ELSBY, J. M. & WILSON, H. (1967). Lacrimal secretion in the cat. *Br. J. Pharmac. Chemother.*, **29**, 1–7.

GOLDING-WOOD, P. H. (1964). The ocular effects of autonomic surgery. *Proc. R. Soc. Med.*, **57**, 494–497.

JENDRASSIK, E. (1894). Sur le role du nerf facial dans la sécrétion des larmes. *Rev. neurol.*, **7**, 186–192.

TEPLIACHINE, A. (1894). Recherches sur les nerfs sécrétoires de la glande lacrymale. *Archs Ophtal., Paris*, **14**, 401–413.

Two simple, relatively inexpensive, devices to measure the locomotor activity of anaesthetized or unanaesthetized animals

A. ANGEL (introduced by K. J. ROGERS), *Department of Physiology, The University, Sheffield, S10 2TN*

In the course of some investigations into the convulsant activity of polyhydroxy phenols it became necessary to be able to measure quantitatively the locomotor activity produced by these chemicals. A system had to be devised which would

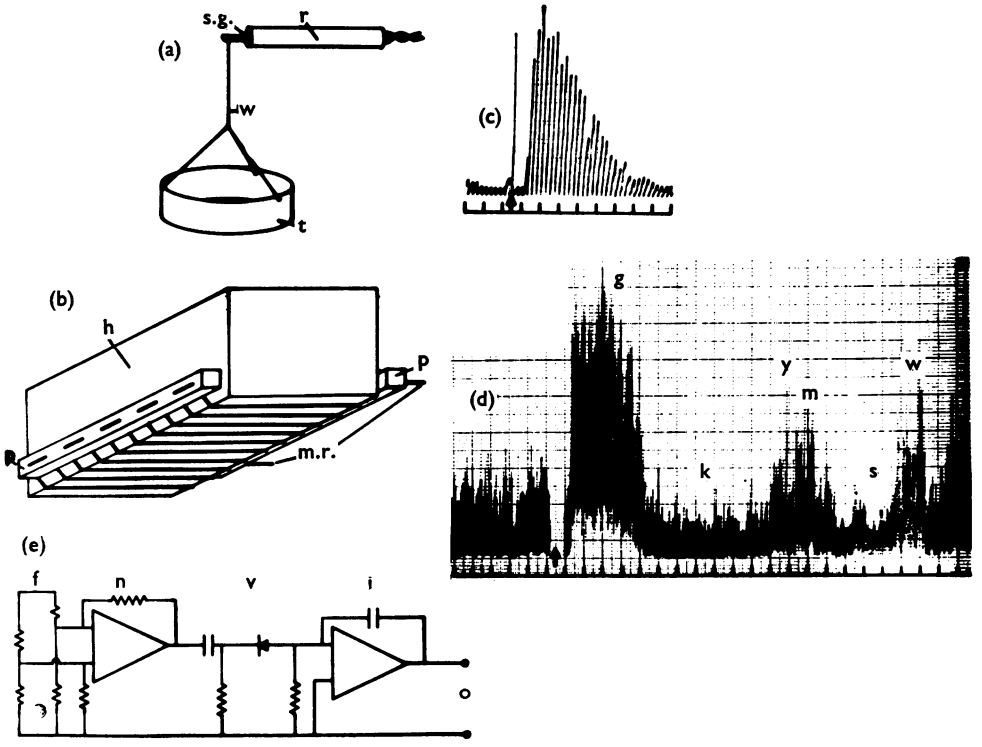

FIG. 1. (a), Diagram of the activity meter for anaesthetized animals. The plastic container *t*, suspended by a stiff wire *w* from the silicon strain gauge *s.g.* mounted on a beam attached to rod *r* which is held firmly in a clamp. (b), Apparatus for unanaesthetized animal showing the cage *h* supported on the mercury in rubber strain gauge *m.r.* which is held between two Perspex bars *p*. (c), Typical record of the convulsant effect of catechol (200 mg/kg) injected intraperitoneally into an anaesthetized mouse (at arrow). Time scale shows 2 min intervals; the integration period was 30 s. (d), Record from an unanaesthetized mouse before and after an intraperitoneal injection of catechol (60 mg/kg) at the arrow, showing the convulsion *g*, the period of post-ictal depression *k* and the activity produced by the animal washing itself *y*, sitting still *s* or moving around the cage *m*. The time scale shows 1 min intervals; the integration period was 3 s. (e), Circuit diagram of the apparatus, *f*, bridge; *n*, amplifier; *v*, AC coupling and half-wave rectification; *i*, integrator and *o*, output.

measure motor activity even when the animal was still—grooming itself, feeding or just twitching; or when the animal was anaesthetized. For anaesthetized preparations the problem was resolved by placing the animal in a plastic container suspended by a stiff piece of wire from a semiconductor strain gauge (Devices Ltd., type 2STO2, Fig. 1a). For unanaesthetized animals the problem was more difficult because the activity had to be measured no matter where the animal was in its cage. Suspending a whole cage from a semiconductor strain gauge posed problems mainly concerned with the weight of the cage. The problem was finally overcome by resting the cage on a continuous length of silicone rubber tube filled with a mercury and supported between two Perspex bars (Fig. 1b). The strain gauges formed part of a d.c. bridge circuit the output from which was led to an operational amplifier for amplification. This amplified signal was then half wave rectified and a.c. coupled into another operational amplifier connected to form an integrator. The integrator was reset to zero at fixed time intervals and its output displayed on a pen recorder. The signal was a.c. coupled into the integrator so that the bridge need only be balanced at the beginning of an experimental run; any drift in the bridge amplifier or bridge can then be disregarded. Records taken from each activity " meter " are shown in Fig. 1c, d.

I wish to thank the M.R.C. for a grant.

The vesicle population of rat ganglionic synapses and the effects of some drugs

J. P. QUILLIAM and D. L. TAMARIND, *Department of Pharmacology, St. Bartholomew's Hospital Medical College, Charterhouse Square, London E.C.*1

The " local vesicle population " (l.v.p.) in synapses of isolated rat superior cervical ganglia have been estimated by counting the number of vesicles within 0·25 μm of the pre-synaptic membrane in preparations after 1 h in a modified McEwen's (1956) solution at 37° C. A mean value of $120 \pm 3{\cdot}88$ (S.E.) vesicles/μm^2 was obtained ($n=107$ synapses in five ganglia).

In drug experiments, unstimulated isolated ganglia were bathed in our McEwen's solution for 30 min. Then the solution was replaced by fresh solution (controls) or one containing the test drug for 30 min, after which ganglia were fixed overnight in phosphate-buffered osmium tetroxide and sections about 500 μm prepared for electron-microscopy. With hexamethonium, the drug treatment period was for only the final 10 min of the hour *in vitro*. Vesicles in at least sixty synapses were counted in three preparations with each drug.

Hexamethonium bromide (0·275 mM) significantly increased ($P<0{\cdot}001$) the l.v.p. from control values to $146 \pm 5{\cdot}2$ (S.E.) vesicles/μm^2, while methylpentynol carbamate (MPC, 13·86 mM) elevated the l.v.p. to $152 \pm 4{\cdot}9$ (S.E.) vesicles/μm^2: $P<0{\cdot}001$. These two increases were significantly ($P<0{\cdot}005$) less than the l.v.p. value of $179 \pm 7{\cdot}3$ (S.E.) vesicles/μm^2 with amylobarbitone sodium (AMY, 4·02 mM). The concentrations of MPC and AMY were at least five times greater than those which blocked ganglionic transmission and impaired acetylcholine (ACh) release from preganglionic nerve stimulation in perfused cat superior cervical ganglia (Matthews & Quilliam, 1964).