($D_{L_{CO}}$), alveolar–arterial oxygen tension difference while breathing air ($A\text{-}aD_{O_2}$) and physiologic deadspace ratio ($V_D/V_T \times 100$) were studied preoperatively, immediately following open-heart surgery, on the first postoperative day, and 20 days later in 12 patients with severe mitral stenosis. Average values for $D_{L_{CO}}$ were 9.8, 4.1, 5.6, and 6.9 ml/min/mm Hg, respectively. Total $A\text{-}aD_{O_2}$ values were 31, 52.5, 49.8, and 35.2 mm Hg. The corresponding non-shunt components of the $A\text{-}aD_{O_2}$ were 25.3, 38.6, 40.9, and 23.8 mm Hg. $V_D/V_T \times 100$ values were 47, 53, 56, and 49 per cent. Following cardiac bypass the distribution of pulmonary blood flow is uneven, as evidenced by a decrease in pulmonary diffusing capacity, an increase in physiologic deadspace, and an increase in the non-shunt component of $A\text{-}aD_{O_2}$. (*Kaplan, S. L., and others: Effect of Cardiac By-pass on Pulmonary Diffusing Capacity, J. Thorac. Cardiov. Surg. 57: 738 (May) 1969.*)

OXYGEN TOXICITY Pure isobaric oxygen therapy for 72 hours produces pulmonary hyaline membranes in guinea pigs. Coagulation profiles were made for guinea pigs after 48 hours of inhalation of pure oxygen to determine whether hyaline-membrane formation is preceded by changes in the clotting or fibrinolytic systems. Statistically significant decreases in total profibrinolysin, free profibrinolysin and fibrinolytic inhibitor were found. Other changes in the coagulation profile suggested partial activation of the clotting mechanism. These results demonstrate that deficient fibrinolysis precedes the development of hyaline membranes, and suggest a causal relationship between hyaline membrane disease and deficient fibrinolysis. In neonatal and adult human hyaline membrane disease, deficient fibrinolysis has been demonstrater after the membranes already have been formed. (*Phillips, L. I., and others: Fibrinolytic Deficit in Oxygen Intoxicated Guinea Pigs, Aerospace Med. 40: 744 (July) 1969.*)

RESPIRATORY CARE The Bird Mark 7 respirator (IPPB) effectively treated 42 episides of severe acute respiratory failure in 32 patients with chronic obstructive pulmonary disease. Mean P_{CO_2} decreased within 24 to 48 hours by 32.5 mm Hg, from the pre-IPPB level of 80.8 mm Hg. No patient died in the first three days, only six patients (14 per cent) died within the first two weeks, 33 episodes ended with removal from the respirator, and 21 patients (50 per cent) ultimately were discharged from the hospital. Pressure-cycled respirators can be as effective as any other type of respirator, including those that are volume-cycled, if they are properly used and the results monitored by frequent arterial blood gas and *p*H measurements, with due regard to regulation of inspiratory, expiratory and oxygen flows. (*Billingham, M., and Eldridge, F.: Use of a Pressure-cycled Respirator (Bird) in Respiratory Failure Due to Severe Obstructive Pulmonary Disease, Ann. Intern. Med. 70: 1121 (June) 1969.*)

HOT-WATER DISINFECTION Oxygen therapy equipment (Bird respirator, nebulizers, Bennett monitoring spirometer and anesthetic bags and face masks) used on patients with respiratory infections (*Staphylococcus aureus, Pseudomonas pyocyaneus,* diphtheria) were dismantled, washed with commercial detergent, then immersed for 15 minutes in an instrument-boiler sterilizer with a thermostat set to keep the water between 80 and 85 C. Cultures taken after the equipment was removed from the water and allowed to cool showed complete disappearance of gram-negative bacilli, with only "skin flora organisms" growing in a few cases. This latter was attributed to handling of the equipment with bare hands after sterilization. The authors concluded that this was an efficient, economical, rapid and safe method for disinfecting such equipment. (*Roberts, F. J., Cockcroft, W. H., and Johnson, H. E.: A Hot Water Disinfection Method for Inhalation Therapy Equipment, Canad. Med. Assoc. J. 101: 30 (July) 1969.*)

OXYGEN EFFECTS ON THE EYE Increased oxygen pressure results in two overlapping groups of effects on the eye. These overlapping groups have "physiologic" or "pathologic" consequences. Included in the former category are retinal vessel constriction and reduction in the peripheral visual field. They can be expected to occur immediately

when O_2 breathing is begun and reverse promptly on return to a normal inspired O_2 tension. Among the "pathologic" effects are: enzymatic derangements, visual-cell death, retinal detachment, cytoid-body microinfarct formation and retrolental fibroplasia. These effects are considered to be secondary to the effect of true enzymatic oxygen poisoning and/or to consequences of toxic effects upon chemical systems, physiologic mechanisms or anatomic structures that persist or appear even after removal from exposure to high O_2 tension. The fact that, with the exception of retrolental fibroplasia, O_2 effects on the eye that are truly irreversible have been described in animals only emphasizes the need for further careful studies in man. (*Nichols, C. W., and Lambertsen, C. J.: Effects of High Oxygen Pressures on the Eye, New Engl. J. Med. 281: 25 (July) 1969.*)

AIRWAY RESISTANCE Esophageal pressure and respiratory flow rates were studied and correlated with respiratory volume in healthy young volunteers in the sitting position. Total viscous resistance of the lung was calculated from pressure–volume diagrams by correlating respiratory flow rates and esophageal pressure changes throughout the entire respiratory cycle in 50-ml volume increments. During inspiration there was a continuous decrease of resistance of about 20 per cent when the inspired volume increased from zero to 600 ml. During transition from inspiration to expiration, a significant abrupt increase in resistance of about 50 per cent (from 1.15 to 1.71 cm H_2O/100 ml) was noted. During expiration resistance increased to a maximum of 1.87 cm H_2O/100 ml, then decreased toward the initial level of 1.45 cm H_2O/100 ml at the end of expiration. The directional change of the transmural pressure and the effect of the elastic pull on the bronchial and bronchiolar diameters are considered the main factors contributing to changes in total viscous resistance. (*Berger, W.: Total Viscous Resistance of the Lung during the Respiratory Cycle, Zschr. Ges. Exp. Med. 150: 41 (May) 1969.*)

LUNG STRETCH RECEPTORS Responses of the heart, total peripheral vascular resistance, and the resistance of the innervated hindlimb perfused at a constant rate were investigated during positive-pressure inflation of the lungs. An inflation pressure of 20 mm Hg produced significant negative inotropic and chronotropic effects. Heart rate, intracardiac pressure and contractile force, measured with a Walton-Brodie strain gauge arch, all decreased. Peripheral vascular resistance decreased by an average of approximately 22 per cent and perfusion pressure in the isolated hindlimb by 26 per cent. Bilateral cervical vagotomy abolished the reflex cardiovascular responses to inflation of the lung. Lung receptors are sensitive to stretch, and the afferent pathway runs predominantly in the vagus nerves. (*Glick, G., Wechsler, A. S., and Epstein, S. E.: Reflex Cardiovascular Depression Produced by Stimulation of Pulmonary Stretch Receptors in the Dog, J. Clin. Invest. 48: 467 (March) 1969.*)

COLLATERAL CHANNELS IN HUMAN LUNGS The resistance of collateral channels in interlobar fissures was measured in eight normal and eight emphysematous excised human lungs. Similar measurements were made in other areas of normal and emphysematous lungs. Excised segments were inflated through a bronchial cannula while air leaked through collateral channels out of the other lobe or segment through a pneumotachograph measuring flow. Alveolar pressure difference producing collateral flow was also measured. By measuring inflating pressure and airway pressure at the pneumotachograph, lobar airway resistance could be calculated. In normal lungs, collateral channel resistance varied inversely with lung volume and was higher on inflation than on deflation. Airway resistance was small compared with collateral channel resistance. In emphysematous lungs, collateral channel resistance was considerably lower, and was less than airway resistance. Collateral channels are important ventilatory pathways in emphysema. When many units within a lung are ventilated by such pathways, disturbances of gas exchange and phase differences between normally- and abnormally-ventilated areas may occur. (*Hogg, J. C., Macklem, P. T., and Thurlbeck, W. M.: The Resistance of Collateral Channels in Excised Hu-*