was dissected. Lastly muscle length and weight were determined. More details of the method may be found in the papers by BINKHORST[1,3].

Results and discussion. There are significant differences between the absolute values of muscle weight and length of male and female rats of the same age and the contraction parameters closely related to these values such as force and velocity. The main reason for these differences is that the male rats are larger and heavier than the females. To study the effect of sex mainly relative values will be used.

In Table I and II the data of female and male rats respectively are presented. The results of the statistical analyses are also included in the Tables. The analysis of variance and the SCHEFFÉ[6] multiple comparison test were done on the subgroups.

Rat weight in the male groups differed significantly: the trained rats have the lowest weight which is in agreement with a recent report[7]. In female rats this difference is not found. This finding might be important for studies of physical activity, caloric intake and obesity, in so far as there are differences in reaction depending on the sex of the rat. Muscle hypertrophy (from muscle weight/rat weight) can be seen in the operated rats: hypertrophy is larger in the operated and trained rats (DT rats).

Maximal speed of contraction per unit muscle length (V_o/l_o) is significantly different only in the CNT-DNT comparison in female as well as male rats: the CNT rats show the highest speed. It could be shown[3] that there is a significant positive correlation betwen V_o/l_o and the cosine of the fibre angle, which is significantly increased in the hypertrophic pennate fibred MP. Is it assumed that the increased angel at least partly offers an explanation for the lower speed in the hypertrophic muscles.

The parameters twitch/tetanus, twitch contraction time, and a/P_o (curve parameter for the force-velocity relation according to Hill's formula) to not show any significant differences. It is conlcuded that the overloaded MP remains fast, which is in agreement with previous findings[1].

The results as presented in the Tables indicate that for the MP there are no differences in reaction to overload and training between the sexes.

Lastly Student's t-test was applied to the data of the CNT, DNT and DT rats to compare the female and male groups. Significant difference were found in the rat weight of all equal subgroups (all $P < 0.005$). For the relative parameters only 2 significant differences were found which may be meaningless taking in account the large number of tests Generally speaking, the female and male rats do not react differently to the applied procedure, except for body weight.

Zusammenfassung. Bei der Ratte wurde durch Denervation des M. gastrocnemius und des M. soleus eine kompensatorische Hypertrophie des M. plantaris bewirkt und die distale Sehne des M. plantaris auf diejenige des M. gastrocnemius transplantiert. Ein Teil der operierten Tiere wurde trainiert, die Muskelgewichte bestimmt und isotonische sowie isometrische Kontraktionen untersucht. Die Muskelhypertrophie war bei den trainierten Ratten am stärksten ausgeprägt.

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Effects of Prostaglandins PGE_1 and $PGF_{2\alpha}$ on Oxygen Consumption, Sodium and Potassium Content of Renal Tissue

Prostaglandin E_1, a hypotensive substance, elicits natriuresis when infused in the kidney. This natriuretic effect is coincident with an increase in renal plasma flow, a decrease in the para-amino hippuric extraction rates, but produces no observable change in glomerular filtration rate and systemic blood pressure[1-3]. Since the natriuretic effect of PGE_1 cannot be explained by an increase in sodium tubular load, the other explanation is that sodium reabsorption is reduced either by the increase in blood flow per se[4,5] or by the action of PGE_1 on tubular cells.

The natural occurrence of prostaglandins in the kidney suggest that they may have a physiological role in the regulation of sodium and water excretion. This possibility led us to study the effect of PGE_1 on oxygen consumption, sodium and potassium content of renal cortical slices of rats to find out whether PGE_1 has a direct effect on the kidney.

Material and methods. Male Wistar rats (150–200 g) were used. The animals were decapitated and both kidneys and the liver removed and cut into slices of 0.3 mm thickness[6]. The slices were kept in Petri dishes with

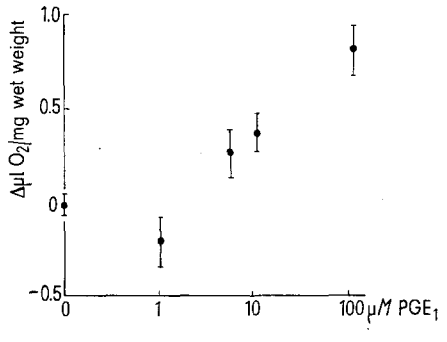

Fig. 1. Relationship between the increment of oxygen consumption ($\Delta\mu l$) and concentration of prostaglandin E_1 (PGE_1) in the medium. Each point represent the mean ± SD.

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Table I. Effects of prostaglandins PGE_1 and $PGF_{2\alpha}$ on oxygen consumption, sodium and potassium content of renal tissue

Prostaglandin	Concentration	O_2 Consumption µl/mg wet wt.	Na Content µEq/g dry wt.	K Content µEq/g dry wt.
Control		4.29 ± 0.29 n:23	368 ± 40 n:10	221 ± 14 n:10
PGE_1	$10^{-4} M$	5.11 ± 0.33 $P < 0.001$ n:8	306 ± 18 $P < 0.005$ n: 7	240 ± 15 $P < 0.025$ n: 7
	$10^{-5} M$	4.62 ± 0.45 $P < 0.005$ n:24	328 ± 21 $P < 0.01$ n:12	233 ± 10 $P < 0.01$ n:12
	$5 \times 10^{-6} M$	4.56 ± 0.33 $P < 0.025$ n: 8	325 ± 18 $P < 0.025$ n: 8	231 ± 11 $P < 0.2$ n: 8
	$10^{-6} M$	4.10 ± 0.23 $P < 0.2$ n: 8	350 ± 11 $P < 0.4$ n: 8	230 ± 9 $P < 0.2$ n: 8
$PGF_{2\alpha}$	$10^{-4} M$	4.59 ± 0.36 $P < 0.025$ n: 8	$310 \pm$ $P < 0.005$ n: 8	238 ± 12 $P < 0.005$ n: 8

Values represent the means $\pm$ S.D. n indicated number of experiments.

Table II. Effect of prostaglandin PGE_1 $10^{-4} M$ on oxygen consumption, sodium and potassium content of hepatic tissue

Prostaglandin	O_2 Consumption µl/mg wet wt.	Na Content µEq/g dry wt.	K Content µEq/g dry wt.
Control	1.14 ± 0.09 n:8	350 ± 36 n:8	246 ± 13 n:8
PGE_1	1.27 ± 0.18 n:8 $P < 0.1$	329 ± 62 n:8 $P > 0.1$	250 ± 26 n:8 $P > 0.5$

Values represent the means $\pm$ S.D. n indicated number of experiments.

balanced saline medium for about 15 min, blotted on filter paper (Carl Schleicher and Schule No 529) weighed on a torsion balance and placed (50–60 mg) in chilled Warburg flasks with 2.8 ml of balanced saline medium[7]. The flasks were gassed with O_2 100% for 2 min. Prostaglandins PGE_1 and $PGF_{2\alpha}$ (obtained from Dr. J. E. PIKE, Upjohn, Michigan) in the salt form were tested at different concentrations: $10^{-4} M$; $10^{-5} M$; 5×10^{-6} and $10^{-6} M$ for PGE_1 and $10^{-4} M$ for $PGF_{2\alpha}$. Following incubation the persistence of prostaglandin in the medium was tested by its action on the rat blood pressure. The slices were incubated for a total of 75 min and the O_2 consumption measured each 15 min during the last 60 min.

Following incubation the dry weight was estimated after drying the slices in an oven for 18 h at 105 °C. Sodium and potassium content were measured by flame photometry after digesting the dry tissue with nitric acid $16 N$[8].

Results. Effect of prostaglandins PGE_1 and $PGF_{2\alpha}$ on renal cortical slices. Table I shows that PGE_1 at doses ranging from 5×10^{-6} to $10^{-4} M$ increased the O_2 consumption of renal cortical slices while simultaneously depressing its sodium content and increasing its potassium, although with the latter only doses of 10^{-5} and $10^{-4} M$ were effective. Similar effects were obtained with $PGF_{2\alpha}$ $10^{-4} M$. There is correlation between the dose of PGE_1 and the increase in O_2 consumption (Figure).

Effect of prostaglandin PGE_1 on hepatic slices. Table II shows that PGE_1 at $10^{-4} M$ increased O_2 consumption but the absolute difference was 0.13 µl O_2/mg moist tissue, so the amount of the increase brought about by PGE_1 is several times greater in the kidney than in the liver. The average sodium and potassium content shows no significant difference. Our results support those of BARRY[9] and JESSUP[10], who showed that PGE_1 increased the sodium movement across frog skin.

The increase in O_2 consumption coincident with a reduction in the sodium content of the slices and increase in its potassium seems to indicate a stimulation of the sodium pump in renal cortical slices[11]. The O_2 consumption of the kidney in situ is increased when more sodium is being reabsorbed[12]. Since the renal cortical slices contain mostly proximal and distal convoluted tubules[13] PGE_1 most probably exerts its action on the cells of the proximal or the distal tubules or in both.

Under physiological conditions, tubular cells pump sodium from the lumen to the interstitial space. There is no evidence that cells can transport sodium actively in the reverse direction. If the cells maintain their polarity in vitro, the effects of PGE_1 on renal slices would mean that more sodium is reabsorbed, leading to a sodium retention in the intact animal. Aldosterone whose antinatriuretic effect is well known has the same effects as PGE_1 on O_2 consumption, sodium and potassium content of renal cortical slices[14]. So the changes produced by PGE_1 on tubular cells in vitro cannot explain its natriuretic effect in vivo. Furthermore another prostaglandin devoid of natriuretic effect $PGF_{2\alpha}$ in vivo[15,16] was found to have similar effect to PGE_1 on tubular cells in vitro.

The results obtained, using renal tissue, as well as the lack of effect on hepatic slices, suggest that PGE_1 may have a physiological role on kidney function.

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Résumé. Chez le rat la prostaglandine E_1 augmente la consommation d'oxygène et la teneur en potassium, tandis qu'elle abaisse la teneur en sodium dans le tissu rénal et hépatique. On y a observé une corrélation entre l'effet et la dose de PGE_1 employée. Cependant dans les coupes du tissu hépathique, la PGE_1 est restée sans action. A même dose la $PGF_{2\alpha}$ a produit des effets comparables à ceux qu'on obtient avec la PGE_1. Ces résultats indiqueraient que la réponse natriurétique à la PGE_1 ne semble pas due à une inhibition de la «pompe» de sodium des cellules tubulaires, mais à une élévation du flux sanguin rénal.

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Adrenergic Mechanisms in the Cerebral Vascular Bed

From the investigations of Molnár[1,2], Mchedlishvili[3], d'Alecy and Feigl[4] and others, the existence of a neural vasomotor regulation in the cerebral vascular bed emerges as a firmly established fact. It is most likely that adrenergic fibres play a dominant role in this regulation. However, there is no clear pharmacological proof concerning the quality of the adrenergic responses of the cerebral vessels. The existence of a β-adrenergic dilator effect is especially uncertain. This study was designed to clear up the latter problems by using the specific blocking capacity of α- and β-blocking agents.

Methods. Experiments were carried out on 25 mongrel dogs (10–25 kg) lightly anaesthetized with chloralose (90–100 mg/kg). Cerebral blood flow was measured by cannulating the confluence of the sagittal and straight sinuses, with the lateral sinuses occluded, according to the method described by Rapela and Green[5]. Blood flow was determined frequently by measuring the filling time of a calibrated horizontal glass tube attached to the cannula circuit by means of a T-branch at the right atrial pressure level.

The cerebral venous outflow was continuously returned to the right jugular vein. Blood pressure was measured with a mercury manometer in the brachial artery. Occasionally the fluctuations of the blood pressure were damped by a pressure-stabilized chamber connected to one of the femoral arteries. In 8 cases, instead of venous outflow, the local tissue blood flow of the parietal cortex and/or of the subcortical white matter was recorded with the aid of the heat clearance technique using needle-shaped heated copper constantan thermocouples. Adrenaline (Tonogen, Richter) and isoprenaline (Isuprel, Winthrop) were used as adrenergic activators, while α- and β-adrenergic blockade was effected by phentolamine (Regitine, Ciba) and propranolol (Inderal, I.C.I.), respectively. The drugs were injected i.v. Heparin 500 I.U./kg was administered as an anticoagulant. The results were examined statistically using Student's t-test for paired data.

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Adrenergic responses of the cerebral vascular bed

	Mean arterial blood pressure (mmHg)		Cerebral venous outflow (ml/min)		Cerebral vascular resistance (mmHg/ml/min)	
	I. Effect of 1.0 μg/kg isoprenaline before (A) and after (B) 0.3 mg/kg propanolol administration. $n = 7$.					
	Control	Isoprenaline	Control	Isoprenaline	Control	Isoprenaline
A)	86.4 ± 10.2	72.3 ± 6.3[a]	14.7 ± 2.9	13.4 ± 3.1[a]	6.76 ± 1.26	6.88 ± 1.27[a]
B)	83.6 ± 9.0	84.8 ± 9.9[a]	15.1 ± 3.0	15.4 ± 2.9[a]	6.49 ± 1.19	6.41 ± 1.25[a]
	II. Effect of 2.0 μg/kg adrenaline before (A) and after (B) 0.3 mg/kg phentolamine administration. $n = 7$.					
	Control	Adrenaline	Control	Adrenaline	Control	Adrenaline
A)	89.9 ± 9.6	135.4 ± 14.0[b]	17.3 ± 4.2	16.6 ± 3.7[a]	6.57 ± 1.43	10.74 ± 2.55[b]
B)	74.2 ± 10.4	84.1 ± 12.8[a]	18.6 ± 3.7	20.3 ± 3.9[a]	4.41 ± 0.77	4.64 ± 0.78[a]
	III. Effect of 2.0 μg/kg adrenaline before (A) and after (B) 0.3 mg/kg propranolol administration. $n = 9$.					
	Control	Adrenaline	Control	Adrenaline	Control	Adrenaline
A)	84.7 ± 10.4	129.7 ± 7.3[d]	20.2 ± 4.7	23.3 ± 4.2[a]	5.22 ± 0.98	7.90 ± 1.87[b]
B)	82.3 ± 8.3	132.7 ± 13.4[d]	20.3 ± 2.3	25.8 ± 3.0[a]	4.62 ± 0.87	6.01 ± 0.83[b]
	IV. Effect of asphyxia of 1 min duration before (A) and after (B) propranolol administration. $n = 10$.					
	Control	Asphyxia	Control	Asphyxia	Control	Asphyxia
A)	90.2 ± 9.1	97.4 ± 14.4[a]	21.7 ± 4.1	44.8 ± 12.4[b]	5.60 ± 1.50	2.92 ± 0.32[b]
B)	83.5 ± 8.4	89.2 ± 10.7[a]	20.6 ± 2.9	37.8 ± 5.9[d]	4.70 ± 0.81	2.58 ± 0.30[c]

n = number of animals. [a] $p > 0.05$. [b] $p < 0.05$. [c] $p < 0.02$. [d] $p < 0.01$. Mean values ± S.E.