

**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-a $D_{O_2}$  increased during exercise, periods of exercise were well tolerated. Pulmonary arterial pressure increased on going to altitude 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., Ad-

dington, W. W., and Gaensler, E. A.: *Dyspnea and Bronchospasm from Inappropriate Post-exercise 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-a $D_{O_2}$  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-a $D_{O_2}$  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

shunt-like intrapulmonary changes. Persistence of these changes is an important factor in the decreased arterial oxygen tension in the early postoperative period. (Marshall, B. E., and others: *Pulmonary Venous Admixture before, during, and after Halothane: Oxygen Anesthesia in Man*, *J. Appl. Physiol.* 27: 653 (Nov.) 1969.)

**A-aD<sub>O<sub>2</sub></sub>** DIFFERENCE Respiratory gas exchange was measured in a group of healthy subjects and in patients with pulmonary disease during breathing of air at ambient pressure and during breathing of oxygen at a simulated altitude. In both conditions, inspired oxygen tensions were equal at about 145 mm Hg. In every subject alveolar-arterial oxygen tension differences were less during oxygen breathing at altitude than during breathing of air at ambient pressure. In one subject who had been studied previously during breathing of 1.44 per cent oxygen in helium at a simulated pressure of 14.6 atmospheres (P<sub>IO<sub>2</sub></sub> = 149), A-aD<sub>O<sub>2</sub></sub> was greater under hyperbaric conditions than during breathing of air at ambient pressure. The observed results were attributed to the effect of ventilation-perfusion abnormalities on alveolar inert gas concentration, and provide a method for quantitating ventilation-perfusion abnormalities. An incidental observation was that physiologic deadspace increased during breathing of oxygen at altitude. (Overfield, E. M., and Kylstra, J. A.: *Distribution Component of Alveolar-Arterial Oxygen Pressure Difference in Man*, *J. Appl. Physiol.* 27: 634 (Nov.) 1969.)

**OXYGEN TRANSPORT BY FLUOROCHEMICALS** Oxygen is extremely soluble in certain synthetic fluorine compounds. The present study was designed to determine whether fluorochemicals in dispersed form might carry out the function of oxygen transport *in vivo*. Frogs whose blood contained fluorochemicals survived longer in carbon monoxide atmospheres than control non-treated frogs. Mice which had received intravascular infusions of fluorochemicals appeared to maintain consciousness longer and survived longer in CO than did non-treated mice. Thus, fluorochemicals were able to transport appreciable quantities of oxygen when hemoglobin

was inactivated by CO. The extreme chemical inertness of fluorochemicals renders it unlikely that their site of action in prolonging survival was at the tissue cellular level. (Soloviter, H. A., and others: *Dispersed Fluorochemicals as Substitutes for Erythrocytes in Intact Animals*, *J. Appl. Physiol.* 27: 666 (Nov.) 1969.)

**HYPEROXYGENATION** Thirteen elderly male patients with measured cognitive deficits were treated with 30 intermittent exposures to pure oxygen at 2.5 atmospheres absolute. Five controls, each exposed at the same time and in the same manner as a paired experimental patient, breathed a low-oxygen mixture that maintained essentially normal alveolar oxygen tension despite increased ambient pressure. Arterial samples for blood gas determinations were obtained from each patient during one of the early hyperbaric exposures. Analysis showed the expected large intra-exposure increase in arterial oxygen tension in the experimental subjects. Negligible changes in arterial oxygen tension were found in the control patients. Posttreatment performances on psychological tests of cognitive function showed highly significant gains over pretreatment levels in experimental subjects, suggesting an improved performance that persisted beyond the temporary increase in arterial oxygen levels. Control patients showed no improvement in posttreatment cognitive function. (Jacobs, E. A., and others: *Hyperoxygenation Effect on Cognitive Functioning in the Aged*, *N. Eng. J. Med.* 281: 753 (Oct.) 1969.)

**RUBEN VALVE** The considerable re-breathing often seen with the Ruben valve can be eliminated by decreasing the size of the valve disk that closes at the end of inspiration. (Vogel, H., Hakim, A., and Pfluger, H.: *Re-breathing during Use of Ruben Valves*, *Der Anaesthetist* 18: 247 (Aug.) 1969.)

**ISOLATED DOG LUNG METABOLISM** Oxygen consumption, CO<sub>2</sub> production, glucose utilization, lactic acid production, glycogen deposition and plasma free fatty acids were measured in isolated perfused canine lungs respired with 5 per cent CO<sub>2</sub> in air under conditions of normal and elevated plasma glucose levels and subsequently after the addition