nervous system plays a role in the regulation of circulatory function and that the medullary portion of the adrenal gland secretes mainly epinephrine, while the sympathetic nerve endings liberate mainly norepinephrine[3,4]. In consequence it seemed interesting to study the hemodynamic changes induced by irreversible hypovolemic shock in rats without adrenal medulla.

Material and methods. Studies were carried on 42 rats from the strain bred at the 'Instituto de Fisiología'. They had free access to water and were fed with the usual mixed diet of the Institute. All animals were anesthetized i.p. with sodium pentobarbital (4 mg/100 g body wt.). Hypovolemic shock was induced by permanent occlusion of the portal vein, as described previously[5]; sham occlusion of the portal vein consisted in dissection of the vessel and placing a loose thread around it, without actual occlusion of the vein. Adrenal medullectomy and sham adrenal medullectomy were done following the technique reported previousley by us[2]. Blood volume, cardiac output and mean arterial blood pressure were measured in each animal 45 min after occlusion (or sham occlusion) of the portal vein, but no studies were carried out before 4 weeks had elapsed from the adrenal medullectomy or sham adrenal medullectomy. Blood volume and cardiac output were measured by dilution of radioiodinated human serum albumin labelled with Iodine-131, as described elsewhere[6,7]. Mean arterial blood pressure was measured with a critically damped mercury manometer connected with the right carotid artery; total peripheral resistance was calculated from cardiac output and mean arterial blood pressure and expressed in dyn. sec. $cm^{-5} \times 10^{-4}$ per 100 g body wt.

Four groups of animals were studied:

(A) Normal controls, 10 rats (sham adrenal medullectomy and sham occlusion of the portal vein). (B) Hypovolemic shock, 10 rats (sham adrenal medullectomy plus occlusion of the portal vein). (C) Medullectomized controls, 12 rats (adrenal medullectomy and sham occlusion of the portal vein). (D) Hypovolemic shock in medullectomized rats: 10 animals (adrenal medullectomy plus occlusion of the portal vein). Results obtained were expressed as mean $\pm$ S.E.; the significance of the differences was assessed with Student's *t*-test, as suggested by BANCROFT[8].

Results. The statistical analysis of the data obtained (Table) showed no difference between normal controls (group A) and medullectomized controls (group C). In relation to control groups, hypovolemic shock provoked a sharp decrease ($p < 0.001$) in blood volume, cardiac output and mean arterial blood pressure, either in sham medullectomized (group B) or in medullectomized rats

[1] This work has been partially supported with a grant from the 'Consejo Nacional de Investigaciones Científicas y Técnicas'. The isotopes were obtained through the 'Comisión Nacional de Energía Atómica'.
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Hemodynamic changes in normal and medullectomized rats during irreversible hypovolemic shock (for group references see text)

Group	No. of cases	Mean body weight (g)	Blood volume ml/100 g body wt.	Cardiac output ml/min per 100 g	Mean arterial blood pressure mm Hg	Total peripheral resistance $\frac{dyn/sec/cm^{-5}/10^{-4}}{100\ g\ body\ wt.}$
A	10	259	6.8 ± 0.3	35.5 ± 4.0	110.7 ± 5.0	24.9 ± 4.0
B	10	246	3.8 ± 0.3	7.9 ± 1.4	20.3 ± 2.9	20.6 ± 0.7
C	12	182	7.2 ± 0.3	33.2 ± 2.5	101.2 ± 3.1	25.6 ± 1.8
D	10	251	3.2 ± 0.2	3.1 ± 0.2	36.7 ± 4.0	98.0 ± 13.0

Mean $\pm$ S.E.

(group D); total peripheral resistance remained unchanged in group B, and in group D increased significatively ($p < 0.001$). Comparison between group B (sham adrenal medullectomy plus hypovolemic shock) and group D (adrenal medullectomy plus hypovolemic shock) showed a significant difference in cardiac output ($p < 0.01$) which was higher in group B, and in mean arterial blood pressure ($p < 0.01$) which was higher in group D; consequently total peripheral resistance was higher in medullectomized rats than in rats with adrenal medulla, during hypovolemic shock ($p < 0.001$).

Discussion. After the original description of HOUSSAY and LEWIS[9] on the survival of dogs without adrenal medulla. LASCANO GONZALEZ[10] observed that rats with total adrenalectomy, if kept with saline solution in their drinking water, not only survived but developed new cortical tissue. This new cortical tissue was fully developed in 4 weeks, and these animals were considered as animals without adrenal medulla, with normal function of adrenal cortex[2]. There are no observations on the hemodynamic pattern of rats without adrenal medulla, except a report of BRODY[11] who did not find changes in basal heart rate and arterial blood pressure in rats after immunological sympathectomy.

In medullectomized dogs, a decrease has been observed in basal cardiac output, heart rate, stroke volume and arterial blood pressure with an increase in total peripheral resistance[12–14]; if sympathectomy is added the same pattern is present, but there are no changes in total peripheral resistance[15]. In rabbits KORNER and WHITE[16] did not observe any changes in cardiac output, arterial blood pressure and heart rate after medullectomy, either at basal condition or during hypoxia.

As postulated by FOLKOW[4] and CELANDER[3], the adrenal medulla is the main source of epinephrine in mammals while nerve endings liberate mainly norepinephrine. It can be deduced from their studies that medullectomized animals have a very scarce output of epinephrine, and HARRISON and SEATON[17] observed in medullectomized dogs that the tissue storage of epinephrine is almost depleted.

From the results reported in this paper and those previously reported[2] it can be stated that medullectomized rats submitted to irreversible hypovolemic shock have a longer survival time, a lower cardiac output and a higher blood pressure than control rats during shock; these differences can be attributed to a decreased output of epinephrine, since the administration of epinephrine to normal rats undergoing shock induced a shorter survival. Since a more prolonged survival time in this shock model can be obtained not only with adrenal medullectomy but also with continuous injection of norepinephrine[2], it seems that survival time may be related to the ratio norepinephrine/epinephrine available, instead of being related to the available amounts of each cathecolamine separately. However there is not enough experimental evidence to support this hypothesis and more work should be done to clarify this point.

Resumen. La extirpacion de la medula suprarrenal no provoca alteraciones de la hemodinamia en ratas estudiadas en condiciones basales, mientras que durante el shock hipovolemico las ratas meduloprivas presentan un volumen minuto cardiaco menor, y un aumento de la presion arterial media y de la resistencia periferica total, en relacion a los testigos normales sometidos al mismo tipo de shock.

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Demonstration of Post-Hypophysectomy Changes in the Neurosecretory System of a Fish with in situ Staining Technique

There are few instances where bulk staining techniques were adopted to study the topographic pattern of the hypothalamo-neurohypophysial (HN) complex in vertebrates[1–8]. However, experimental studies using in situ staining techniques are wanted. In this study wild-caught Indian freshwater catfish *Rita rita* (Ham) is used. Brains were fixed in Bouin's fluid and stained in Gabe's aldehyde fuchsin (AF) with modifications. The normal HN complex has a paired stainable nucleus preopticus (*npo*) situated on either side of the third ventricle. The *npo* gives rise to the left and the right main tracts. Several pairs of lateral tracts separate off from the main