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

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should be watched for and treated. (*Tyler, F. H., Hyperosmolar Coma, Amer. J. Med. 45: 485 (Oct.) 1968.*)

Respiration

CSF OXYGEN TENSION Oxygen and carbon dioxide tensions were determined in 320 CSF samples. In a few patients, cisternal fluid P_{O_2}, P_{CO_2} and *p*H values were compared with simultaneously-obtained arterial and jugular venous blood values for calculation of the mean P_{O_2} in cerebral capillaries. Normal cisternal fluid was found to have values between those of arterial and jugular venous blood. P_{O_2} in lumbar CSF (31 mm Hg) is lower than that in cisternal fluid (47 mm Hg), and the P_{CO_2} is higher (44 *vs.* 37 mm Hg). There was significant *correlation* of the gas tensions in cisternal fluid with the mean capillary P_{O_2} and P_{CO_2} of the brain. There was, however, no evidence that cisternal fluid P_{O_2} is *representative* of mean P_{O_2} of cerebral tissue. In fluid samples with increased cell counts, P_{O_2} decreased with the degree of pleocytosis. An increase in protein was not associated with a decreased oxygen tension in CSF. AV malformations produced high P_{O_2} values in the jugular vein, provided the vein drained the shunt. In these cases, jugular P_{O_2} was occasionally found to be higher than cisternal fluid P_{O_2}. A clinical application of these findings may be the measurement of CSF oxygen tension in lieu of the carotid–jugular AV difference, thus avoiding puncture of two vessels and potential heparinization when continuous monitoring of intravascular gas tensions is indicated. In drainage of ventricles (which may extend over many hours or several days), continued monitoring of CSF gas tensions is simple, without hazard, and can provide information about perfusion and oxygen consumption of the brain. Monitoring CSF gas tensions may also be useful in the study of the effects of vasopressors or anesthetic drugs on the brain. (*Gaenshirt, H.: Oxygen Tension in Cerebrospinal Fluid of Man. Physiological and Clinical Significance, Klin. Wschr. 46: 771 (July) 1968.*)

RESPIRATORY RESISTANCE Total respiratory, lung and chest wall flow resistances were measured in spontaneously-breathing patients with obstructive lung disease by imposing flow oscillations at the airway. Total respiratory and lung resistance decreased with increasing breathing frequency. Compliance was also frequency-dependent. Such frequency dependence was interpreted as a function of uneven distribution of mechanical properties of the lungs. (*Grimby, G., and others: Frequency Dependence of Flow Resistance in Patients with Obstructive Lung Disease, J. Clin. Invest. 47: 1455 (June) 1968.*) ABSTRACTER'S COMMENT: This paper is vital not only for its conclusions but because of the thoroughness with which the technique of forced oscillations was investigated. Although more than a decade has elapsed since the oscillator technique for investigating airflow resistance was proposed, the application of this technique is apparently coming into fashion. We have witnessed cumbersome techniques such as the plethysmographic, esophageal balloon and interrupter methods. It will remain for time to determine whether the oscillator technique is better. Simultaneous determinations of lung volume should be made but, unfortunately, this remains extremely difficult in a supine, anesthetized patient.

DIFFUSING CAPACITY Changes in pulmonary capillary blood volume have been studied by various techniques. The carbon monoxide diffusing capacity (DL_{CO}) is of special interest. Since exercise causes greater increases in breath-holding pulmonary diffusing capacity than can be produced by other means, the mechanism by which this change occurs is important. The increase in DL_{CO} implies an enlargement of the effective pulmonary capillary bed. Previous studies have shown that increases in pulmonary blood flow alone do not increase DL_{CO}. Procedures which transfer blood from the peripheral circulation to the lung or increase pulmonary vascular pressure do increase DL_{CO} somewhat, primarily by increasing pulmonary capillary blood volume. Breath-holding DL_{CO} was determined in 12 normal subjects, seated and supine, at

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