5. There are disturbances in carbohydrate metabolism, a decreased oxygen consumption, a sharp drop in the respiratory quotient, decreased utilization of pyruvate by liver homogenates (for literature, see Bacq & Goutier, 1968).

6. There is mitotic delay and temporary inhibition of DNA and RNA synthesis in the rat's regenerating liver after injection of aminoethylisothiourea (AET) which by transguanylation in aqueous solution is isomerized to 2-mercaptoethylguanidine (Baugnet-Mahieu, Goutier & Semal, 1967). There is also decreased thymidine kinase and DNA polymerase activity in spleen and thymus after AET or MEA injection (Bacq & Goutier, 1968).

None of these phenomena is produced by control injections of β-mercaptoethanol which has no radioprotective action in mammals (see for instance, Plomteux *et al.*, 1968; Hugon *et al.*, 1966).

REFERENCES

BACQ, Z. M. (1965). *Chemical Protection against Ionizing Radiation.* U.S.A.: Springfield, C. Thomas.

BACQ, Z. M. & GOUTIER, R. (1968). Mechanisms of action of sulfur containing radioprotectors. *Brookhaven symposium on Recovery from Radiation Injury*, June 1967, in the press.

BAUGNET-MAHIEU, L., GOUTIER, R. & SEMAL, M. (1967). Effects of total-body irradiation and AET on the activity of thymidine-phosphorylating kinases during liver regeneration. *Radiat. Res.*, **31**, 808–825.

BEAUMARIAGE, M. L., VAN CANEGHEM, P. & BACQ, Z. M. (1966). Étude de certaines propriétés pharmacodynamiques de la cystamine et de la cystéamine chez diverses espèces animales. *Strahlentherapie*, **131**, 342–351.

FIRKET, H. & LELIÈVRE, P. (1966). Effet de la cystamine sur la respiration, la phosphorylation oxydative de l'ultrastructure des mitochondries du rat. *Int. J. rad. Biol.*, **10**, 403–415.

HUGON, J., MAISIN, J. R. & BORGERS, M. (1966). Modifications ultrastructurales après radioprotecteurs. *Int. J. rad. Biol.*, **11**, 105–116.

LECOMTE, J., CESSION-FOSSION, A., LIBON, J. C. & BACQ, Z. M. (1964). Sur quelques effets pharmacodynamiques généraux de la cystamine chez le rat. *Archs int. Pharmacodyn. Thér.*, **148**, 487–510.

LECOMTE, J. & BACQ, Z. M. (1965). Propriétés vasomotrices de la 2-mercaptoethylguanidine (MEG) chez le rat. *Archs int. Pharmacodyn. Thér.*, **158**, 480–497.

PLOMTEUX, G., BEAUMARIAGE, M. L., BACQ, Z. M. & HEUSGHEM, C. (1967). Variations enzymatiques dans le plasma du rat après injection d'une dose radioprotectrice de cystéamine. *Biochem. Pharmac.*, **16**, 1601–1607.

PLOMTEUX, G., BEAUMARIAGE, M. L., BACQ, Z. M. & HEUSGHEM, C. (1968). Influence du β-mercaptoethanol sur certains enzymes plasmatiques du rat. *Biochem. Pharmac.*, in the press.

VAN CANEGHEM, P. & STEIN, F. (1967). Influence de l'injection de cystamine et de divers états de choc sur les protéines plasmatiques du rat, du lapin et du poulet. *Archs int. Physiol. Biochem.*, **75**, 769–786.

Drug-induced neonatal myasthenia

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A male infant born of a myasthenic patient was clinically normal until 24 hr after delivery, when he became lethargic, had only a faint cry, and had weak sucking and grasping reflexes. Several severe cyanotic and apnoeic attacks occurred and the baby was clinically myasthenic.

A normal electromyogram (e.m.g.) had been obtained at 12 hr after delivery but at 24 hr the e.m.g. pattern was characteristic of myasthenia. The diagnosis was confirmed by the improvement in clinical condition, and by an increase in neuro-muscular transmission measured by e.m.g. after an injection of neostigmine (0.1 mg I.M.). The baby was treated for 12 days with intramuscular injections of neostigmine in progressively decreasing dosage, and at the end of this period anticholinesterase therapy was stopped without the re-appearance of any myasthenic symptoms.

During pregnancy, e.m.g. tests indicated that the mother was overtreated with pyridostigmine (mestinon) and measurements with ^{14}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

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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° 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 (0.1–10 μg/ml.) were antagonized by the β-receptor blocking drugs, propranolol (1.0 μg/ml.) or 4-(2-isopropylamino-1-hydroxyethyl) methane sulphonanilide HCl, Mead Johnson 1999 (10 μg/ml.).

Adrenaline or noradrenaline (0.1–5.0 μg/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 α- and β-receptor blocking drugs the responses appeared to involve an inhibitory component mediated via β-receptors and an excitatory component mediated via α-receptors.

Metabolic actions associated with stimulation of α- and β-receptors for adrenaline in smooth muscle

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