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.*)