ACIDOSIS IN BURNS Twenty patients in shock were divided into four groups: A) shock from severe burns with lethal outcome (six patients); B) survival from severe burn shock (seven patients); C) survival from moderate burn shock (four patients); D) shock as the result of severe trauma without burn (three patients). Lactic and pyruvic acid concentrations, *p*H, P_{CO_2} and P_{O_2} were measured in axillary-vein blood. Lactic acidemia and elevated lactate–pyruvate ratios were found in group A patients. In group B, acid and blood gas values were without prognostic significance. In groups C and D, anaerobic metabolism was less pronounced than in group A patients. Lactic acidemia developed immediately after the burn injury. Treatment, which was started immediately, consisted of replacement of fluid loss (150 ml per one per cent burned surface per 48 hours, of which half is given in the first eight hours). Colloid solutions, plasma, dextran or blood were used. Electrolytes were given by mouth if possible. Mannitol was used if oliguria occurred. Sodium bicarbonate was given when metabolic acidosis was encountered. Lactic acidemia and low *p*H generally should not be considered responsible for a poor clinical status. These changes are the result of peripheral circulatory collapse and, together with other sequelae of shock and burn trauma, determine the fate of these patients. (*Ulmer, W. T., and others: Development, Cause and Significance of Hypoxic Acid–Base Balance in Burns, Klin. Wschr. 46: 945 (Sept.) 1968.*)

EXTRACELLULAR FLUID Extracellular fluid space (ECF) and the erythrocyte volume were measured by sodium sulfate ^{35}S and sodium chromate ^{51}Cr, respectively, in two groups of dogs before and during hemorrhagic shock. Blood was removed to keep mean arterial blood pressures at steady levels of 50 mm Hg for five hours. In group I (catheters in both femoral arteries) the following decreases in the body fluids were noted: erythrocyte volume: 42 per cent; plasma volume: 28 per cent; interstitial fluid: 18 per cent; ECF: 20 per cent. In group II (catheter in one carotid artery) the decreases were: erythrocyte volume: 47 per cent; plasma volume: 35 per cent; interstitial fluid: 9 per cent; ECF: 14 per cent. The decrease in ECF which was not due to plasma loss in group I was 12 per cent; in group II, 6 per cent. This "corrected ECF" takes into account the amount of plasma lost during shock: removal of plasma bleeding lowers ECF (of which plasma is a part) by an equivalent amount. The difference between the two groups of animals is explained by failure to establish total isotope equilibrium in group I, caused by the introduction of catheters in both femoral arteries and consequent inadequate circulation distal to the arteriotomies. The decrease of 42 per cent of the ECF reported by other investigators is considered to be due to methodological and experimental errors. A significant source of error would be failure to consider that total equilibration between injected isotope and blood does not occur before 60 to 90 minutes after injection in control animals and 90 to 135 minutes after injection in shocked animals. An investigation of the ECF in hemorrhagic shock in patients would require an observation period of two to three hours without treatment and therefore is not feasible. The administration of huge amounts of crystalloid solutions for the treatment of hemorrhagic shock cannot be justified by assuming a highly depleted extracellular fluid space. (*Roth, E., Lax, L. C., and Maloney, J. V.: Changes in Extracellular Fluid and Blood Volume During Hemorrhagic Shock, Zschr. ges. exp. Med. 147: 346 (July) 1968.*)

HYPEROSMOLAR COMA Hyperosmolar coma is a syndrome characterized by extreme hyperglycemia, hypovolemia, hyperosmolality, and coma without acidosis or ketosis. The hyperglycemia causes osmolar diuresis with loss of water in excess of loss of electrolytes and urea. Renal concentrating mechanisms are lost, further perpetuating the problem. Hypovolemia, shock and neurologic dysfunction follow. Serum osmolality may be estimated as follows: $2(Na^+ + K^+)$ + blood glucose (mg/100 ml)/18 + BUN (mg/100 ml)/1.4. Treatment consists of insulin and fluid replacement. Initial fluids should be 0.45 per cent sodium chloride without glucose. Six to eight liters may be needed. Symptoms of hypokalemia