Peterson & Feigel (1967). These changes could also be responsible for the decreased sensitivity of the carotid baroreceptors to stretch which we observed in cats with nephrogenic hypertension in agreement with McCubbin, Green & Page (1956) and Kezdi's (1967) observations in renal hypertensive dogs.

An increase of the catecholamine content of the brain and adrenals and a slight decrease in heart noradrenaline content can be demonstrated in rats with renal hypertension. This change could reflect reduced neuronal activity and transmitter pile-up, or enhanced rate of transmitter formation. The evidence, however, that transmitter content can increase during neuronal rest or during chronic hyperactivity makes it difficult to relate these changes to the functional state of adrenergic neurones.

Adrenergic mechanisms appear not to contribute to hypertension beyond their physiological role. The dilatation of arterioles by antiadrenergic drugs in a vascular system with a reduced diameter would produce greater decrease in resistance and fall in systemic pressure than is possible in normotension with vessels of normal diameter and low resistance.

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The state of shock induced by cystamine and cysteamine

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We believe that the pharmacological actions of sulphur-containing radioprotecting substances (cysteamine=MEA; cystamine; AET or mercaptoethylguanidine, etc.) play a decisive part in the mechanisms of the protection of mammals against ionizing radiation (Bacq, 1965). The following facts have been established mainly in the rat but also in other animal species. The effects of cystamine (S–S) are usually more marked than those of cysteamine (SH).

1. There is cardiovascular shock and hypotension which is long lasting after large radioprotective doses (150 mg/kg, I.P. or I.V.) (Lecomte, Cession-Fossion, Libon & Bacq, 1964; Lecomte & Bacq, 1965; Beaumariage, Van Caneghem & Bacq, 1966).

2. There is haemoconcentration, hypoproteinemia without decrease of the albumin/globulin ratio and a marked decrease (30–40%) of sialic acid in the plasma (Van Caneghem & Stein, 1967).

3. There is considerable leakage into the plasma of five intracellular enzymes (Plomteux, Beaumariage, Bacq & Heusghem, 1967).

4. There are early (10–20 min) and rapidly repaired (1–2 hr) lesions of the mitochondria and endoplasmic reticulum (Hugon, Maisin & Borgers, 1966; Firket & Lelievre, 1966).

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).

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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.