

determined in a series of both normal subjects and cardiac patients. Diffusing capacity was significantly lowered in severe mitral stenosis with pulmonary congestion but was insignificantly affected by mild or moderate cardiac lesions. Capillary blood volume was elevated in severe mitral stenosis and in interatrial septal defects. Aortic valvular lesions had little effect on either of these parameters. (Flatley, F. J., and others: *Pulmonary Diffusing Capacity and Pulmonary Capillary Blood Volume in Normal Subjects and in Cardiac Patients*, *Amer. Heart J.* 64: 159 (Aug.) 1962.)

**PULMONARY EXCHANGE** During anesthesia with barbiturates or barbiturates combined with ether there was in some subjects a decrease of arterial oxygen pressure and an increase in the alveolar-arterial oxygen difference. A slight respiratory acidosis developed but there was no significant change in bicarbonate. Functional dead space increased independent of respiratory volume. During anesthesia there may be marked disturbance of distribution and pulmonary shunting in some parts of the lung. (Reichel, G., and Harfeldt, H. P.: *Investigations Concerning the Disturbances of Gas Exchange in the Lungs Caused by Anesthesia*, *Der Anaesthetist* 11: 231 (July) 1962.)

**APNEA** Intravenous administration of a barbiturate causes a short period of apnea if 100 per cent oxygen is being breathed but very little respiratory depression with absence of apnea when room air is being breathed. Sodium bicarbonate, given intravenously, has the same alkalinity and shows identical results. Respiration is not influenced for a short time when the oxygen binding power of the hemoglobin is blocked by carbon monoxide or sodium nitrite but respiration stops as soon as the physically dissolved oxygen has been used up. This shows the importance of the physically dissolved oxygen which together with blood carbon dioxide regulates respiration. If a strongly alkaline solution is given intravenously, blood carbon dioxide is bound. If at the same time by breathing pure oxygen the plasma carries five times more oxygen than normal, then two substances which stimulate

respiration are removed. (Lenggenhager, K.: *Explanation of a Strange Phenomenon During Narcosis*, *Der Anaesthetist* 11: 181 (June) 1962.)

**RESPIRATORY CHEMOREFLEXES** Existence of a respiratory chemoreflex mechanism requires the demonstration of (1) an anatomical receptor structure and afferent fibers in the brain stem, (2) sensitivity of this receptor in response to normal chemical changes in its environment, and (3) ventilatory changes which may be induced by blocking conduction in afferent fibers. On the basis of studies in both animals and adult humans it is probable that the fall in ventilation during transient hyperoxia and the ventilatory increase during transient hypoxia indicate that there is a chemoreceptor drive of ventilation. In humans, in short-term altitude acclimatization or acute hypoxia a powerful ventilatory chemoreflex oxygen drive exists. Although both chemoreflex carbon dioxide and pH drive have been established, it is not presently possible to differentiate this effect from their centrogenic drive. (Dejours, P.: *Chemoreflexes in Breathing*, *Physiol. Rev.* 42: 335 (July) 1962.)

**EFFECTS OF ALTITUDE** At an altitude of 25,000 feet, barometric pressure was 288 mm. of mercury, the inspiratory oxygen tension of warmed saturated air was 50 mm. of mercury, the oxygen tension of alveolar air was 33 mm. of mercury and that of carbon dioxide, 14 mm. of mercury. Arterial oxygen saturation was about 57 per cent at rest. The basal metabolic rate and the ventilatory equivalent for oxygen were the same as at sea level. Cheyne-Stokes respiration was prevalent during sleep. Even after three months at 19,000 feet the main respiratory drive was still furnished by hypoxia. With increase in altitude maximum work, maximum oxygen intake, maximum heart rate and cardiac output declined though maximum work of ventilation increased even though air density falls about 20 per cent at 20,000 feet. In subjects working to exhaustion at 19,000 feet, arterial oxygen saturation fell to 44 to 50 per cent and was associated with impaired oxygen diffusion at the low alveolar oxygen tension and with a large (20 to 30 mm. of mercury) alveolar-