of the substantia nigra and median raphe nucleus. For example, (+)-amphetamine (7.5 mg/kg) and p-chloroamphetamine (5 mg/kg) are known to release dopamine and 5HT respectively. Both drugs increase the current produced in the caudate and the increase with p-chloroamphetamine does not occur in rats pretreated with p-chlorophenylalanine (2 × 150 mg/kg) indicating it to be associated with 5HT release.

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Monitoring motor activity using doppler shift radar

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Many psychoactive drugs not only induce changes in total motor activity but also produce a shift from one form of activity (e.g. normal exploratory behaviour) to another (e.g. continuous body movements without exploratory behaviour). Existing automatic activity monitors have only limited capabilities to differentiate between different types of activity. The present system uses the principle of doppler shift radar to analyse both the speed, and the time pattern of bursts of activity. The measurements obtained are recorded using a multi-channel microprocessor controlled printer.

Activity has been classified in two ways. First, in terms of times in seconds the animal spends in each selected form of activity such as fast speed activity (typically normal exploratory behaviour) or slow speed activity (head turning). Second in terms of the number of bursts of either fast or slow activity. A burst ends when the animal has stopped moving for a specified time. The speed bands can be subdivided into three sensitivity levels and burst times adjusted to suit the type of behaviour being measured.

The monitor has been used to observe the behavioural effects on rats of p-chloroamphetamine (5 mg/kg i.p.) at different times during a 12 h light-dark cycle. p-Chloroamphetamine produces a behavioural response consisting of hyperactivity, lateral head weaving, forepaw treading and tremor (Trulson & Jacobs, 1976). The activity monitor is able to distinguish two phases in this response. The first starts within 10 min of administration and consists of increased fast and slow activity but very little burst activity (continuous body movements, head weaving, tremor). Fifty–70 min after injection while total activity is still above normal there is a marked increase in burst activity indicating atypical highly spasmodic locomotive behaviour (Figure 1). There is evidence that the two components of the behavioural response involve different neurotransmitter systems (Crow & Deakin, 1977).

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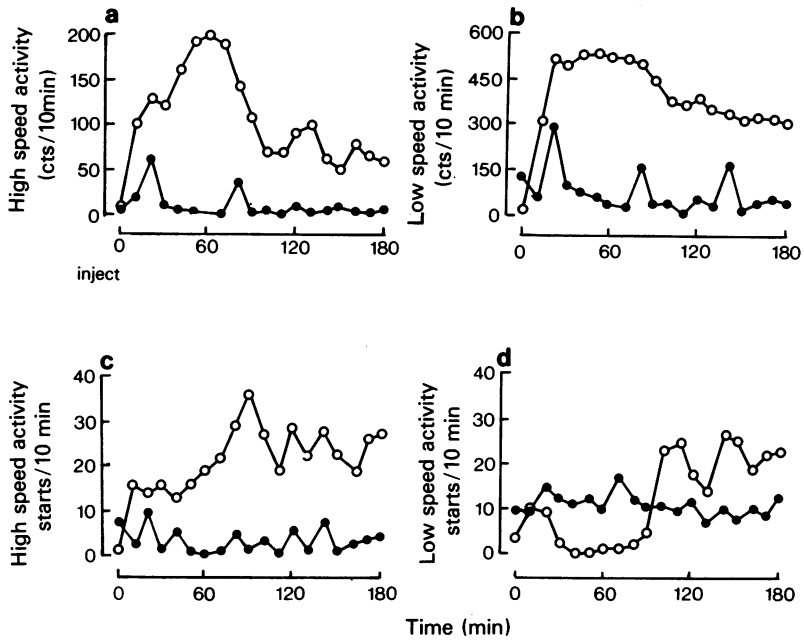

Figure 1 Effect of (○) p-chloroamphetamine (5 mg/kg) and (●) 0.9% saline on rat activity measured by doppler shift radar. The initial effect of p-chloroamphetamine is to produce increased and almost continuous high and low speed movements (head turning, forepaw treading) indicated by the increased counts in a and b and reduced starts in c and d. From 90 min after injection the main effect of the drug is to increase exploratory behaviour (increased counts in a and b associated with increased starts in c and d). Results are given as the mean of 4 experiments.

An indirect effect of isoprenaline on α-amylase release by amphibian pancreas organ cultures

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Amphiuma means pancreas fragments survive in organ culture for several weeks, and Gater & Balls (1977) found that the factors affecting amylase release were similar to those which influence the mammalian acinar pancreas. However, it was not clear whether isoprenaline-stimulated amylase secretion resulted from a direct β-adrenoceptor effect on the acinar cells themselves or an indirect effect via the release of endogenous acetylcholine.

Groups of four pancreas cultures were treated with methacholine chloride, isoprenaline hydrochloride, atropine sulphate and/or timolol maleate. The culture medium was changed each day and assayed for α-amylase content (Gater & Balls, 1977).

Methacholine stimulated amylase release, and this effect was blocked by atropine, but not by timolol (Table 1). Isoprenaline-stimulated amylase release was also reduced by atropine, but not by the β-adrenoceptor antagonist, timolol. Timolol, however, did block the glycogenolytic effect of isprenaline on liver organ cultures from the same animal: glycogen content (% wet weight, 48 h after addition, $n = 4$) was: control, 1.5 ± 0.1; 10^{-5} M isoprenaline, 0.2 ± 0.05 ($P < 0.001$); isoprenaline + 10^{-4} M timolol, 1.3 ± 0.2.

These results suggest, as in the case of cat isolated perfused pancreas (Pederson & Schulz, 1974), that the isoprenaline-induced secretion of amylase could have resulted from the release of endogenous acetylcholine (possibly from intact cholinergic nerve endings), which then stimulated the muscarinic receptors of the