24 h after injection. The maximal increase in blood glucose levels occurred 2 h after adrenaline administration. Noradrenaline (0·67 μg/g subcutaneously) failed to elicit any significant change in concentration of 5-HT or 5-HIAA at 3 h.

When insulin (66 i.u./kg subcutaneously) was administered to fed male rats that had undergone bilateral surgical removal of the adrenal medulla 3 weeks previously, an increase in brain 5-hydroxyindole levels, similar to that seen in non-surgically treated animals, was observed.

These results indicate (1) that adrenaline increases brain 5-HT metabolism, and (2) that the increased metabolism of 5-HT observed after insulin administration is not dependent on the secondary release of adrenaline.

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A comparison of the analgesia produced by morphine and the sympathomimetic drugs

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It is well established that some sympathomimetic drugs are actively analgesic in animals, but the involvement of adrenergic mechanisms in either morphine analgesia or, indeed, sympathomimetic analgesia has yet to be proved.

The effects of various drugs, known to cause changes in the amounts of noradrenaline and 5-hydroxytryptamine in the brain, were examined on the increase in hot-plate reaction time induced, in the mouse, by morphine sulphate (10 mg/kg) and by methylamphetamine hydrochloride (10 mg/kg). Effects of these drugs on the characteristic "stereotype" behaviour produced by methylamphetamine was also noted. The results are summarized in Table 1.

TABLE 1. *Effect of drugs on the increase in hot-plate reaction time induced by morphine sulphate, and methylamphetamine hydrochloride and on methylamphetamine "stereotype" activity*

Drug	Dose (i.p.)	Hot-plate reaction time: Morphine (10 mg/kg)	Hot-plate reaction time: Methylamphetamine (10 mg/kg)	"Stereotype" activity methylamphetamine
Morphine+methylamphetamine	Various	Additive		0
Reserpine	5 mg/kg 4 h*	−	−	0 to +
Iproniazid	200 mg/kg 2 h*	0	0	0
α-methyl-*p*-tyrosine	150 mg/kg 4 h*	+	+	−
α-methyl-*p*-tyrosine +iproniazid	As above	++	++	0
5-hydroxytryptophan	75 mg/kg 10 min*	+	+	0 to+
p-chlorophenylalanine	150 mg/kg twice daily for 3 days	−	−	0
5-hydroxytryptophan+ *p*-chlorophenylalanine	As above	+	+	0

−, Decrease; 0, no change; +, increase; ++, further increase. * Pretreatment.

The potentiation of the action of morphine on hot-plate reaction time induced by pretreatment with α-methyl-*p*-tyrosine is contrary to the findings of Verri, Graeff & Corrado (1967), but the abolition of the activity of morphine by *p*-chlorophenylalanine confirms the work of Tennen (1968).

There were no qualitative differences between the interactions of these drugs with morphine and with methylamphetamine when the hot-plate reaction times were considered. The mechanism by which methylamphetamine induces " stereotype " activity has been separated from that by which it increases hot-plate reaction time.

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Effects of psilocybin, dimethyltryptamine and various lysergic acid derivatives on photically-induced epilepsy in the baboon (*Papio papio*)

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Baboons (*Papio papio*) from the Casamance region of Senegal commonly show myoclonic responses to intermittent light stimulation (ILS). During ILS electroencephalographic records (e.e.g.) show polyspikes and spikes and waves predominating in the fronto-rolandic cortex (Killam, Killam & Naquet, 1967).

The myoclonic responses and cortical spikes and waves induced by ILS can be abolished by intravenous injection of lysergic acid diethylamide (50–100 μg/kg) (Walter, Balzano, Vuillon-Cacciuttolo & Naquet, 1970).

We have therefore investigated the effects of some other hallucinogenic drugs and various derivatives of lysergic acid on the motor and e.e.g. responses to ILS in conscious baboons. The animals were selected for consistently high responsiveness to photic stimulation and were chronically implanted for e.e.g. recording.

Psilocybin (0·5–4·0 mg/kg) and N,N-dimethyltryptamine (0·5–4·0 mg/kg) both produced a marked mydriasis with an increase in spontaneous eye movements and a tendency to keep the eyes open during ILS. Generalized motor activity was reduced. The lower doses led to a disappearance from the e.e.g. of activities in the range 4–12 Hz with preservation of fast activity. The higher doses caused the appearance of diffuse delta activity. ILS at 15 or 30 min after psilocybin (1–2 mg/kg) failed to provoke either myoclonic responses or the usual cortical spikes and waves. This protective effect lasted for more than 60 min after 4 mg/kg. Both motor and e.e.g. paroxysmal responses to ILS were blocked 15 min after dimethyltryptamine (2–4 mg/kg), but, as with the autonomic and behavioural changes, recovery after dimethyltryptamine was more rapid than after psilocybin.

Methysergide bimaleate (2–4 mg/kg) produced sedation, reduction in muscle tone and enhancement of e.e.g. slow activities. After the injection, ILS failed to produce myoclonus or the usual spikes and waves. These responses returned progressively 30–120 min after the injection.