tilation and Perfusion as a Function of Body Position, J. Appl. Physiol. 21: 767 (May) 1966.)

VENTILATION DISTRIBUTION Distribution of inspired gas was studied using radioactive Xenon in isolated, perfused dogs' lungs. No change in distribution of inspired gas occurred when the magnitude of blood flow to different regions of the lung was altered by large variations in pulmonary arterial and venous pressure. When the isolated lungs were suspended in air, the relative distribution of inspired gas to upper and lower lung zones was uniform. When lungs were suspended in egg-albumin foam, to simulate the gradient in pleural pressure down the intact lung, inspired gas was distributed preferentially to upper zones at low lung volumes and to lower zones at higher lung volumes. Uneven distribution of ventilation in the human lung is not caused by the uneven distribution of blood flow, but by a vertical gradient of pulmonary pressure. *(Zardini, P., and West, J. B.: Topographical Distribution of Ventilation in Isolated Lung, J. Appl. Physiol. 21: 794 (May) 1966.)*

A-A P_{CO_2} DIFFERENCE Pulmonary artery pressure was varied in anesthetized, paralyzed, artificially ventilated subjects by rapid infusion of 1,000 ml. of saline or by head-up or head-down tilt. Arterial-alveolar carbon dioxide difference varied inversely as pulmonary artery pressure and in subjects with a mean pulmonary artery pressure in the region of 20 mm. of mercury, a-A CO_2 difference was about 1 mm. of mercury, which is comparable to that in normal, conscious subjects. The increased a-A P_{CO_2} during anesthesia may be explained by a marked reduction of pulmonary capillary blood flow in the uppermost part of the lung, a reduction that is caused by a drop in pulmonary perfusion pressure. *(Askrog, V.: Changes in the (a-A) CO_2 Difference and Pulmonary Artery Pressure in Anesthetized Man, J. Appl. Physiol. 21: 1299 (July) 1966.)*

A-A P_{CO_2} DIFFERENCE When inspired gas is changed from air to oxygen in conscious subjects, increase in ventilation occurs, alveolar carbon dioxide tension falls, arterial carbon dioxide tension increases and arterial to alveolar carbon dioxide difference increases. The predominant effect is on alveolar carbon dioxide tension. Although the Haldane effect is present, the most important cause for the observed increase in a-A CO_2 tension difference is change in distribution of ventilation perfusion ratios secondary to shift of blood flow during oxygen breathing. *(Lenfant, C.: Arterial-Alveolar Difference in P_{CO_2} During Air and Oxygen Breathing, J. Appl. Physiol. 21: 1356 (July) 1966.)*

ATELECTASIS Four of 7 subjects exposed to 100 per cent oxygen at sea level and at several simulated altitudes for varying periods of time exhibited signs of pulmonary atelectasis as manifested by decreased vital capacity and/or X-ray evidence of areas of plate-like atelectasis. Individuals with alterations of pulmonary function predisposing to air trapping may have increased susceptibility to atelectasis during oxygen breathing. Absorption atelectasis occurring during oxygen breathing may be prevented by addition of 5 to 30 per cent nitrogen to the oxygen. *(Dubois, A. B., and others: Pulmonary Atelectasis in Subjects Breathing Oxygen at Sea Level or at Simulated Altitude, J. Appl. Physiol. 21: 828 (May) 1966.)*

SHUNTING Physiological shunt exceeding 8 per cent of cardiac output was uniformly observed in anesthetized, artificially ventilated subjects during hypocapnia. When carbon dioxide was added to the inspired mixture, the resulting elevation in arterial carbon dioxide tension was accompanied by an increase in arterial oxygen tension and a decrease in shunt. There changes occurred during use of both 40 per cent and 100 per cent oxygen in the inspired gas and when the cardiac output was unchanged. If cardiac output changed, then the magnitude of shunt changed in a similar direction and the effect of changing cardiac output interacted with or overrode the effect of carbon dioxide. The explanation for the observed changes is obscure. *(Michenfelder, J. D., Fowler, W. S., and Theye, R. A.: CO_2 Levels and Pulmonary Shunting in Anes-*

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 increase 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