**GAS EXCHANGE AT ALTITUDE** Pulmonary gas exchange was studied in seven healthy volunteers at a simulated altitude of 15,000 feet (barometric pressure = 430 mm Hg). During the first three hours at altitude, arterial oxygen and carbon dioxide tensions decreased and the alveolar–arterial oxygen tension differences increased progressively. A possible explanation for the observed changes in oxygenation is the development of a diffusion barrier for oxygen due to pulmonary congestion associated with the hypoxic state. The lowest values for $Pa_{O_2}$ at altitude occurred during sleep. Although $A\text{-}aD_{O_2}$ increased during exercise, periods of exercise were well tolerated. Pulmonary arterial pressure increased on going to atitude and increased further with exercise at altitude in the one subject who had right-heart catheterization. Most subjects reported hypoxic symptoms, and one developed a persistent dry cough. (*Reeves, J. T., and others: Increased Alveolar–Arterial Oxygen Difference during Simulated High-altitude Exposure, J. Appl. Physiol. 27: 658 (Nov.) 1969.*)

**DYSPNEA** Inappropriate postexercise hyperventilation resulted in dyspnea in ten patients in whom physical examinations and roentgenograms of the chests showed no abnormalities. Chronic obstructive lung disease, pulmonary vascular disease, and interstitial pneumonitis were excluded because mechanics of breathing and respiratory gas exchange were normal. Three minutes after completion of the exercise $P_{CO_2}$ values averaged 23 mm Hg, compared with 37 mm Hg for normals and 31 mm Hg for patients with diffuse pulmonary disease. The decreased $P_{CO_2}$ in the airways produced bronchospasm that could be reversed by adding carbon dioxide to the inspired gas. Dyspnea appeared to result from increased work of breathing, in part from hyperventilation and in part from increased resistance to air flow. In some, the postexercise hyperventilation could be related to anxiety over previous chest disease, which had been slight and from which they had recovered. It is suggested that exercise-induced asthma may be part of the hyperventilation syndrome. Therapy consisting of reassurance and physical training was effective. (*Ferguson, A., Addington, W. W., and Gaensler, E. A.: Dyspnea and Bronchospasm from Inappropriate Postexercise Hyperventilation, Ann. Int. Med. 71: 1133 (Dec.) 1969.*)

**HYPERTENSION AND VENTILATION** Stimulation of arterial baroreceptors in animals causes a depression of ventilation. In nine healthy subjects, phenylephrine was given intravenously to increase arterial blood pressures transiently 15 to 25 mm Hg. Breath-by-breath ventilation was recorded by open-circuit spirometry. The subjects were seated on a Krogh ergometer at rest or exercising, breathing oxygen-rich or oxygen-poor mixtures ($Pa_{O_2} = 220$ or 50 torr). In 135 tests, ventilation decreased as arterial pressure increased on 100 occasions, the depressions being greater than 5 per cent 76 times and greater than 10 per cent 48 times. The average depression was 7.4 per cent. Ventilation was more than 5 per cent greater during than before the pressure rise in only 13 tests, and 10 per cent greater in only three. Since the majority of observations were made during hyperoxia, when chemoreceptor activity was presumably minimal, the effect on ventilation probably can be attributed to a direct reflex effect of the pressure increase on the baroreceptors. (*Cunningham, D. J. C., and others: The Effect of Raising Arterial Blood Pressure on Ventilation in Man, J. Physiol. (London) 204: 89P (Oct.) 1969.*)

**$A\text{-}aD_{O_2}$ DURING ANESTHESIA** Respiratory function and circulatory function were measured in a group of healthy patients before, during, and after anesthesia with halothane and oxygen administered for surgical procedures. Respiration was spontaneous throughout. In conscious, premedicated patients, $A\text{-}aD_{O_2}$ and physiologic shunt during breathing of oxygen were within normal limits. During anesthesia, cardiac output decreased, but a parallel decrease in oxygen consumption resulted in an unchanged arteriovenous oxygen difference. Physiologic shunt increased significantly during anesthesia. Most values had returned to normal by the third hour after termination of anesthesia. Increased alveolar–arterial oxygen tension differences during anesthesia were attributed to