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).

This study was supported by U.S. Department of Army through its European Research Office (Contract DAJA 37-67-C-0586).

† Visiting scientist from the Department of Experimental Pharmacology, Medical Academy, Warsaw, Poland.

REFERENCE

KÖNING, G. & KLIPPEL, R. (1963). The rat brain. *A sterotaxic atlas of the forebrain and lower parts of the brain stem.* Baltimore, U.S.A.: Williams and Wilkins Co.

Brain monoamines and adrenocortical activation

A. F. DE SCHAEPDRYVER*, P. PREZIOSI and U. SCAPAGNINI, *Heymans Institute of Pharmacology, University of Ghent, Belgium, and Second Department of Pharmacology, University of Naples, Italy*

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-

mined after oxidation with potassium ferricyanide (von Euler & Lishajko, 1961), and DA according to Carlsson & Waldeck (1958). For extraction and dosage of 5-HT the method of Snyder, Axelrod & Zwerg (1965) was used. Plasma corticosterone was determined by the method of Silber, Bush & Oslapas (1958); methylene chloride was purified according to Mattingly (1962). After prenylamine, no correlation could be observed between brain amine content and adrenocortical activation. Restraint stress, however, markedly increased plasma corticosterone as well as brain 5-HT content. Nialamide strongly increased brain amine content but did not change the high plasma corticosterone levels provoked by restraint stress (Table 1).

TABLE 1

Treatment	Hours after administration or restraint stress	5-HT	NA	DA	Plasma corticosterone
None (controls)		616±10 (50)	394±4 (50)	492±21 (50)	20·8±2·4 (40)
Prenylamine	6	480±26 (18)*	212±21 (18)*	281±21 (18)*	28·5±2·5 (22)*
	24	540±20 (12)*	301±18 (12)*	343±14 (12)*	15·7±2·9 (10)
	168	601±15 (12)	531±10 (12)*	411±8 (12)*	15·4±3·4 (10)
Restraint stress (RS) (6 hr)		723±15 (16)*	327±10 (16)*	433±12 (16)*	43·3±4·5 (8)*
Nialamide	26	931±31 (16)*	640±15 (16)*	756±24 (16)*	19·2±1·8 (12)
Nialamide+RS (6 hr)	26	1,040±36 (18)**	621±25 (18)**	750±20 (18)**	49·3±3·1 (18)

5-HT, NA and DA values were expressed in ng/g, and plasma corticosterone concentrations in μg/100 ml. The values shown are means±standard errors. The figures within brackets show the number of determinations. One and two asterisks signify $P<0·01$ with respect to the controls or stressed rats, respectively.

This investigation was supported by a grant from the Italian National Research Council (115/926/926).

REFERENCES

CARLSSON, A. & WALDECK, B. (1958). A fluorimetric method for the determination of dopamine (3-hydroxytryptamine). *Acta physiol. scand.*, **44**, 293–298.

EULER, U. S. von & LISHAJKO, F. (1961). Improved technique for the fluorimetric estimation of catecholamines. *Acta physiol. scand.*, **51**, 348–355.

LAVERTY, R., SHARMAN, D. F. & VOGT, M. (1965). Action of 2,4,5-trihydroxyphenylethylamine on the storage and release of noradrenaline. *Br. J. Pharmac. Chemother.*, **24**, 549–560.

MATTINGLY, D. (1962). A simple fluorimetric method for the estimation of free 11-hydroxy-corticoids in human plasma. *J. clin. Path.*, **15**, 374–379.

PREZIOSI, P., SCAPAGNINI, U. & NISTICÒ, G. (1968). Brain serotonin depletors and adrenocortical activation. *Biochem. Pharmac.*, **17**, 1309–1313.

SILBER, R. H., BUSH, R. D. & OSLAPAS, R. (1958). Practical procedure for estimation of corticosterone or hydrocortisone. *Clin. Chem.*, **4**, 278–285.

SNYDER, S. H., AXELROD, J. & ZWEIG, M. (1965). A sensitive and specific fluorescence assay for tissue serotonin. *Biochem. Pharmac.*, **14**, 831–835.

Spinal reflex activity during exposure to saxitoxin and tetrodotoxin

M. H. EVANS†, *Sherrington School of Physiology, St. Thomas's Hospital Medical School, London S.E.1, England*

Monosynaptic and polysynaptic reflexes were tested in spinal cats to find out whether intravenous administration of saxitoxin or tetrodotoxin had any effects on the functions of the spinal cord during the time course of an acute experiment, other