that epithelial damage is the principal lesion in these two experimental models of glomerular disease, and that epithelial cell damage is likely to be an important prognostic factor in clinical renal disease. This hypothesis can and should be tested by scanning electronmicroscopy.

STUDIES ON MUSCLE IN MALIGNANT HYPERPYREXIA

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The purpose of the investigation was to examine the nature of the muscle disease which predisposes to malignant hyperpyrexia.

Muscle biopsies were taken from the vastus lateralis in 12 individuals from five families, who were shown by previous investigations to be susceptible to malignant hyperpyrexia. A detailed study was made on these muscle samples, *in vitro*, of the effects of caffeine, halothane, succinylcholine, potassium chloride and procaine. The muscle was also processed for histochemistry and electronmicroscopy.

The most striking *in vitro* abnormality in all the affected individuals was a pronounced contracture of the muscle when exposed to halothane. This does not occur in normal individuals. Succinylcholine also induced an abnormal muscle contracture, and the contractures induced by caffeine and potassium chloride were greater than normal. Procaine inhibited the contractures produced by any of the agents used.

Histochemical and electronmicroscopic abnormalities characteristic of central-core disease were found in two of the five families studied, but in the other three only mild non-specific myopathic changes were seen.

These studies suggest that the essential abnormality in malignant hyperpyrexia is an abnormal release by general anaesthetic agents of large amounts of calcium from the sarcolemmal membrane. The resulting increase in calcium concentration in the muscle cells leads to hyperpyrexia, lactic acidosis and muscle rigidity. The heterogenity of the pathological findings in the present investigation support the suggestion made, after previous clinical studies, that several different types of myopathy have the muscle membrane abnormality which predisposes to malignant hyperpyrexia.

LOW DOSE INTRAVENOUS INSULIN INFUSION — AN ADVANCE IN THE THERAPY OF DIABETIC HYPERGLYCAEMIA

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The mortality from diabetic ketoacidotic hyperglycaemia remains significant and is probably due in part to profound hypoglycaemia and potassium fluxes that may result from conventional insulin therapy. The purpose of this study was to examine the clinical and biochemical effects of low dose insulin infusions in hyperglycaemic diabetic patients.

In addition to fluid and electrolyte replacement, neutral insulin was administered by an intravenous infusion from a syringe pump or controlled drip to eleven such patients, three of whom also had proven ketoacidosis, at one or more of the following rates of 20, 40 80 and 160 mU/min (1.2, 2.4, 4.8 and 9.6 U/hour). Venous blood samples were collected frequently for plasma glucose estimation in all cases and for serum insulin, serum growth hormone and plasma cortisol estimation in some cases.

This therapy resulted in a prompt and continuous decline in plasma glucose in most patients at a rate varying between 50 and 150 mg/100 ml/hour. Increasing the insulin infusion rate above 40 mU/min did not appreciably accelerate the rate of fall in plasma glucose. As found by other workers, plasma cortisol and serum growth hormone were generally low initially despite the metabolic stress, but rose soon after the onset of insulin infusion.

The timing of attainment of euglycaemia was predictable. When the plasma glucose was satisfactory the infusion was stopped and hypoglycaemia was not encountered. Despite rapid falls in plasma glucose in some patients, this therapy was not complicated by cerebral oedema or profound hypoglycaemia.

Several patients who had previously been treated with insulin had a slower rate of fall of plasma glucose, presumably as a result of insulin-antibody interaction. A loading dose schema for such patients is suggested.

The intravenous infusion of low doses of insulin is an improvement in the therapy of diabetic hyperglycaemia with or without ketoacidosis. This therapy is simple to initiate, has a predictable end-point and prevents hypoglycaemia.

THE PHYSICIAN'S ROLE IN THYROID CARCINOMA

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Although often in the past considered a purely surgical disorder, the management of thyroid carcinoma is now firmly in the realm of the physician, since it is he who is responsible for its precise pre-operative diagnosis, post-operative care and early detection and management of recurrence. The possibility of malignancy, first raised on clinical grounds, is heightened by the demonstration of non-function in a nodule, by routine technetium thyroid scanning.

Further diagnostic information is obtained by next scanning with radio-Caesium-131 (^{131}Cs), an isotope suggested to preferentially accumulate in highly cellular structures. When a nodule accumulates ^{131}Cs to a greater extent than the rest of the gland, it is classified as "hot"; if it accumulates ^{131}Cs equal to or less than the surround-