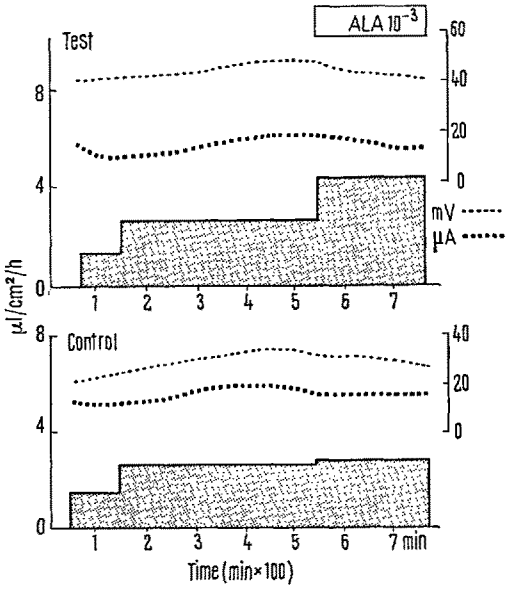

Fig. 2. The effects of ALA and ADH on short-circuit current ($\Psi\,\mu A$), potential difference (mV), and water movement ($\mu l/cm^2/h$) across a skin exposed to an osmotic gradient. The abscissa is in hundreds of minutes.

was also seen, although more rarely: ALA occasionally reversed ADH-induced increases in SCC, PD, and skin conductance.

ALA also increased the osmotic permeability of the skin to water. This was observed whether or not simultaneous changes in SCC or PD had occurred (Figure 2).

These studies thus support the hypothesis of a renal tubular locus of action of ALA, and suggest that the accumulation of this substance in advanced cases of acute porphyria may well be responsible, in part, for the water and electrolyte disturbances seen in these patients.

Zusammenfassung. Δ-Aminolävulinsäure (ALA) beeinflusst die Passage von Natrium und Wasser durch ein in vitro Präparat der Froschhaut. Dies bestätigt teilweise die Hypothese, dass ALA eine Rolle in gewissen Abnormalitäten der Nierenkanälchen bei akuter klinischer Porphyrie spielt.

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Urinary Renin and Norepinephrine Excretion in Dogs after Unilateral Renal Artery Constriction

There are only a few reports on renin excretion in urine[1–7]. Renin activity in urine of dogs was measured during variations in sodium balance[8] with the micromethod of Boucher et al.[9] with minor modifications[10]. In the present study we investigated renin activity and norepinephrine content in the urine collected separately from each kidney of dogs after constriction of one renal artery.

Material and methods. 14 femal mongrel dogs were anesthetized with pentobarbital and 1 renal artery was constricted according to the Goldblatt technique; the other kidney remained untouched. After 4 days (group A: 6 dogs) and after 4–6 weeks (group B: 8 dogs) the animals were anesthetized and catheters were placed in both ureters. The urine was collected in glass containers immersed in an ice-bath.

The following parameters were measured: Group A: Renin activity in renal venous blood (RVRA), in urine (URA), and renal cortex (RRA) was measured as recently described[9,10]. Group B: RVRA, URA, and RRA as in group A. In addition, granularity of the juxtaglomerular cells was determined by the juxtaglomerular index (JGI) of Hartroft and Hartroft[11]. Norepinephrine (NE) was measured by the method of Anton and Sayre[12]. Sodium was determined by flamephotometer. Results are expressed as mean value ± standard error (SE). Whenever possible, statistical analysis was made on paired measurements using Student's *t*-test; when the

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Table I. Renin activity[a] in renal venous plasma (RVRA), urine (URA), and renal cortex (RRA) of dogs 4 days after unilateral constriction of the renal artery ($n = 6$)

	Untouched kidney	*p*-value	Clipped kidney
RVRA/ml	6.73 ± 2.20	< 0.05	12.67 ± 3.35
URA/ml	10.34 ± 3.71	< 0.05	14.72 ± 3.66
URA/urine vol/h	82.02 ± 42.32	N.S.[b]	89.80 ± 39.27
RRA/g	28,400 ± 6,400	N.S.[b]	25,800 ± 3,000

[a] Expressed in ng angiotensin/h incubation, mean ± S.E. [b] Not significant.

Table II. Renin activity[a] in renal venous plasma (RVRA), urine (URA), and renal cortex (RRA), juxtaglomerular granulation index (JGI), sodium content of the renal cortex, norepinephrine content (NE)[b] in renal cortex and urine of dogs 4–6 weeks after unilateral constriction of the renal artery ($n = 8$)

	Untouched kidney	p-value	Clipped kidney
RVRA/ml	7.3 ± 1.1	< 0.05	12.4 ± 2.1
URA/ml	4.2 ± 1.0	< 0.01	9.7 ± 2.6
URA/urine vol/h	59.8 ± 16.0	< 0.01	87.7 ± 25.8
RRA/g	9,900 ± 5,100	< 0.001	85,000 ± 10,200
JGI	1.6 ± 0.7	< 0.001	25.2 ± 3.2
Sodium/renal cortex μEq/g dry weight	382 ± 32.9	< 0.05	339 ± 20.8
NE/g renal cortex	155 ± 23.6	N.S.[c]	344 ± 119.7
NE/ml urine	38.5 ± 10.2	< 0.05	47.5 ± 11.6
NE/urine vol/h	550.5 ± 90.8	N.S.[c]	420.7 ± 86.3

[a] Expressed in ng angiotensin/h incubation, mean ± S.E. [b] In ng norepinephrine, mean ± S.E. [c] Not significant.

differences were not normally distributed, the ranking test of Wilcoxon was applied.

Results and discussion. In both groups of dogs the mean arterial pressure increased after constriction of the renal artery (group A: from 143 ± 12 to 165 ± 12; group B: from 143 ± 7 to 177 ± 5 mm Hg). After 4 days, RVRA and URA/ml urine from the ischemic kidney rose significantly ($p < 0.05$), URA per volume urine/h from this kidney was moderately increased, whereas RRA was essentially similar to that of the untouched kidney (Table I). In contrast, in group B of dogs studied 4–6 weeks after renal clipping RVRA, URA, and RRA were significantly higher in the clipped kidney than in the untouched one (Table II). These results indicate that, besides plasma renin activity and changes in urine volume, the renin content of the renal cortex might be one of the factors determining the urinary renin excretion.

The cortex of the clipped kidney contained less sodium per g of dry weight. Similar findings in rats were recently reported by KNOWLTON and LARAGH[13]. Renin activity, as well as JGI in renal cortex, was inversely correlated to the sodium content. However, the time course of these changes needs further study.

The norepinephrine content of the renal cortex of the ischemic kidney is slightly increased, whereas the NE concentration of the urine of this kidney is significantly ($p < 0.05$) increased compared to the untouched one. HICKLER et al.[14] injected i.v. in rats fresh urine collected separately from each kidney in patients with renovascular hypertension, and reported a greater pressure response with urine from the involved kidney. In our experiments, the injection of urine after dialysis did not produce any blood pressure increase in the rat, whereas the dialyzed urine formed angiotensin during the usual 12 h incubation. The pressor agent measured in HICKLER's study might be NE, which is enhanced in the urine of the ischemic kidney. The increase of renin activity and NE in the urine from the ischemic kidney of dogs after unilateral renal clipping suggests similar studies for the diagnosis of human renovascular hypertension.

Zusammenfassung. Nach einseitiger Drosselung der Nierenarterie beim Hund fanden sich höhere Renin-Aktivität und Norepinephrin-Konzentration im Urin der ischämischen Niere als im Urin der Gegenseite. In der Rinde der gedrosselten Niere waren Renin-Aktivität und Granulationsindex der juxtaglomerulären Zellen höher als in der Rinde der ungeklemmten Niere, während der Natriumgehalt niedriger war.

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The Control of Amygdaloid and Temporal Paroxysmal Activity by the Caudate Nucleus

Experimental data point to the inhibitory role of the caudate nucleus on the central nervous system spontaneous[1–3], evoked[4–8] and paroxysmal[9] bioelectrical activity. However, the function played by the caudate nucleus on the focal epileptic seizure in the limbic system is not completely clear[10–12]. The following work is an attempt to show the inhibitory action of the caudate nucleus on the focal epilepsy in the amygdala and in the temporal cortex of the cat.

Material and methods. The experiments were performed on 22 curarized cats with local anaesthesia of painful points and on 13 cats with chronically implanted electrodes. We recorded the focal paroxysmal activity (intrastimulatory discharge and after discharge) in the basoventral complex of the amygdala and in the temporal cortex by stimulation, respectively, of the controlateral and homolateral amygdala. The conditioning activation of the head and body of the homolateral caudate nucleus at the site of deriving electrodes was performed. Conventional stereotaxic and electrophysiological techniques were used. The electrodes were localized in paraffin sections of the fixed brain (Prussian blue mark). The