thetized Man, J. Appl. Physiol. 21: 1471 (Sept.) 1966.)

PULMONARY ARTERY LIGATION The left main pulmonary artery was ligated for periods of 2 to 98 days in dogs. Studies on subsequently excised lobes revealed a 40 to 50 per cent reduction in lung volume for the first 35 days which returned to normal by 98 days. Pressure-volume studies with both air and saline showed non-inflatable areas in ligated lobes, increased retractive force of inflatable alveoli, but no increase in tissue elasticity. Minimum surface tension of saline extracts from ligated lobes remained elevated for 25 to 35 days. Chronic pulmonary artery ligation causes increase in surface tension forces of inflatable alveoli for the first two weeks as well as mechanical obstruction of airways for the first month. These changes revert to normal after 50 days of pulmonary artery ligation. *(Chernick, V., Hodson, W. A., and Greenfield, L. J.: Effect of Chronic Pulmonary Artery Ligation on Pulmonary Mechanics and Surfactant, J. Appl. Physiol. 21: 1315 (July) 1966.)*

PULMONARY VENTILATION Studies in 14 patients with chronic obstructive disease of the lungs were made at rest, during intermittent positive pressure breathing, and during voluntary hyperventilation. Arterial blood gas changes measured during air breathing were correlated with changes in nitrogen washout curves measured during oxygen breathing. Intermittent positive pressure breathing and voluntary hyperventilation had the same effect on arterial blood gases, raising arterial oxygen saturation to a normal value and lowering arterial carbon dioxide tension to a subnormal value. During both procedures there was an increase in oxygen consumption that was about four times as much per liter of added ventilation during voluntary hyperventilation as it was during intermittent positive pressure breathing. During intermittent positive pressure breathing about one-tenth of the extra ventilation was directed into the slow space. This was enough to account for the observed rise in arterial oxygen saturation. During voluntary hyperventilation the incerease in ventilation of the slow space was less than one-tenth and was not enough to account for the rise in arterial oxygen saturation. It is suggested that during voluntary hyperventilation the rise in arterial oxygen saturation was partially due to a reduced fractional perfusion of the slow space. *(Emmanuel, G. E., Smith, W. M., and Briscoe, W. A.: The Effect of Intermittent Positive Pressure Breathing and Voluntary Hyperventilation upon the Distribution of Ventilation and Pulmonary Blood Flow to the Lung in Chronic Obstructive Lung Disease, J. Clin. Invest. 45: 1221 (July) 1966.)*

SURFACE TENSION Rate of fall of lung compliance during constant volume artificial ventilation in rabbits and newborn lambs was markedly decreased by raising transpulmonary pressure by 2 cm. of water. Failure to find trapped nitrogen in the lung and the examination of histologic sections of frozen lungs indicated that the observed difference was not due to airway closure. Isoproterenol or ventilation with 100 per cent oxygen or CO_2 in air did not affect the results. Occasional large inflations which replenish surface active material are required for maintenance of low surface tension in alveoli. Increasing surface tension may cause change of air space configuration and eventually lead to atelectasis, and small changes in transpulmonary pressure may greatly affect rate of decrease of lung surface area. *(Williams, J. V., Tierney, D. F., and Parker, H. R.: Surface Forces in the Lung, Atelectasis, and Transpulmonary Pressure, J. Appl. Physiol. 21: 819 (May) 1966.)*

PULMONARY CIRCULATION Previous work has shown that distribution of pulmonary blood flow depends on relationships of pulmonary arterial, alveolar, and venous pressures. In the present study, pulmonary blood flow rose higher in isolated, erect dogs' lungs when a given volume was approached from the deflated state than when the same volume was approached from the fully expanded state. Pulmonary vascular resistance was higher in the deflation state than in the inflation state. It is concluded that surface tension changes in the alveolar lining plan an important role in