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

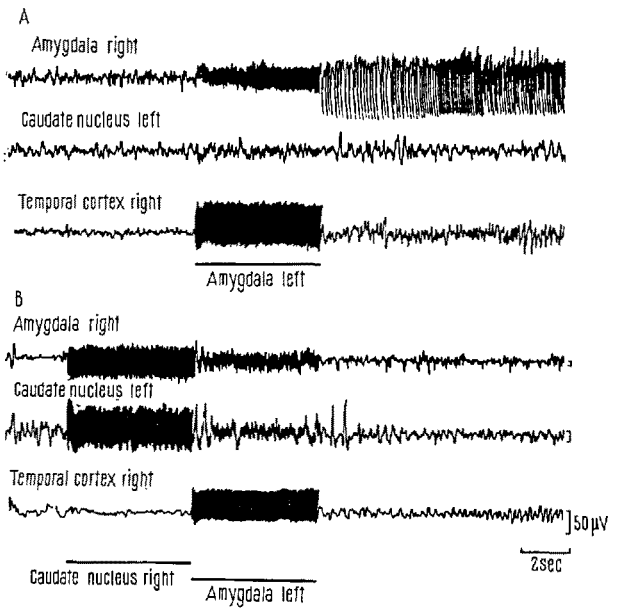
Experimental data point to the inhibitory role of the caudate nucleus on the central nervous system spontaneous[1–3], evoked[4–8] and paroxysmal[9] bioelectrical activity. However, the function played by the caudate nucleus on the focal epileptic seizure in the limbic system is not completely clear[10–12]. 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

technical details are described in preceding works[6,9] and in the work in extenso[13].

Results and discussion. The conditioning stimulation (50 c/sec, 1 msec, for 5 sec) of the head and body of the caudate nucleus constantly inhibited the focal after discharge in the basoventral amygdaloid complex, obtained by stimulation of the homologous controlateral region (30–50 c/sec, 1 msec, for 5 sec) in acute animals as well as in animals with chronically implanted electrodes. Calculating, for 1 animal, the duration in seconds of the amygdaloid after discharges we found values of $86{,}8 \pm 6.7$ sec; conditioning prestimulation of the caudate nucleus, which in 5 cases out of 9 completely suppressed the after discharges, reduced its duration to 2.06 ± 2.9 sec. Even average values of the temporal cortex after discharges, for 1 animal, obtained through repeated stimulation of the homolateral amygdala, decreased from 59.3 ± 5.4 sec to 2.8 ± 3.2 sec. Following conditioning stimulation of the caudate nucleus, the amplitude of the amygdaloid intrastimulatory discharge decreased from 240 ± 37 μV to 31 ± 13 μV. If the activation of the caudate followed a test stimulation able to evoke the after discharge in the amygdala and in the temporal cortex, a reinforcement of the focal convulsive activity was observed. In animals with chronically implanted electrodes, the stimulation of the amygdala determined, during the electrocortically recorded after discharges both in the controlateral amygdala and then (after 8–28 sec) in temporal cortex (Figure A), the appearance of epileptic phenomena, comparable to those shown in human psychomotor epilepsy, i.e.: clonic face twitches, stationary attitude of the animal, fixed look, mydriasis, open mouth, loss of saliva from the lips, vomit reflexes and, at the end of the crisis, mewing. The conditioning activation of the caudate nucleus (same parameters as for the acute animals) blocked the amygdaloid and temporal paroxysmal activity (Figure B) as well as the behavioral aspects of the epileptic phenomenon. The cat constantly showed only a slow movement of forced head rotation on the opposite side to the caudate nucleus stimulated. This reaction was followed, during the activation of the amygdala, by a static behaviour void of any epileptic equivalent.

The results shown above indicate that the high frequency stimulation of the head and body of the caudate nucleus determines suppression of the intrastimulatory as well as of the after-discharges in the basoventral amygdaloid complex and in the temporal cortex. In the cat with chronically implanted electrodes, the action of the caudate nucleus on the corticographic as well as on the behavioural phenomena of the focal epilepsy, was evidenced. During the convulsive seizure either the focal or generalized, slow regular waves were shown in the caudate nucleus indicating phenomena of active inhibition[14]. These experimental results show the existence of an extra-rhinencephalic control of the rhinencephalic paroxysmal activity. A functional rhinencephalo-caudate-rhinencephalic circuit can therefore be postulated which, while controlling the paroxysmal phenomena at the amygdaloid and temporal level, blocks its diffusion to the whole neocortex.



Cat with chronically implanted electrodes and free to move. A) The stimulation of the left amygdala (50 c/sec, 1 msec, for 5 sec, 2.8 volts) determined the appearance of a behavioral epilepsy comparable to the psychomotor epilepsy. It was characterized electrocortically by a high frequency and high voltage paroxysmal activity focalized in the right amygdala and by spikes in the temporal cortex; the caudate nucleus show slow waves 2–3/sec. B) The conditioning stimulation of the right caudate nucleus (50 c/sec, 1.5 msec, for 5 sec, 5 volts) blocked EEG and behaviorally the epilepsy in the right amygdala and in the right temporal cortex. The notable decrease in the amygdaloid intrastimulatory discharge amplitude, and the appearance of two slow waves only in the caudate nucleus, should be underlined.

Riassunto. In gatti curarizzati ed in gatti portatori di elettrodi a dimora e liberi di muoversi, la stimolazione ripetitiva ad alta frequenza del nucleo caudato inibisce la comparsa sia dei fenomeni bioelettrici parossistici focalizzati nell'amigdala e nella corteccia temporale sia dei fenomeni comportamentali omologabili all'epilessia psicomotoria.

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