

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 car-

bon 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. These 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-*