times higher than that of oxprenolol. (—)-Propranolol showed marked protective action in a dose range of 0·375–3 mg/kg intravenously. The doses required to exhibit 50% protection against ouabain-induced ventricular fibrillation ((—)-propranolol : oxprenolol : I.C.I. 50,172) were 2·7 : 3·3 : 6·8 μ-moles/kg, the relative molar activities to procaine for local anaesthesia 3·5 : 1·4 : 0·04. All the three compounds exhibited quinidine-like actions on the parameters of atrial function. They decreased the maximum driving frequency, conduction velocity and the rate of rise of the intracellularly recorded atrial potentials.

Since the protective effect of I.C.I. 50,172 against ouabain-induced ventricular fibrillation correlates much better with its *in vivo* β-receptor blocking potency than with its local anaesthetic and quinidine-like activity, our observations support the view that " specific " blockade of β-receptors might contribute to the anti-digitalis effect of the β-receptor blocking agents.

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Sodium fluxes in cultured cells in the presence of ouabain

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L cells (fibroblasts) were explanted and cloned from mouse subcutaneous tissue (Sanford, Earle & Likely, 1948) and where they have been selected to grow in suspension (Earle, Bryant, Schilling & Evans, 1956) they are called L suspension or L.S. cells. Lamb & MacKinnon (1967) showed that 10^{-3}M ouabain caused an immediate reduction in the K^+ influx in L.S. cells. However, after 4 hr in the presence of ouabain, K^+ influx had recovered to a value slightly greater than the control. Both Na^+ removal and the addition of 10^{-3}M di-nitrophenol and 10^{-4}M iodo-acetic acid reduced the K^+ influx in cells treated with ouabain for 4 hr to about 20%. These results indicated the existence of an ouabain-insensitive Na^+–K^+ pump.

In the present experiments we measured the Na^+ fluxes in the presence of ouabain. For this, L cells grown in plastic Petri dishes at 37° C were used. The cells were loaded with $^{24}Na^+$ and then washed with five changes of inactive Krebs solution

(30 sec). One plate of cells was used to measure the $^{24}Na^+$ content at zero time and several plates were used to determine the $^{24}Na^+$ left in the cells after various time intervals in radioactive-free solution, a separate plate of cells being used for each time interval. All experiments were done at 21° C.

The Na^+ exchanges with a $t\frac{1}{2}$ of about 3 min, with a mean value of about 4·5 p-mole/cm² per sec. This is similar to other cells (Burrows & Lamb, 1962). Ouabain (10^{-3}M) for 30 sec reduces the Na^+ efflux to about 40% of its previous value. Removal of KCl from Krebs (KCl < 0·3 mM) reduces the Na^+ efflux to about 50% within 30 sec. These results indicate that the Na^+ efflux is largely active, coupled to K^+ and sensitive to ouabain.

Further experiments showed that after 4 hr in the presence of ouabain, the Na^+ efflux was slightly greater than the control. Removal of K^+ (in the presence of ouabain) reduced the Na^+ efflux to about 60%. Neither the immediate application (30 sec) nor the prolonged treatment (4 hr) of ouabain appears to affect the Na^+ influx, measured over 30 sec. The cellular content of Na^+ in cells treated with ouabain increased from 8 mmoles/l. intracellular water to about 35 mmoles/l. intracellular water in the first 2 hr, and then remains about this level for the next 6 hr. If, after 4 hr treatment with ouabain, the cells are transferred to a K^+-free Krebs solution containing ouabain for a further 2 hr, the Na^+ content rises to just over 50 mmoles/l. intracellular water.

We interpret these results as showing the existence of an ouabain insensitive K^+ coupled Na^+ pump in L cells. Recent experiments suggest that either an increase or decrease in the cellular levels of Na^+ or K^+ respectively control the amount of active transport in the presence of ouabain.

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The uptake of tetracycline by human blood cells

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The native fluorescence of tetracycline in blue light has been used to determine its localization in human blood cells. Samples of venous blood were incubated for one hour at 37° C with concentrations of tetracycline hydrochloride between 10 μg/ml. and 1 mg/ml. Unstained smears of the cell suspensions were then examined by phase-contrast and fluorescence microscopy.

The intense yellow fluorescence characteristic of tetracycline was found only in the leucocytes; the erythrocytes appeared uniformly dark. Measurement of the cellular fluorescence revealed a dose-dependency over the concentration range examined. Uptake of tetracycline appeared to depend also on incubation time;