general, the exogenous pharmacologic agents inhibit cilial motion, although local anesthetics can stimulate cilial motion at low concentrations. Various diseases that affect the bronchial tract also affect cilial motion and mucus production and transport. Necrosis and cell destruction, viral infections, vitamin A deficiency, and chronic bronchitis all inhibit cilial motion to some degree. (*Okeson, G. C., and Divertie, M. B.: Cilia and Bronchial Clearance: The Effects of Pharmacologic Agents and Disease, Mayo Clin. Proc. 45: 361 (May) 1970.*)

**COLLATERAL VENTILATION** The superior segmental bronchi of the right lower lobes of anesthetized dogs were ligated and divided, and the distal bronchi cannulated. The cannulae were connected to an underwater seal and a spirometer to measure the volume of collateral ventilation. Transducers were used to measure the pressure changes in the trachea and in the bronchi that had been cannulated. In one group of dogs each lung was ventilated separately through a tracheal divider to measure effects of varying tidal volumes. In another group of dogs, ventilation was done through an ordinary endotracheal tube to measure effects of changes in tracheal pressure. Epinephrine given intravenously (0.01 mg/kg) produced a 66 to 160 per cent increase in collateral gas flow with no change in peak tracheal pressure, while intravenous methacholine (0.1 mg/kg) resulted in a 50 to 170 per cent increase in peak tracheal pressure and a reduction in gas flow to zero. Hypoxia (10 per cent $O_2$ and 90 per cent $N_2$) and hyperoxia (pure $O_2$) did not appreciably change the amount of collateral ventilation. Hypercapnia (15 per cent $CO_2$) produced marked increases in collateral ventilation and reduced the pressure gradient between the trachea and the cannulated bronchus. These results show that the principal pathways for intersegmental collateral ventilation contain smooth muscle fibers. (*Chen, C., Scaly, W. C., and Scaber, A. V.: The Dynamic Nature of Collateral Ventilation, J. Thorac. Cardiov. Surg. 59: 518 (April) 1970.*)

**PULMONARY FUNCTION** Airway resistance, compliance, alveolar–arterial oxygen gradients breathing pure oxygen, and dead-space-to-tidal volume ratios were determined in adults and children early and late in anesthesia. Operations performed included cardiopulmonary bypass and thoracic, abdominal, and surface procedures. Airway resistance increased when the pleura was opened. However, when the pleura was intact, even cardiopulmonary bypass had no adverse effects on pulmonary function. The deleterious effects of pleurotomy were abolished by humidification of the inspired gas mixture and by providing intermittent deep breaths during cardiopulmonary bypass. Pleurotomy impairs pulmonary function primarily through retraction of the lungs and through retainment of secretions. (*Ghia, J., and Andersen, N. B.: Pulmonary Function and Cardiopulmonary Bypass, J.A.M.A. 212: 593 (April) 1970.*)

**PULMONARY DISEASE** Chronic obstructive pulmonary disease in 25 patients was classified according to whether the predominant changes were loss of lung elastic recoil, suggesting primarily emphysema, or increased airway resistance, suggesting primarily chronic bronchitis. Emphysema predominated in six, seven had primarily chronic bronchitis; 12 had functionally combined disease. Impairment of steady-state exercise carbon monoxide transfer related linearly to loss of lung elastic recoil. From clinical, laboratory, and radiographic findings, the combined-disease group had the greatest sputum production, the most severe hypoxemia and hypercapnia, and the most numerous histories of respiratory insufficiency with heart failure. Maximal expiratory flow rates were limited in patients with emphysema or bronchitis, but less than in patients with combined disease. In patients with emphysema, limitation in expiratory flow was directly proportional to magnitude of lung elastic recoil, whereas in patients with bronchitis limitation of expiratory flow rates was proportional to increase in airway resistance. (*Dulfell, M., Marcus, J. H., and Ingram, R. H., Jr.: Limitation of Expiratory Flow in Chronic Obstructive Pulmonary Disease. Relation of Clinical Characteristics, Pathophysiological Types and Mechanisms, Ann. Intern. Med. 72: 362 (March) 1970.*)