

During pregnancy, e.m.g. tests indicated that the mother was overtreated with pyridostigmine (mestinon) and measurements with  $^{14}\text{C}$ -labelled pyridostigmine showed that she excreted this drug more slowly than other myasthenic patients.

The diagnosis of drug-induced neonatal myasthenia due to placental transfer of pyridostigmine was made on the basis of the low level of plasma cholinesterase in maternal and cord blood, the onset of symptoms 24 hr after birth and the decreasing requirement for neostigmine.

Pyridostigmine has two actions, inhibition of cholinesterase activity and interaction with acetylcholine receptors. It is suggested that sufficient pyridostigmine was transferred so that at birth it produced neuro-muscular depression which was offset by inhibition of endplate acetylcholinesterase. It is postulated that the anticholinesterase action of the retained pyridostigmine or its metabolites has a shorter duration than the depressant action and that 24 hr after birth depression is dominant. The course of neostigmine therapy served to maintain inhibition of cholinesterase activity during the slower recovery from depression.

#### **Some effects of sympathomimetic amines on isolated smooth muscle preparations from the stomach of the guinea-pig**

DIANA M. BAILEY (introduced by G. BROWNLEE), *School of Pharmacy, Leicester Regional College of Technology, Leicester*

Strips of smooth muscle from the wall of the stomach of the guinea-pig were prepared by cutting parallel to the lesser curvature (longitudinal muscle) or at right angles to the lesser curvature (circular muscle). Motor responses of the strips were greatly enhanced after removal of the mucosa by separating the tissues of the stomach wall at the level of the submucosal connective tissue.

All preparations showed some spontaneous activity when suspended in Krebs solution at  $39^{\circ}\text{C}$ , gassed with 95% oxygen and 5% carbon dioxide. Most preparations possessed some degree of tone as defined by their ability to relax in the presence of isoprenaline.

Responses to isoprenaline ( $0.1\text{--}10\text{ }\mu\text{g/ml.}$ ) were antagonized by the  $\beta$ -receptor blocking drugs, propranolol ( $1.0\text{ }\mu\text{g/ml.}$ ) or 4-(2-isopropylamino-1-hydroxyethyl) methane sulphonilide HCl, Mead Johnson 1999 ( $10\text{ }\mu\text{g/ml.}$ ).

Adrenaline or noradrenaline ( $0.1\text{--}5.0\text{ }\mu\text{g/ml.}$ ) caused either a relaxation, a contraction, or most commonly a biphasic response in which a relaxation was followed by a contraction. On analysis with  $\alpha$ - and  $\beta$ -receptor blocking drugs the responses appeared to involve an inhibitory component mediated via  $\beta$ -receptors and an excitatory component mediated via  $\alpha$ -receptors.

#### **Metabolic actions associated with stimulation of $\alpha$ - and $\beta$ -receptors for adrenaline in smooth muscle**

R. G. G. ANDERSSON\* and ELLA MOHME-LUNDHOLM, *Metabolic Division, Department of Pharmacology, University of Göteborg, Göteborg, Sweden*

The relaxing action of the catecholamines in intestinal smooth muscle is mediated by both adrenergic  $\alpha$ - and  $\beta$ -receptor mechanisms (Ahlquist & Levy, 1959; Jenkinson &