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.

2-Bromo-lysergic acid diethylamide hydrogen tartrate (BOL 148) (2–4 mg/kg) produced drowsiness, hypotonia and excessive salivation for 20–60 min. The highest dose suppressed both the myoclonic and the paroxysmal e.e.g. responses to ILS.

Methergoline (FI 6337), a powerful central antagonist of 5-hydroxytryptamine (Beretta, Ferrini & Glässer, 1965; Mawson & Whittington, 1970), at 4 mg/kg produced some hypotonia and increased salivation. Myoclonic responses to ILS were reduced 15 min after the injection but cortical spikes and waves persisted.

LSD, psilocybin and dimethyltryptamine have complex effects on activity in the visual pathways which may contribute to the observed change in responsiveness to ILS.

Among the lysergic acid derivatives which antagonize some of the actions of 5-hydroxytryptamine (methysergide, BOL 148 and Methergoline), the reduction in myoclonic response to ILS correlates with the degree of sedation and loss of muscle tone produced by the drug. However, the possibility that both groups of compounds act by a common mechanism cannot yet be excluded.

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Acute toxicity of heroin, alone and in combination with cocaine or quinine

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Cherubin (1968) states that more than 70% of deaths in heroin-dependent persons in New York are due to " overdose—in reality sudden collapse after intravenous injection with the major finding at autopsy being pulmonary oedema ". Fulton (1965) showed by analysis of confiscated drug samples that adulterants are commonly present in illicit samples of heroin. Quinine is one such substance. Addicts frequently combine heroin with cocaine. Heroin, heroin with cocaine, and heroin with quinine were dissolved in a solution of sodium chloride (0·9% w/v). The acute toxicity was determined by injection into the tail vein of male white mice of 20 g weight, kept at 30° C, at a rate of 0·1 ml/min until fatal collapse occurred. Control mice were injected with an equal volume of saline and killed by dislocation of the neck. The lungs were removed from all animals, blotted dry and weighed.

The acute toxicity of heroin hydrochloride (calculated as base) was 56·7± 2·4 mg/kg (mean±S.E. of mean) and the lung weight of 5·59±0·06 g/kg did not differ from that of controls. The mean lethal dose for cocaine hydrochloride (calculated as base) was 30·7±1·7 mg/kg and quinine hydrochloride (calculated as base) 137·8±3·3 mg/kg.