hyperacidity in patients with acute gastroduodenal disease suggests that acid may be one of the factors potentiating mucosal injury in these patients and may be particularly important in the evolution of life-threatening ulceration.

Rosenthal A., Czaja A. J. and Pruitt B. A. (1977) Gastrin levels and gastric acidity in the pathogenesis of acute gastroduodenal disease after burns. *Surg. Gynecol. Obstet.* **144,** 232.

Fluid therapy with hypertonic lactate saline

Twelve patients with burns covering approximately 62 per cent of the body surface were treated with various lactate solutions hypertonic with respect to the sodium ion (sodium concentrations of 300, 250, 200 and 150 mmol/l). Twenty-six other patients with comparable burned areas and body weights, forming a control group, were treated with isotonic lactate, saline solutions. Both the isotonic and hypertonic solutions were given at rates sufficient to produce urine volumes of between 30 and 50 ml/h in adults and proportionately less in children. Studies of these various inputs of 'saline' indicated:

1. That the two groups of patients received almost equal inputs of the sodium ion whereas the total fluid input received by the patients given the hypertonic therapy was only about two-thirds of that received by the patients receiving isotonic therapy.

2. That the hypertonic form of therapy maintained sufficient functional extracellular fluid volume during the 'shock' period yet limited its rise during the period after active resuscitation. (The functional extracellular fluid volume was measured with ^{35}Sulphate and an equilibrium time of up to 70 min.) In contrast, very large extracellular fluid volumes were observed in the patients receiving isotonic therapy.

It should be emphasized that the treatment with hypertonic fluids was not without hazard. In some patients the serum sodium concentrations exceeded 170 mmol/l and the serum osmolalities were over 350 mosmol/kg. While the mortality rate in the two groups of patients was almost identical, death from pulmonary oedema was less common in the group of patients receiving hypertonic therapy.

Shimazaki S., Yoshioka T., Tanaka N. et al. (1977) Body fluid changes during hypertonic lactated saline solution therapy for burn shock. *J. Trauma* **17,** 38.

ANIMAL STUDIES

Phagocytosis in burns

Variations in phagocytic activity were measured for 9 days in burns of rats covering 10 per cent of the body surface. Phagocytic activity was measured in peripheral blood, spleen and liver by measuring both the clearance of bacteria (*Salmonella typhimurium*) and the bactericidal index of macrophages. A considerable reduction of the bactericidal index was observed within 12 h of burning. The decreased phagocytic activity of macrophages in the liver and peripheral blood is important but transient. The decreased phagocytic activity of splenic macophages is drastic and persists unimproved for at least 9 days after burning.

DiMaio A., DiMaio D. and Jacques L. (1976) Phagocytosis in experimental burns. *J. Surg. Res.* **21,** 437.

Cardiovascular responses to burns

Studies of several renal, cardiovascular and metabolic variables were carried out in 8 dogs with flame burns covering 25 per cent of the body surface. In order to determine the role played by the sympathetic nervous system in the genesis of kidney dysfunction during the immediate post-burn period, the dogs were treated before burning with a ganglionic blocking agent (chlorisondamine hydrochloride). Eight other dogs received a placebo before burning.

From studies made 1 h before and 4 h after burning it was determined that ganglionic blockade prevented or minimized most of the renal and cardiovascular functional changes that occur after severe burns. The degree of metabolic acidosis was also reduced. It is suggested that the sympathetic system is at least partly responsible for the state of renal and cardiovascular dysfunction that invariably follows severe burns. Oliguria probably results from a combination of factors; from a reduction in glomerular filtration rate and secretion of antidiuretic hormone. Ganglionic blockade may also offer some protection against a marked increase in vascular permeability.

Carvajal H. F., Reinhart J. A. and Traber D. L. (1976). Renal and cardiovascular functional response to thermal injury in dogs subjected to sympathetic blockade. *Circulatory Shock* **3,** 287.

Glucose turnover in burned guinea-pigs

Studies in burned guinea-pigs using 6-^{3}H-labelled glucose indicated increases in the plasma content of glucose during the burn shock phase (first 24 h after injury), the hypermetabolic phase (days 2 and 3) and the septic phase (days 4 and 5 after burning). The rate of disappearance of glucose was not elevated initially, resulting in hyperglycaemia during the first 24 h. Subsequently the rate of disappearance of glucose increased to the same extent as the rate of appearance of glucose. During the hypermetabolic and septic phases the glucose turnover rate was elevated but the plasma glucose concentration was normal. Glucose turnover was poorly correlated with total body oxygen consumption, suggesting that not all of the increased turnover of glucose was due to increased glucose oxidation.

Wolfe R. R., Miller H. I., Elahi D. et al. (1977) Effect of burn injury on glucose turnover in guinea-pigs. *Surg. Gynecol. Obstet.* **144,** 359.

LABORATORY STUDIES

Healing of burns of the skull

This histological study indicates the depth of necrosis, the incidence and effects of pyogenic infection and