stimulated by rectangular pulses (60 Hz, 20 mA, 5 ms) for 30 s. This caused a significant increase in the efflux of radioactivity, as illustrated in Fig. 1. The maximum increase was 2·3 times the resting efflux (mean of eight experiments), and occurred during the period of stimulation. The increased efflux of glycine was not a non-specific effect on the cell membrane, as electrical stimulation did not cause an increased release of ^{14}C-urea from cord slices (Fig. 1). Although *in vitro* experiments in which the release of substances from nervous tissue after electrical stimulation must be interpreted with caution, these results are not inconsistent with a neurotransmitter role of glycine.

This work was supported by a grant from the S.K.F. Foundation.

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The pharmacological effects of ouabain administered intracerebrally to conscious mice

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In certain species, cardiac glycosides may release endogenous catecholamines, which act on the myocardium to produce a positive inotropic effect (Tanz, 1967). Also, the lethal effects of ouabain in guinea-pigs are brought about partially by a liberation of catecholamines (Hermansen, 1970). The pharmacological effects of these glycosides on the central nervous system have not been examined fully; we have studied the pharmacological effects of ouabain administered by direct intracerebral injection.

Ouabain (0·1 to 0·4 μg in 10 μl) was injected into the cerebral ventricles of conscious male TO mice, using the method of Brittain & Handley (1967). These injections were followed by a central nervous depression lasting 2–3 h and characterized by a loss of locomotor activity, lowered body posture and lack of response to external stimuli. Ouabain was not anti-convulsant, but predisposed mice to electrically-induced convulsions. There was a whole-body hypothermia which reached a maximum fall of 11° C at 90 min. This hypothermia, together with the other effects, was dose dependent. The hypothermia was accompanied by peripheral vasodilatation associated with a transient rise in skin temperature. Higher doses of ouabain (1–100 μg) induced convulsions and death in at least 80% of the animals. The effects appeared to be centrally mediated, for doses of up to 20 μg subcutaneously produced no behavioural depression and only slight transient hypothermia of about 3° C fall at 30 min.

The ouabain-induced hypothermia and behavioural depression were rapidly and completely reversed by the intraperitoneal injection of dexamphetamine (10 mg/kg). Similarly, intraperitoneal desipramine reduced (5 mg/kg) or abolished (10 mg/kg) these effects. In contrast, nialamide (20 mg/kg intraperitoneally 2 h beforehand) produced no reversal. In a recent report (Abdulla & Hamadah, 1970) successful treatment of clinical depression by anti-depressant drugs was shown to be accompanied by increased levels of urinary cyclic adenosine monophosphate. In the present experiments the intracerebral injection of a mixture of ouabain (0·3 μg) and dibutyryl cyclic adenosine monophosphate (25 μg) produced a level of depression and hypothermia significantly less than the same dose of ouabain administered alone ($P<0.05$). Finally, the effects of intracerebral ouabain on whole-brain amine levels were determined. Using a spectrophotofluorimetric assay (Spencer & Turner, 1969) ouabain increased dopamine levels by 103% ($P=<0.01$), while noradrenaline and 5-hydroxytryptamine remained unchanged.

The pharmacological effects of centrally-administered ouabain show a number of similarities to peripherally-administered reserpine, but there seems to be no depletion of brain amines. Although nialamide was unable to antagonize ouabain, the antagonistic effects of dexamphetamine and desipramine suggest that the effects of ouabain are mediated through central adrenergic mechanisms. It is possible that centrally administered ouabain might prove to be an alternative tool to reserpine in the evaluation of potential anti-depressant drugs.

We wish to acknowledge grants from the Pharmaceutical Society of Great Britain and the Medical Research Council.

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Some effects of muscarine on the central nervous system of chickens

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The central effects of cholinergic drugs have been little studied in avian species. In the present investigations, cholinergic drugs were introduced into the third ventricle of adult fowls in volumes of 10 μl or micro-infused intracerebrally in 1 μl volumes for 30 s using chronically implanted cannulae (Marley & Stephenson, 1968a) in 12 to 20 day old chicks.

Muscarine (0·0067 μmol) within 4 min of intraventricular injection induced marked electrocortical arousal in drowsy birds, lasting 60–90 min; in alert birds, arousal