exhalation permits use of a simple, objective method for calculating respiratory resistance in anesthetized patients using the time constant of exhalation. Respiratory resistance calculated using the time constant of exhalation agreed well with that calculated by the conventional technique of simultaneous measurements of transthoracic pressure and expiratory flow rate. Values for respiratory resistance during anesthesia are also in good agreement with values previously reported for conscious, spontaneously breathing subjects. (*Bergman, N. A.: Measurement of Respiratory Resistance in Anesthetized Subjects, J. Appl. Physiol. 21: 1913 (Nov.) 1966.*)

PULMONARY SURFACTANT Radiolabeled palmitic acid intravenously injected into dogs was incorporated into pulmonary phospholipids four times as fast as oleic acid. After induction of pulmonary edema with rapidly injected dextran, atelectatic areas of edematous lung having decreased surface activity showed significantly less phospholipid radioactivity than aerated areas. (*Harlan, W. R., and others: Metabolism of Pulmonary Phospholipids in Normal Lung and during Acute Pulmonary Edema, Amer. Rev. Resp. Dis. 94: 938 (Dec.) 1966.*)

PHYSIOLOGIC DEAD SPACE During light thiopental anesthesia in dogs, lowest physiologic dead spaces were associated with the highest cardiac indexes. On changing from spontaneous to controlled ventilation, increases in physiologic dead space were correlated with decreases in cardiac index. Also, a 2 mm. of mercury fall in mean pulmonary artery pressure was associated with an increased ratio of physiologic dead space to tidal volume of 0.1. Differences in physiologic dead space of dogs with healthy lungs are probably due to concomitant changes in the state of the pulmonary circulation. (*Suwa, K., Hedley-Whyte, J., and Bendixen, H. H.: Circulation and Physiologic Dead Space Changes on Controlling the Ventilation of Dogs, J. Appl. Physiol. 21: 1855 (Nov.) 1966.*)

ASTHMA Death in asthma is usually due to airway obstruction with mucous plugs that cannot be cleared by coughing. Sudden fatal decompensation may be associated with rapid, massive formation of or extension of existing mucous plugs. Deterioration in asthmatic patients is indicated by falling peak flows and rising carbon dioxide tensions. Tracheostomy or bronchoscopic lavage may then be life saving. (*Williams, M. H., and others: Sudden Death from Bronchial Asthma, Amer. Rev. Resp. Dis. 94: 608 (Oct.) 1966.*)

EMPHYSEMA Ten patients with obstructive pulmonary emphysema were placed in positions ranging from upright to 20-degree Trendelenberg. Positional changes caused no changes in blood gases, *p*H or residual volume. Vital capacity, functional residual capacity and flow rates were reduced in the 20 degree Trendelenberg position or when a 15 pound weight was placed on the lower abdomen in the supine position. (*Erwin, W. S., and others: The Effect of Posture on Respiratory Function in Patients with Obstructive Pulmonary Emphysema, Amer. Rev. Resp. Dis. 94: 865 (Dec.) 1966.*)

AMINOPHYLLINE The hemodynamic and respiratory effects of aminophylline were assessed in patients with chronic obstructive pulmonary disease without cor pulmonale. The intravenous infusion of aminophylline (1 g. in 30 minutes) resulted in significant decreases in arterial pressure, pulmonary artery pressure, right and left ventricular end diastolic pressures, peripheral and pulmonary vascular resistance. Heart rate, oxygen consumption and cardiac index all increased. There was a variable effect upon arterial oxygen saturation and a consistent increase in alveolar and minute ventilation. No improvement of the abnormal ventilation/perfusion ratios present in chronic obstructive pulmonary disease was seen. (*Parker, J. O., and others: Hemodynamic Effects of Aminophylline in Chronic Obstructive Pulmonary Disease, Circulation 35: 365 (Feb.) 1967.*)

SURGERY FOR PULMONARY BULLAE In addition to emergencies amenable to surgical treatment such as pneumothorax and bleeding, bullous emphysema should be treated by