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-*

man Lungs, J. Clin. Invest. 48: 421 (March) 1969.)

CHRONIC RESPIRATORY CARE A comprehensive-care program for patients with chronic airway obstruction (emphysema and chronic bronchitis) uses existing hospital facilities, organized outpatient care and home care, with relatively few personnel. Effectiveness depends on patient education, bronchial hygiene using simple home equipment, breathing retraining, and physical reconditioning. Portable oxygen equipment allows the participation of otherwise-very-disabled hypoxemic individuals. An intensive care unit is used for episodes of acute respiratory failure. The program has resulted in marked subjective improvement in the majority of patients and a highly significant increase in exercise tolerance. Modest but significant objective pulmonary function improvement was observed in patients after as much as a year of follow-up. *(Petty, T. L., and others: A Comprehensive Care Program for Chronic Airway Obstruction: Methods and Preliminary Evaluation of Symptomatic and Functional Improvement. Ann. Intern. Med. 70: 1109 (June) 1969.)*

EXPERIMENTAL EMPHYSEMA Inhalation or instillation of the enzyme papain into the lungs of hamsters produces pulmonary emphysema with little involvement of the conducting air passages. This experimentally-produced emphysema differs from clinical emphysema in man, which is almost always complicated by some degree of chronic bronchitis. Compliance of the lungs from emphysematous hamsters was greater than that of lungs of control animals, except near the upper limits of inflation. Pulmonary resistances in the two groups were comparable. The moderate flow limitation observed in emphysematous lungs was considered largely a result of the reduced lung recoil force. *(Park, S. S., and others: Mechanical Properties of the Lung in Experimental Pulmonary Emphysema, J. Appl. Physiol. 26: 738 (June) 1969.)*

ANESTHETICS AND PULMONARY SURFACTANT Lungs excised from dogs ventilated with halothane in air had significantly greater retractive forces at corresponding lung volumes than lungs ventilated with air or 80 per cent nitrous oxide. Chloroform gave qualitatively similar results, but the changes were not significant statistically. Increases in retroactive forces produced by both halothane and chloroform varied with inhaled concentration. These changes did not result from alterations in tissue elasticity, and were attributed to alterations in pulmonary surfactant activity caused by the presence of volatile anesthetic agents during cyclic ventilation of the lungs. Possible mechanisms include depression of surfactant synthesis, interference with replacement of surfactant, or acceleration of surfactant breakdown. *(Woo, S. W., Berlin, D., and Hedley-Whyte, J.: Surfactant Function and Anesthetic Agents, J. Appl. Physiol. 26: 571 (May) 1969.)*

POSITIVE-PRESSURE BREATHING A model of the respiratory distress syndrome in adult humans was created in dogs by injection of oleic acid into the venous circulation. Animals not receiving subsequent respiratory support developed marked hyperpnea and progressive anoxemia and died within five hours in respiratory failure. Eight of ten dogs subsequently subjected to intermittent positive-pressure breathing (subject-triggered ventilation with atmospheric pressure during exhalation) and nine of ten dogs subjected to continuous positive-pressure breathing (controlled ventilation with 10 cm H_2O end-expiratory pressure) survived the experimental period. CPPB animals had better oxygenation, lower oxygen consumption and more pulmonary compliance at lower total minute ventilation than IPPB animals. Cardiac output and oxygen transport were reduced to a greater extent with CPPB than with IPPB, but no adverse effects of the lower cardiac output were apparent. *(Uzawa, T., and Ashbaugh, D. G.: Continuous Positive-pressure Breathing in Acute Hemorrhagic Pulmonary Edema, J. Appl. Physiol. 26: 427 (April) 1969.)*