extravascular pressure were kept constant. Both vasodilatation and vasoconstriction may occur during the cooling period. Active renal vasoconstriction occurred during the rewarming period secondary to release of adrenergic agents. (*Hinshow, L. B., and others: Renal Vascular Response to Hypothermia, Proc. Soc. Exp. Biol. Med. 118: 623 (Mar.) 1965.*)

HYPOTHERMIA An 11 month old child was admitted to the hospital with coma, generalized convulsions, fever, and a diagnosis of meningitis. Therapy to stop convulsions was unsuccessful. Her body temperature was lowered inadvertently to 74° F. and then elevated and kept between 94° and 96° F. for a week. This therapy rapidly controlled the convulsions and the child was discharged cured. (*Robinson, A., and Buckler, J. M. H.: Emergency Hypothermia in Meningococcal Meningitis, Lancet 1: 81 (Jan. 9) 1965.*)

HYPOTHERMIA Bile was collected from rats anesthetized with pentobarbital, both intact and nephrectomized, at various time intervals after the intravenous injection of C^{14}-atropine, and at body temperatures of 37°, 25° and 17° C. Urine was collected and analyzed at the termination of the 4 hour experiment. In the intact animal 50 per cent of the atropine appears in the bile and in the nephrectomized animals 70 per cent appears in the bile within 4 hours. Deep hypothermia appears to more markedly impair renal excretion of atropine and/or metabolites than it does hepatic excretion. At the lowest temperature studied, 17° C., the liver was capable of completely altering the material excreted in the bile. On the other hand, the kidney was unable to excrete C^{14} into the urine at 17°. (*Kalser, S. C., and others: Drug Metabolism in Hypothermia. I. Biliary Excretion of C^{14}-Atropine Metabolites in the Intact and Nephrectomized Rat, J. Pharmacol. Exp. Ther. 147: 252 (Feb.) 1965.*)

HYPOTHERMIA Of 277 patients with acute head trauma, 24 were treated by therapeutic hypothermia. Five patients soaked in ice water and 1 treated with cooled blanket were anesthetized with ether, while 1 soaked in ice water and 6 treated with cooled blanket were anesthetized with nitrous oxide. Of the 24 patients, 17 recovered and were uneventfully discharged, and 7 died. The cause of death was pneumonia in 1 patient and respiratory paralysis in 4; in 2 fatal cases therapeutic hypothermia was considered to have been protracted beyond the optimal time. The most suitable level of hypothermia was 25–30° C. and the optimal duration was less than 48 hours. (*Makino, K., and others: Therapeutic Hypothermia For Head Trauma (Japanese), Operation (Tokyo) 18: 45, 1964.*)

CENTRAL VENOUS PRESSURE Central venous pressure provides a sensitive and instantaneous picture of circulatory hemodynamics. It measures the resultant of three components which comprise the circulation, namely blood volume, cardiac pump, and the vascular bed and its resistance. Its use is advocated in every elderly patient undergoing extensive surgical procedures, and in patients who are oliguric or in those patients in whom there has been a forced diuresis. (*Borow, M., and others: Use of Central Venous Pressure as an Accurate Guide for Body Fluid Replacement, Surg. Gynec. Obstet. 120: 545 (Mar.) 1965.*)

SHOCK The two best monitors of fluid and blood volume replacement are the hourly uninary output and the central venous pressure. Blood volume determinations are useful, but they tend to be unreliable in shock. Before considering the use of either vasopressors or vasodilators, one should be certain that blood volume deficits have been corrected. In septic shock, the use of a steroid along with an adrenergic blocking agent seems indicated. Adrenergic blocking agents may be of value when used with adequate blood volume replacement, but considerable thought must be given to a technic that considers lowering an already low blood pressure in order to promote perfusion of tissue. (*Hamit, H. F.: Current Trends of Therapy and Research in Shock, Surg. Gynec. Obstet. 120: 835 (Apr.) 1965.*)

PULMONARY RESISTANCE In young calves, pulmonary vascular resistance was found to increase after total cardiopulmonary bypass, secondary to an increase in pulmonary arterial pressure without changes in cardiac

output or left atrial pressure. Hypoxia produced an equal increase in pulmonary resistance both before and after bypass, adding to the already elevated pulmonary arterial pressures after bypass. When ventilation was carried out during the time of perfusion, the rise in pulmonary vascular resistance and pulmonary arterial pressure was much less than when the lungs were left inflated but motionless during perfusion. Overventilation after cardiopulmonary bypass lowered the pulmonary vascular resistance and mean pulmonary arterial pressure and decreased the cardiac output. (*Kahn, D. R., and others: Effects of Total Cardiopulmonary Bypass on Pulmonary Vascular Resistance in the Calf, Circulation 31: 1-117 (Apr.) 1965.*)

PULMONARY RESISTANCE Effect of prolonged bubble oxygenation of blood on pulmonary vascular resistance (PVR) has been quantitated by using an excised dog lung insufflated with air and perfused via the pulmonary artery. Comparison of the vasoactive effect of fresh blood perfusate with blood that has been bubble oxygenated for 2 hours demonstrated marked elevation in pulmonary vascular resistance immediately following perfusion with the blood that had previously undergone 2 hours of bubble oxygenation. Although increased erythrocyte destruction accompanied bubble oxygenation, the vasoactive effect was not caused by free hemoglobin. Both histamine and serotonin were elevated in the blood that produced an increased PVR. This suggests, but does not prove, a causative relationship between these vasoactive substances and pulmonary vasoconstriction. (*Yong, N. K., and others: Increased Pulmonary Vascular Resistance Following Prolonged Pump Oxygenation. J. Thor. Cardiov. Surg. 49: 580 (Apr.) 1965.*)

CARDIAC OUTPUT Left ventricular outputs were computed from dye dilution curves during the performance of the Mueller and Valsalva maneuvers at different levels of intraoesophageal pressures. Output was indicated to decrease by 0.1 liters per minute with each rise of 1 mm. of mercury above zero. After an initial decrease in ventricular output of 13 per cent in the range of minus 3 to minus 7 mm. of mercury intraesophageal pressure, mean output increased by approximately 40 per cent with progressive decrease of pressure. (*O'Neill, A., Valet, L., and Cudkowicz, L.: Effect in Man of Changes in Intrathoracic Pressure on Cardiac Output, Canad. J. Physiol. 43: 203 (Mar.) 1965.*)

DECONDITIONING A problem in manned space flight is deconditioning caused by physical confinement and by decreased work load secondary to the absence of body weight. It is manifested by decreased plasma volume, decreased red blood cell mass, decreased red blood cell production, increased resting heart rate, decreased exercise tolerance, decreased orthostatic tolerance, decreased coronary blood flow, increased storage of catecholamine products in the myocardium, decreased muscle mass and muscle tone with resultant increased nitrogen excretion and increased calcium mobilization. Acclimatization produces clinical features which are exactly opposite of those noted in deconditioning: increased organ activity erythropoiesis, vagotonia with decreased heart rate, and increased coronary blood flow. Prolonged hypoxia of a sufficient degree to produce suitable acclimatization is a useful agent in preventing deconditioning during manned space flight and in those situations on earth that result in deconditioning. (*Hypoxia—An Antideconditioning Factor For Manned Space Flight, Aerospace Med. 36: 97 (Feb.) 1965.*)

BLOOD pH In patients subjected to short periods of acute acidosis and alkalosis no electrocardiographic changes of note could be described to changes in *p*H. The range of *p*H (7.30 to 7.64) is well within that seen in medical conditions and is sufficient to cause hemodynamic alterations. (*Reid, J. A., and others: Effect of Variations in Blood pH Upon the EKG in Man, Circulation 31: 369 (Mar.) 1965.*)