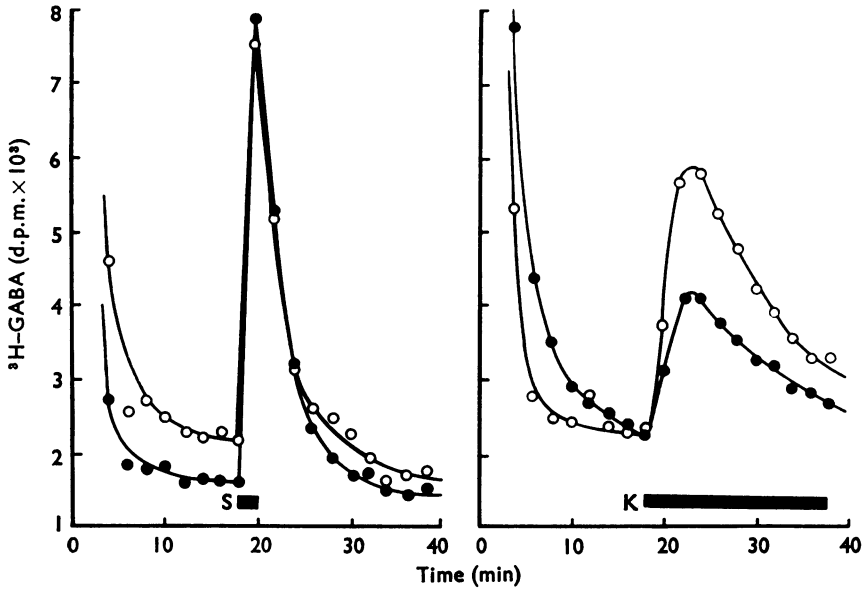

FIG. 1. Effect of electrical stimulation (S) and high potassium (K) on the efflux of ^{3}H-GABA from brain slices in normal (○) and in calcium-free (●) medium. Each point is the mean of seven results.

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5-hydroxytryptamine and 5-hydroxyindoleacetic acid in rat brain: effect of some psychotropic drugs and of electrical stimulation of various forebrain areas

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In chronically implanted, unrestrained rats the following centres were stimulated: dorsal hippocampus, frontal and piriform cortex, striatum (putamen-caudatus), anterior hypothalamus and medial thalamus. 5-Hydroxytryptamine (5-HT) and 5-hydroxyindoleacetic acid (5-HIAA) were determined in ipsilateral and contralateral parts of forebrain as well as in the brain stem. An increase of 5-HIAA concentration was observed after stimulation but 5-HT remained unchanged or even slightly decreased. The most marked increase of 5-HIAA occurred in both parts of forebrain and to a lesser extent in brain stem after stimulation of dorsal hippocampus.

During stimulation, inhibition of motor activity, tremor, upright posture and salivation were observed. Chlorpromazine completely blocked the increase of forebrain 5-HIAA as well as the behavioural effects induced by stimulation of the dorsal hippocampus (see Table 1).

TABLE 1

No. of expts.	Experimental conditions	Content in brain (μg/g) 5-HIAA A	5-HIAA B	5-HIAA C	5-HT A	5-HT B	5-HT C
12	Sham-operated non-stimulated saline	0·269 ±0·01	0·292 ±0·01	0·488 ±0·03	0·318 ±0·02	0·345 ±0·02	0·496 ±0·05
6	Stimulation of dorsal hippocampus saline	0·494* ±0·03 (+84%)	0·471* ±0·03 (+61%)	0·734* ±0·07 (+50%)	0·370 ±0·03	0·392 ±0·04	0·525 ±0·07
6	Sham-operated treatment with CPZ non-stimulated	0·366† ±0·03‡ (+36%)	0·355† ±0·03‡ (+21%)	0·636† ±0·06 (+30%)	0·382 ±0·02	0·363 ±0·01	0·608 ±0·06
5	Treated with CPZ stimulation of dorsal hippocampus	0·367† ±0·03‡	0·380† ±0·02‡	0·747* ±0·06	0·314 ±0·02	0·372 ±0·04	0·571 ±0·06

Female Sprague Dawley rats (200–220 g) were chronically implanted under ether anaesthesia with steel wire bipolar electrodes of 0·2 mm diameter by use of a sterotaxic apparatus and an atlas of brain (Köning & Klippel, 1963). Rats were stimulated 12–14 days after surgery (10 c/s, 0·5 msec, 5–6 V for 60 min). Immediately after stimulation brains were rapidly frozen and cut precollicularly (posterior to hypothalamus) into three parts—ipsilateral and contralateral half of forebrain and stem. 5-HT and 5-HIAA were estimated in the same sample by using the method of Giacalone & Valzelli (to be published). Chlorpromazine (5 mg/kg intraperitoneally) was given 30 min before stimulation.

A, Ipsilateral part of forebrain; B, contralateral part of forebrain; C, brain stem.
* $P<0·01$ (related to sham operated, unstimulated, untreated animals).
† $P<0·05$ (related to sham operated, unstimulated, untreated animals).
‡ $P<0·05$ (related to stimulated, untreated animals).

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Brain monoamines and adrenocortical activation

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Previous observations (Preziosi, Scapagnini & Nisticò, 1968) have shown that substances which deplete brain 5-hydroxytryptamine (5-HT) such as *p*-chlorophenylalanine, prenylamine, and α-methyl-dopa do not provoke an adrenocortical activation at doses which are known to decrease brain 5-HT content. This study deals with the effects of prenylamine (as gluconate, 100 mg/kg 0.1% aqueous solution, subcutaneously), of a monoamine oxidase-inhibitor, nialamide (50 mg/kg 0.5% aqueous solution, intramuscularly), and of restraint stress both in normal and in nialamide-treated rats, on the brain amine content and blood corticosterone levels in normal adult rats (120–150 g).

Brain tissue was homogenized with 0.4 N perchloric acid, 20 ml./g of tissue. Noradrenaline (NA) and dopamine (DA) were extracted by the method of Laverty, Sharman & Vogt (1965) and adsorbed on a Dowex 50 W × 4 column. Noradrenaline was eluted with 0.4 N HCl and DA with 2 N HCl. Noradrenaline was deter-