a reaction that is non-essential in the short term for the survival of the cell, and that as one consequence of this primary injury there is a secondary disturbance, for example, the entry of sodium and water into the cell. It is this secondary disturbance which is prevented so long as phenergan is present. It seems that the primary injury caused by carbon tetrachloride will continue in force, after its concentration has dropped, until the vital constituents of the damaged system are re-synthesized. This process appears to take up to 48 hr.

The hypothetical secondary disturbance could be associated with the breakdown of phosphoprotein, a system which Judah[5] has shown to be sensitive to the action of phenergan and has postulated as part of the mechanism controlling cellular water and electrolyte movements.

The reason for stating that the primary site of attack of carbon tetrachloride is a system not immediately essential for the life of the cell is that, in the presence of phenergan, the damage caused by carbon tetrachloride is reversible. It is clear that this conclusion carries with it some important implications, including therapeutical possibilities.

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Alloxan and Hypersensitivity

During the course of another investigation, it was found that a diabetogenic dose of alloxan (175–200 mgm./kgm.)[1] protects approximately 50 per cent of rats sensitized to horse serum and later injected intravenously with this antigen. The time of death was lengthened in those rats suffering lethal shock. Controls consistently showed 0–10 per cent survivors.

Rats were of Sprague–Dawley strain, sensitized by a single intraperitoneal injection of a mixture of horse serum and *H. pertussis*. Alloxan was injected subcutaneously into the sensitized rats just prior to, or four days before, challenge with horse serum. Of the various routes tested, the subcutaneous route for injection of alloxan proved the least toxic. The purpose of this communication is to describe some aspects of the effect of alloxan on hypersensitivity reactions in the rat and guinea pig.

One unit of insulin, injected subcutaneously into alloxan-treated sensitized rats 1 hr. before challenge, partially reversed the protective effect of the alloxan. This amount of insulin also increased the speed with which death occurred in untreated sensitized controls (Fig. 1). Ganley[2] recently reported similar findings in mice.

In the guinea pig, alloxan injected either intraperitoneally or subcutaneously at 300 mgm./kgm. body weight four days prior to challenge reduced the size and intensity of the skin reaction to PPD in animals sensitized by injection of killed *M. tuberculosis* in paraffin oil emulsion. Long, Miles and Perry found a similar action of alloxan in guinea pigs infected with living *BCG*[3]. Alloxan did not protect against either 'immediate' or 'delayed' type anaphylaxis. However, it did appear to give protection during the first 24 hr. to the type of shock induced by intraperitoneal challenge with old tuberculin. Failure to protect from anaphylactic shock in the guinea pig may be due to the very swift occurrence of emphysema of the lungs and death from bronchospasm.

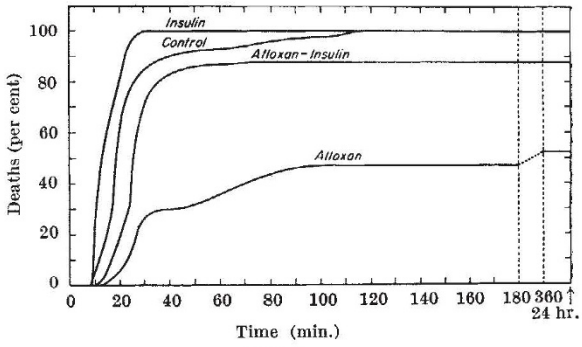

Fig. 1

The protective effect of a diabetogenic dose of alloxan in rat anaphylaxis and delayed skin hypersensitivity in the guinea pig, with its reversal in the rat by insulin, suggests the involvement of a lack of insulin or an excess of glucose. We have evidence that large amounts of glucose (4 gm./kgm. × 2) injected intraperitoneally prior to challenge gives at least some degree of protection.

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ANATOMY

Function of the Fœtal Thyroid of the Rabbit with regard to Cholesterol Metabolism studied by Fœtal Thyroidectomy

In earlier communications, it was reported that the fœtal pituitary–thyroid system influenced cholesterol and fat metabolism. Following removal of this system, a significant rise in the serum cholesterol occurred[1], and also in the fat content of the fœtal liver[2]. The technique for removing the fœtal pituitary and thyroid involves decapitation of the fœtus either through the neck to remove both the thyroid as well as the pituitary, or through the mouth to preserve the thyroid. It has already been shown that this operation has no significant effect on the weight development of the fœtus[2–4], but it may be objected that the metabolic changes may be the result of removing structures other than the pituitary and the thyroid, such as the brain.

To overcome this objection and to investigate this problem further, the fœtal thyroid was removed