SURFACTANT The syndrome of progressive respiratory distress, pulmonary edema, and increased pulmonary surface tension was induced in dogs by exposure to oxygen tensions greater than 550 mm. of mercury for 44.5 or more hours. Pulmonary surfactant was extracted by endobronchial washings for measurement of lipid composition and surface activity. Five of the 8 dogs studied developed respiratory distress without pulmonary edema. In these dogs, endobronchial wash surface tension was normal or slightly increased and total lipid distribution was normal. Esterified fatty acids in the lecithin fraction were consistently altered with a reduction in palmitate and total saturated fatty acids. In the three dogs who developed pulmonary edema, there was an increased surface tension, increased total lipid and protein and relatively decreased total phospholipid. Esterified fatty acids in the lecithin fraction were markedly altered with palmitate levels about one-third normal. Esterified arachidonate was present that was attributed to intra-alveolar plasma. Electron micrographs of the lung after oxygen exposure showed thickening of alveolar basement membrane and alterations in structure of the lamellar bodies of the alveolar epithelial cells. (*Morgan, T. E., and others: Alterations in Pulmonary Surface Active Lipids during Exposure to Increased Oxygen Tension, J. Clin. Invest. 44: 1737 (Nov.) 1965.*)

CEREBROSPINAL FLUID Reduction of arterial P_{CO_2} by hyperventilation reduced the rate of choroid-plexus fluid formation without changing its electrolyte composition. Elevation of arterial P_{CO_2} by 10 per cent CO_2 inhalation increased the rate of choroid-plexus fluid formation and increased by 9 mm. the difference between its sodium and chloride concentration, presumably reflecting a rise in bicarbonate ion. There was no change in the choroid-plexus fluid potassium ion. Changing P_{CO_2} on the cerebrospinal fluid side of the choroid-plexus had little effect on the electrolyte composition of the fluid being formed. Topical application of either acetazolamide (a carbonic anhydrase inhibitor) or ouabain caused a marked fall in the rate of choroid-plexus fluid formation. Acetazolamide did not affect electrolyte composition but ouabain caused a 55 per cent increase in the choroid-plexus fluid potassium ion. Although this may reflect an action of the inhibitor on the peculiar secretory process of the choroid-plexus cells, it may well represent a nonspecific loss of intracellular potassium ion of the type produced by ouabain in cells in general. (*Ames, A., Higashi, K., and Nesbett, F. B.: Effect of P_{CO_2} Acetazolamide and Ouabain on Volume and Composition of Choroid-Plexus Fluid, J. Physiol. 181: 516 (Dec.) 1965.*)

PULMONARY EMPHYSEMA Nine of 15 emphysematous patients had an increased blood flow into the thorax during inspiration. In the other 6, however, flow was greatly reduced or even arrested with a simultaneous increase in transmural caval pressure and reduction in transmural atrial pressure. This was associated with gross hyperinflation of the lungs and low diaphragmatic position. Possibly this explains the peripheral edema in emphysematous patients without associated pulmonary hypertension or cardiomegaly. (*Naklyavan, F. J., and others: Influence of Respiration on Venous Return in Pulmonary Emphysema, Circulation 33: 8 (Jan.) 1966.*)

PULMONARY EMPHYSEMA In stable emphysematous patients with carbon dioxide retention, the average diurnal increase in P_{CO_2} was 8.2 ± 2.2 mm. of mercury, which is greater than the increase found in normals. Highest values are obtained upon waking, most representative values at 10 A.M. and the greatest variation among values taken at 8:30 P.M. (*Beerel, F. R., and others: Daily P_{CO_2} and pH Fluctuations in Pulmonary Emphysema with Carbon Dioxide Retention, Amer. Rev. Resp. Dis. 92: 894 (Dec.) 1965.*)

PULMONARY EMPHYSEMA On 16 of 27 occasions, 15 minutes of IPPB did not decrease arterial P_{CO_2} in patients hospitalized with acute ventilatory failure secondary to chronic obstructive pulmonary disease with hypercapnia. Failures were due to inability to increase minute volume or because of increased metabolic rate or increased physiologic dead space. (*Sukumalchantra, Y., and others: The Effect of Intermittent Positive Pressure Breath-*