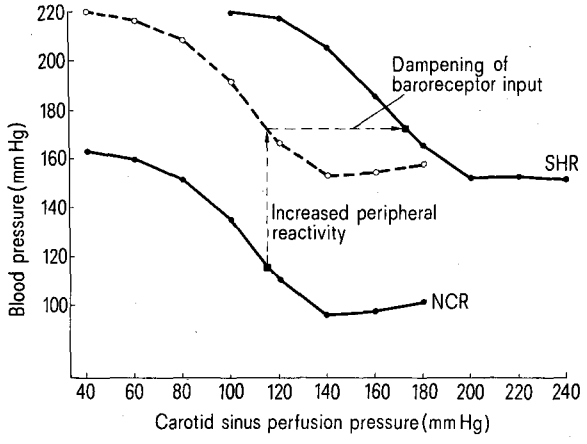
μm for 16- to 28-week-old normotensive rats and of 120 to 140 μm when the animals were made hypertensive[12]. In this latter study, fixation of the arteries was performed in vitro at varying pressures according to the blood pressure level. We assume that the extensive manipulation of the arteries in vitro and in the absence of functional sympathetic innervation has led to unphysiological dilatation.

In our experiments, the systolic and diastolic blood pressures of SHR were much higher than those of NWR, whereas the heart rate values were not different. This was a general feature observed in our SHR colony.

Discussion. Our findings suggest the following interpretation of blood pressure regulation in SHR. The baroreceptor nerve-endings of SHR are protected against excessive stimulation in spite of the hypertension, since the transmission of the pressure signal through the medial smooth muscles is dampened to a larger extent as in NWR. This explains why the efferent sympathetic tone is not depressed at the level of the vasomotor center and why the heart rate is not slowed down in the hypertensive rats. On the contrary, some available data indicate that efferent sympathetic discharge rate is rather increased in SHR, as compared to normotensive[13,14] or renal hypertensive rats[15]. In particular, our findings explain an important aspect of the modified baroreceptor functions reported in SHR[16,17]. NOSAKA and WANG[17] have tabulated their measurements, describing the relationship between systemic arterial pressure and carotid sinus perfusion pressure. We have represented their results in a diagram (Figure) and we have drawn the theoretical curve, which would be obtained if SHR had only a greater 'effector response' in comparison with NWR. These factors, whether they result from a greater amount[4,5] or from an increased reactivity[6,7] of the vascular smooth muscle cells in the resistance vessels, would only elicit an upward shift of the relationship-curve. It is clear that the results of NOSAKA and WANG with SHR also reveal an important shift of the curve from left to right, showing that a greater carotid sinus pressure is necessary before baroreceptor regulatory function comes into play. These authors speculate upon an increased rigidity of the sinus wall. Our morphometric studies on the medial hypertrophy in vascular stretch-receptor areas of SHR provide direct evidence for a structural basis of this horizontal shift of baroreceptor function due to dampening of the input signal.



●—●, experimental values published by NOSAKA and WANG[17]. ○—○, theoretical curve of NCR with increased peripheral reactivity, whithout modification of the baroreceptor setpoint.

Zusammenfassung. Morphometrische Daten bei Ratten mit genetischem Hochdruck zeigen, dass die Media der Gefässwand um etwa 50% dicker ist als bei normotonen Tieren. Diese Verdickungen sind im Aortenbogen und in der Nähe des Karotissinus gemessen worden, wo Pressorrezeptoren an der Aussenseite der Media liegen. Die hervorgerufene Verschiebung der Pressorrezeptor-Steuerung wird im Zusammenhang mit der veränderten Blutdruckregulierung bei genetisch hypertonen Ratten diskutiert.

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Bilateral Resection of Superficial Rat Kidney Cortex: Effect on Sodium Balance

Functional heterogeneity of the nephron population has been postulated since some years[1]. There was no experimental model, however, for direct studies on juxtamedullary nephrons in conscious animals. RASHID et al.[2] recently developed such an experimental model, producing unilateral necrosis of the outer rabbit kidney cortex by surface hyperthermia. We present here a similar model: bilateral surgical ablation of the whole superficial kidney cortex. Sodium balance was studied in rats subjected to this type of partial nephrectomy.

Material and methods. Male Wistar rats weighing 350–450 g were used.

Operative procedures. All rats were anesthetized with ether. Both kidneys were exposed from dorsal and the renal pedicles were clamped for a few minutes. The rats were then divided into 4 groups and each group was treated in a different way. Group I: no further treatment (sham operation). Group II: nephrectomy on the right side. Group III: nephrectomy on the right side + pole resection on the left kidney = approximately 4/6 nephrectomy[3]. Group IV: resection of the whole outer kidney cortex using a sharp scissor. Thus, about 50% of kidney tissue was removed. The wound surfaces were thoroughly dried with filter paper, and then covered with Histoacryl®, Braun, Melsungen. Seconds later the renal pedicle clamp was removed. Generally no bleeding occurred.

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Balance studies. The rats were kept in individual metabolic cages at room temperature of 23°C. They received a daily 15 g portion of a synthetic diet[4] with low or high sodium content (60 or 1500 μEq. sodium/100 g body weight/day). The potassium content of both diets was 0.5%. First the low sodium diet was given for 4 days, followed by the high sodium diet for several days until sodium balance was equilibrated. Then again the low sodium diet was prescribed for 2–3 days. The portion of the 15 g of diet was always completely ingested. Urine was collected in 24 h intervals and analyzed for sodium and potassium, at times also for creatinine. For several days, blood was taken in the morning by cardiac puncture and analyzed for hematocrit and serum concentrations of sodium, potassium, creatinine and total protein. These balance studies were carried out 1 and 12 weeks after surgery on 5 rats in each group (I–IV). In a further experiment, the same balance study was carried out on 9 intact rats. These animals were then subjected to the superficial cortex resection, corresponding to group IV 6 weeks later the balance study was repeated.

Additional examinations. One, 6 and 12 weeks after surgery, only extracellular volume (EZV) or plasma volume (PV) or GFR were measured in 3–5 rats of the groups I–IV, not subjected to balance studies. These animals were kept on low sodium diet for 4 days prior to the investigations. After sacrification by cervical dislocation, the kidneys of the rats in which GFR was measured, were removed, weighed and histologically examined after fixation in formol (10%).

Analytical procedures. Sodium and potassium were measured by flame photometry, creatinine was analyzed colorimetrically after reaction with alkaline picrate (reagents of Haury, Munich), serum protein by the Biuret method (reagents of Boehringer, Mannheim). GF Rwas determined by polyfructosan clearance, in all details as described by HORSTER and THURAU[1], with the exception that urine was collected by a bladder catheter. PV was measured by the Evans blue method, in details following ref.[5], EZF by determining polyfructosan space after nephrectomy. Polyfructosan 25%, 0.6 ml, was injected into a jugular vein 1 h after nephrectomy in ether anesthesia. 90 min later blood was obtained by cardiac puncture. Polyfructosan was measured as described by HILGER et al.[6]. During GFR measurements blood pressure was recorded from a femoral artery by use of a strain gauge.

Statistics. Data of the rats of groups I–IV was compared by an analysis of variance.

Results. The rats of group IV needed some 2 days longer to restore their sodium balance when placed from low to high sodium diet than those of groups I–III. This difference is statistically significant. No significant difference, however, occurred for the sodium excretion at the end of the first low sodium period and at the change from high to low sodium intake at the end of the experiments (Table I). Potassium excretion did not differ in the 4 groups. Serum creatinine levels were (in mg/100 ml) 0.7 ± 0.1, 0.9 ± 0.2, 1.4 ± 0.16 and 1.4 ± 0.31 for the groups I–IV respectively. No significant difference of groups I–IV rats was found for serum sodium, -potassium and -protein levels, hematocrit, PV (about 4–5% of body weight) and EZV (about 17–18% of body weight). During sodium loading, fractional sodium rejection increased more slowly after resection of the superficial kidney cortex (Table II). This data was calculated on the basis of 24 h creatinine clearance, which was about 10% lower during low sodium diet than the values obtained at high sodium intake. This difference, however, was not significant, although it was observed in all experimental groups. Blood pressure was within the normal range in the rats of all groups. Development of renal hypertrophy in respect to kidney weight and GFR per g of kidney tissue are reported in Table III. Weight gain of residual kidney tissue was similar in the groups II–IV. GFR remained lower in the rats of group IV 1 and 12 weeks after surgery in comparison with the animals of groups II and III.

Morphological findings. In the rats of group IV, the enlarged kidneys adhered to the neighboring tissue, the original wound surface was covered with connective tissue. No shrinkage and no calcification was found, remains of Histoacryl were present in varying amounts. In some kidneys, lymphatic infiltration of a mild degree was observed. The glomeruli were generally enlarged and their number was reduced by about 75%. Superficial nephrons were present only in a small area around the hilus (diameter 3–5 mm). Tubular hypertrophy occurred in the rest of the cortex and in the outer medulla. A detailed study of hypertrophy after superficial cortex resection will be given later.

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Table I. Sodium balance in rats 1 week and 12 weeks after partial nephrectomy at different localizations

Day of experiment	Sodium intake	Sodium excretion							
		Experiment 1 week after surgery (group of rats)				Experiment 12 weeks after surgery (group of rats)			
		I	II	III	IV	I	II	III	IV
4	60	74 ± 25	41 ± 30	40 ± 26	73 ± 38	45 ± 15	55 ± 23	50 ± 14	86 ± 54
5	1500	1525 ± 257	2060 ± 434	2045 ± 278	460 ± 254	1070 ± 109	938 ± 95	1030 ± 111	484 ± 191
6	1500	2245 ± 505	1393 ± 261	1583 ± 224	869 ± 428	1442 ± 118	1766 ± 433	1854 ± 204	928 ± 158
7	1500	1500 ± 104	2163 ± 282	2283 ± 274	948 ± 171	1460 ± 306	1598 ± 101	1514 ± 182	1344 ± 77
8	1500	1550 ± 77	1765 ± 134	1728 ± 130	1503 ± 82	1360 ± 122	1534 ± 388	1450 ± 503	1542 ± 151
9	60	123 ± 51	164 ± 64	205 ± 42	101 ± 30	162 ± 53	160 ± 30	131 ± 19	166 ± 67
10	60	39 ± 30	66 ± 23	71 ± 11	37 ± 10	41 ± 13	39 ± 12	46 ± 15	70 ± 41

I, sham operation, II, unilateral nephrectomy, III, 4/6 nephrectomy, IV, bilateral resection of superficial cortex. Means and SD of 5 rats in each group. Values are expressed in μEq. sodium/100 g body weight/24 h.

Table II. Sodium balance in 9 rats before and 6 weeks after bilateral resection of superficial kidney cortex

Day of experiment	Sodium intake	Before resection		After resection	
		Sodium excretion	Sodium rejection (%)	Sodium excretion	Sodium rejection (%)
4	60	47 ± 21	0.06 ± 0.03	62 ± 19	0.1 ± 0.04
5	1500	1947 ± 295	2.4 ± 0.67	753 ± 205	1.1 ± 0.6
6	1500	2265 ± 302		1150 ± 198	2.9 ± 1.0
7	1500	1349 ± 450		1666 ± 330	3.3 ± 0.98
9–11	1500	1991 ± 771		1444 ± 219	
12	60	152 ± 47		50 ± 11	
13	60	48 ± 13	0.08 ± 0.01	40 ± 17	0.16 ± 0.02

Means and SD. values are expressed in μEq. Na/100 g body weight/24 h.

Discussion. Even when kidney mass is considerably reduced, normal sodium balance can be maintained[7,8]. In the present study sodium balances were examined in rats subjected to a special type of partial nephrectomy: bilateral surgical ablation of the whole superficial kidney cortex. In such rats (group IV) a delay in sodium excretion was observed when daily sodium intake was suddenly increased. This seemed to be due to a slower increase in fractional sodium rejection. However, even these rats can maintain their sodium balance at a high intake. They need only about 2 days longer for adaptation. The critical question is: whether the slower adaptation to sodium loading is an intrinsic quality of these kidneys containing almost only juxtamedullary nephrons, or whether this might be due to extrarenal factors. Since serum electrolyte and -protein concentration, hematocrit, PV and EZV were not different in the rats of all groups, the first explanation should be favoured.

Table III. Kidney weight and GFR after partial nephrectomy

	Group of rats	Time after surgery	
		10 days	12 weeks
Kidney weight (% of controls)	I[b]	100	100
	II[c]	60	84
	III[c]	62	82
	IV[b]	72	88
GFR (ml/min/g kidney wt.)	I	1.01	0.98
	II	1.05	0.90
	III	0.85	0.90
	IV	0.68[a]	0.63[a]

I, controls; II, unilateral nephrectomy; III, 4/6 nephrectomy; IV, bilateral resection of superficial cortex. Means of 3–5 rats in each group.
[a] $p < 0.05$ to controls (I). [b] 2 kidneys. [c] 1 kidney.

Reduction of kidney mass and number of glomeruli were comparable in the rats of groups III and IV. Therefore, this reduction alone does not seem to be responsible for the delayed sodium adaptation in group IV. Since this phenomenon was observed in the same way 1 week or 12 weeks after surgery, the rate of hypertrophy obviously does not influence the modification in sodium balance.

With respect to the question of the heterogeneity of nephron populations and their possible relations to sodium excretion[1,9,10], our results may be interpreted as follows: the data presented here may be compatible with the view that superficial nephrons are of some importance – at least for the rapidness of sodium excretion when daily sodium intake is suddenly increased.

Summary. Bilateral resection of the whole superficial kidney cortex (approximately 75% of glomeruli) was carried out in rats. These animals needed some 2 days longer to restore their, sodium balance when placed from low to high sodium intake in comparison with rats subjected to other types of partial nephrectomy.

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Stimulation of Autonomic Nerves to the Urinary Bladder of the Rat

Electrical stimulation of the pelvic nerves[1,2] or hypogastric nerves[2] causes contraction of the detrusor muscle in the rat. The contractile response of the rat bladder to stimulation of the pelvic nerves at physiological frequencies is probably caused via cholinergic fibres[1,3–6] and this seems also to be the case at stimulation of the hypogastric nerves in the guinea-pig[7] and in the rat[2,6].

The contraction of the detrusor muscle caused by stimulation of one pelvic nerve has by a number of investigators been described to be confined to the ipsilateral half of the bladder in the dog, cat and rabbit[8–11]. Others suggest that the entire detrusor can be made to contract by stimulation of one pelvic nerve, although the response of the contralateral side is weaker than that of the ipsilateral in the cat and dog[12,13]. In the rat post-