weight[4]. (2) Erythropoiesis has been shown to be a function of the thymus of healthy mice[5,6]. (3) An increase in dark staining cells was found in the red pulp of spleens from surviving foster-nursed C57BL female mice (decreased survival) but not in spleens from foster-nursed PL mice (unchanged survival) when compared to non-foster-nursed controls[7]. Although these cells were previously considered to be lymphocytes, recent electron microscope studies suggest that they are erythroid[8]. These observations suggest a relationship between the function of the erythropoietic system and infant survival.

Zusammenfassung. Das Überleben der von der eigenen Mutter und von stammesfremden «Ammen» gesäugten Jungen wurde untersucht an Mäusen der Stämme AKR/Sp, PL/Sp und C57BL/Sp. Die Ergebnisse zeigen, dass die genetische Herkunft der Säuglinge wie auch andere, nicht genetisch bedingte Einflüsse der Mutter auf die Jungen in utero für das Überleben der Jungen ausschlaggebend ist.

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Further Observations of the Angiotensin Vasopressive Effect in Rabbits

Certain sympathomimetic amines[1] and digitalis[2] seem to require the presence of catecholamines in the tissue storage sites to exert their characteristic action on the cardiovascular system. It is possible that the action of other cardiovascular agents, such as angiotensin, also depend at least in part on catecholamine activity. For this reason, a study was performed on rabbits exploring the possible role of catecholamines in vasopressor action of angiotensin.

Material and Methods. 16 male rabbits weighing between 2–4 kg were anesthetized with intraperitoneal injections of pentobarbital (35 mg/kg). The blood pressure was recorded from the common carotid artery by means of an arterial catheter connected to a suitable pressure transducer, the output of which was recorded on a Sanborn polygraph. Angiotensin was injected through a catheter in the external jugular vein. The injections were rapid and were washed in with 2 cm^3 of saline.

Doses of angiotensin (Ciba, Hypertensin)[3] producing small (10–20 mm Hg), medium (30–40 mm Hg), and large (60–80 mm Hg) elevations of mean blood pressure were determined. Since angiotensin exhibits tachyphylaxis[4], especially in high doses, care was taken in selecting an interval of injection (4–5 min) which did not show this phenomenon. The order of injections was arranged by a 3×3 latin square. Each dose-response curve required 45 min to determine and was followed immediately by a second and a third determination in the same animal. In this way the influence of time and previous injections on the reactivity to angiotensin was assessed.

To determine the response to angiotensin in a setting in which adrenergic nervous activity was reduced, β-TM 10 ([2-(26)-dimethylphenoxypropyl]trimethyl ammonium chloride)[5], an adrenergic neuronal blocking agent[6], was employed. β-TM 10 was rapidly injected into the ear vein in doses of 5 mg/kg, approximately 3–4 h before the start of the experiment. After each mean the 95% confidence limits are indicated.

Results. By a series of trials it was found that 0.1, 0.5, and 2.5 mg/kg of angiotensin produced the desired responses; namely, small, medium and large elevations of mean blood pressure (Table I). The data are summarized in Table I for each series of injections. It is evident from the data presented that there is no difference in the responses between each series. Thus 0.1, 0.5, and 2.5 mg/kg of angiotensin produced quantitatively similar increases in blood pressure in each group ($p > 0.05$). After β-TM 10 the reactivity to angiotensin was not altered. Thus the response to angiotensin (Table II) was not significantly different to the responses observed at the same doses in animals not treated with β-TM 10 (Table I).

Table I. Dose-response relationship of the angiotensin effect on blood pressure

Series of injections	Number of experiments	Average number of injections	Dose of angiotensin (mg/kg)	Average increase in blood pressure (mm Hg	CL[a])
1st	15	3	0.1	19.0	3.2
	15	3	0.5	38.6	5.0
	15	3	2.5	73.0	5.8
2nd	8	3	0.1	18.1	4.0
	8	3	0.5	36.9	9.8
	8	3	2.5	71.5	11.0
3rd	6	3	0.1	15.3	3.2
	6	3	0.5	29.0	7.0
	6	3	2.5	66.7	11.6

[a] CL = 95% confidence limit.

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Another interesting observation was the finding that in 7 out of 21 experiments following the pressor effect produced by 0.5 mg/kg of angiotensin a persistent hypotension developed ranging between 15–46 mm Hg. Similarly in 10 out of 20 experiments following the pressor effect produced by 2.5 mg/kg a hypotension developed ranging between 16–42 mm Hg. In 5 of these animals death ensued shortly after the onset of hypotension.

Discussion. The results of this investigation clearly show that the prevention, by β-TM 10, of catecholamine release from adrenergic nerve terminals did not in any way influence the blood pressure response to angiotensin. The hypertensive effect of angiotensin is probably due to a direct action on the cardiovascular system[7] and is not mediated by neurotransmitters. A similar conclusion was reached by SAKURAI and HASHIMOTO[8], using reserpine to deplete the tissues of catecholamines.

The secondary hypotensive effects following the use of angiotensin deserves further study since it occurred in many of the animals. It is probably not due to a sympathetic blockade by angiotensin, since, if anything, angiotensin has been shown to enhance blood pressure responses to norepinephrin[8,9].

Table II. Dose-response relationship of the angiotensin effect on blood pressure after β-TM 10

Number of experiments	Average number of injections	Dose of angiotensin (mg/kg)	Average increase in blood pressure (mm Hg	CL[a])
5	2	0.1	22.6	3.8
5	2	0.5	43.4	4.4
5	2	2.5	73.0	12.0

[a] CL = 95% confidence limit.

Résumé. On n'a pas pu observer un effet du β-TM 10 sur l'action de l'angiotensine chez le lapin. Cela indique probablement que l'angiotensine a directement son effet sur les cellules du récepteur. Après l'élévation de pression produite par les doses plus grandes de l'angiotensine, on a pu remarquer une réduction significative de la pression.

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Über die Wirkung von Diäthyläther auf die Aktivität der Glykogenphosphorylase und Phosphorylase *b*-Kinase in Herz, Skeletmuskel und Leber der Ratte

Die Diäthyläther(Äther)-Narkose führt beim Menschen und bei einer Reihe von Versuchstieren zu einer Beeinflussung des Kohlehydratstoffwechsels, die wahrscheinlich mit einer durch das Narkotikum ausgelösten, erhöhten Aktivität des sympathischen Nervensystems in unmittelbarem Zusammenhang steht[1]. In der vorliegenden Untersuchung wurde anhand des Phosphorylase-Systems, das sehr empfindlich auf Veränderungen der Aktivität des Sympathikus zu reagieren vermag[2], der Einfluss von Ätherinhalation näher analysiert.

Material und Methoden. Erwachsenen Ratten wurde unter Narkose der Musculus gastrocnemius, ein Stück der Leber und, unter künstlicher Beatmung, das Gesamtherz mittels der Kühlzangentechnik[3] nacheinander in situ eingefroren. Einige Tiere erhielten 20 min vor der Tötung eine intravenöse Injektion von Pronethalol (1(2-naphthyl)-2-isopropylaminoäthanolhydrochlorid; freundlicherweise von J. W. BLACK, Imperial Chemical Industries Ltd., überlassen), einem Blocker der adrenergen β-Rezeptoren, der die phosphorylaseaktivierende Wirkung von injiziertem Adrenalin und von sympathischer Nervenreizung unterdrückt[4].

100–150 mg Portionen der eingefrorenen Muskelgewebe wurden in einem mit flüssiger Luft vorgekühlten Mörser pulverisiert. Das Gewebspulver wurde zur Extraktion mit dem 5fachen Volumen einer 60%igen Glyzerinlösung, die 0,02 *M* NaF, 0,005 *M* Diäthylaminotetraacetat, 0,03 *M* Cystein und 0,04 *M* Glyzerin-2-Phosphat (pH 6,8) enthielt, bei – 35 °C vermischt und anschliessend mit dem 15fachen Volumen der gleichen, wässrigen Salzlösung versetzt[5,6]. Nach der Zentrifugation (10 min bei 7000 *g*) wurden in der überstehenden Flüssigkeit die Fermentaktivitäten bestimmt. Die Glykogenphosphorylase (EC 2.4.1.1) wurde nach ILLINGWORTH und CORI[7] und die Phosphorylase-*b*-Kinase (EC 2.7.1.38) nach KREBS et al.[8] bestimmt. Der Aktivierungsgrad des letztgenannten Fermentes wurde aus dem Verhältnis der gemessenen Fermentaktivitäten bei pH 6,8 und 8,2 errechnet[6]. Die Ex-

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