version ECG reflects digitalis overdosage will manifest postcardioversion ventricular arrhythmias. The proposed mechanism for the above is that the electrical shock affects myocardial membranes resulting in a loss of intracellular potassium. When a critical loss has occurred, toxic effects of the cardiac bound glycoside ensue. To decrease the risk attending cardioversion, it is recommended that: (a) digoxin be discontinued for 24 hours prior to cardioversion; (b) longer acting drugs be discontinued for 2 days; (c) cardioversion be postponed if the precardioversion ECG shows signs of digitalis toxicity or if hypokalemia is present; (d) the least energy needed for cardioversion be employed by starting with 25–50 watt seconds. If serious ectopic ventricular beats are encountered, they may be abolished by intravenous lidocaine 50 mg., procaine amide 100 mg., diphenyl hydantoin 100 mg., or propanolol 5 mg. (*Kleiger, R., and others: Cardioversion and Digitalis, Circulation 33: 878 (June) 1966.*)

**CILIARY FUNCTION** Using precise methods it was shown that mice inhaling standardized doses of aerosolized bacteria cleared these bacteria at a predictable rate by means of mucociliary transport and alveolar macrophage mobilization and phagocytosis. These mechanisms were most depressed by alcohol injection and cigarette smoke and less so by hypoxia and barbiturates. Mucus flow velocity was reduced to one-third of control by alcohol. (*Laurenzi, G. A., and others: A Study of the Mechanisms of Pulmonary Resistance of Infection: The Relationship of Bacterial Clearance to Ciliary and Alveolar Macrophage Function, Amer. Rev. Resp. Dis. 93: 134 (March) 1966.*)

**DRUGS AND CILIA** Mucociliary transport is slowed by dehydration which causes a greater viscosity of mucus and by local anesthetics which reduce ciliary function, except hexylcaine which causes extensive mucosal sloughing. Of the local anesthetic agents tetracaine, lidocaine and mepivacaine had much less effect on ciliary activity than cocaine and hexylcaine. Cocaine increased the susceptibility of the mucosa to viral infection by 10 to 50 times. (*Bang, F. B., and others: Responses of Upper Respiratory Mucosa to Drugs and Viral Infections, Amer. Rev. Resp. Dis. 93: 142 (March) 1966.*)

**SMOKING** Among males 14 to 19, all types of respiratory illnesses were more frequent among regular smokers, especially among heavy smokers. Severe lower respiratory tract infections were nine times more common among smokers and severe upper respiratory infections were three times more frequent. Even light smokers in both categories were more susceptible to infections. Chronic bronchitis was not found. (*Haynes, W. F., and others: Smoking Habit and Incidence of Respiratory Tract Infections in a Group of Adolescent Males, Amer. Rev. Resp. Dis. 93: 730 (May) 1966.*)

**BRONCHIAL PRESSURES** Pressure was measured in a segmental bronchus and compared to simultaneous intra-esophageal pressure. Pressure changes during respiration ranged from 6 cm. of water in patients with no disease to over 80 cm. of water in patients with severe asthma, patients having the greatest clinical evidence of bronchospasm having the highest pressure excursions. The ratios of bronchial pressure to esophageal pressure ranged from 0.3 in normals to 2.83 in severe asthmatics with the greatest rise in the ratio in severe asthmatics. This change in ratio suggests that active contraction of bronchial muscle is an important factor in airway obstruction in asthma. (*Douglas, A., and others: The Measurement of Endomural Bronchial (or "Squeeze") Pressures in Bronchitis and Asthma, Amer. Rev. Resp. Dis. 93: 693 (May) 1966.*)

**LUPUS ERYTHEMATOSUS** Twenty patients with systemic lupus were found to have various pulmonary abnormalities including airway obstruction, pulmonary restriction and pulmonary vascular obstruction. Out of 17 patients studied, 16 had decreased pulmonary diffusing capacity for carbon monoxide. (*Gold, W. M., and others: Pulmonary Function in Patients with Systemic Lupus Erythematosus, Amer. Rev. Resp. Dis. 93: 557 (April) 1966.*)