

of carbon dioxide, and to the exclusion of excitations of the carotid bulb, hypoxemia due to reduced oxygen pressures in the circulating blood. At altitude, therefore, a dilemma soon arises. Ventilation increases in response to the lowered oxygen pressure. This washes out carbon dioxide from the lungs and blood with the result that ventilation falls off again. Thus a vicious cycle of fluctuating respiration sets in, with a progressive drop in oxygen pressure and saturation of the blood until central nervous system metabolism is so depressed that dizziness, function and failure occur. Cyclic respiration has been described frequently as an early symptom of mountain sickness and is symptomatically equivalent to Cheyne-Stokes breathing. At intermediate altitudes, breathing air and at equivalent altitudes, breathing oxygen, it is observed that equilibrium in respiration may be attained by normal individuals in such a manner that effective ventilation for oxygenation may be maintained by the adjustment of CO_2 level in the blood and accommodation of the respiratory centers to this altered situation. A concise exposition of alveolar gases and ventilation phenomena has been formulated by Fenn, Rahn, and Otis⁸⁰ with numerous charts illustrating the effects of different levels of altitude. In practice this information may be applied industrially at high terrestrial altitudes, for example, in mining, but the use of pressurization and supplementary oxygen in commercial aviation makes it necessary for consideration only in emergencies.

While the acute response to oxygen lack may be unconsciousness and ultimate death, the more chronic exposure to low-grade hypoxia may produce a variable response ranging from inefficiency in both muscular control and mental processes to disturbances of central nervous system structures, inducing a neurocirculatory collapse, or at higher altitudes a peripheral circulatory collapse, which results in a state of shock with its attendant progressive deterioration.⁸¹ It is probable that nearly all these derangements have a common mechanism rooted in general tissue hypoxia,⁸¹ but the variable responses that can be elicited from such a complex structure take on such a confusing aspect that analysis to a single cause is not practicable.⁸²

2. Hypocapnia

As suggested above, the partial pressure of carbon dioxide undergoes change at altitude as a secondary response to the hypoxia. Its effects are nevertheless quite marked and unfortunately unadapted to the circumstances. The loss of carbon dioxide from the blood can be seen from Figure 4 to cause more oxygen to be combined with hemoglobin at the same pressure of oxygen than before such a depletion. While this may at first seem to be satisfactory in increasing the

transport of oxygen, it will be effective in the tissues only if the carbon dioxide pressure is maintained or raised through increased metabolism. The hemoglobin will have a correspondingly increased affinity for oxygen in the capillaries if the partial pressure of carbon dioxide is decreased there as in the lungs. Unfortunately, the conditions that cause the hypoxia are not metabolic and thus carbon dioxide partial pressure falls throughout the body. This falling of the pressure brings on a depression in ventilation discussed above, and also a train of other effects characteristic of this condition.¹¹ Thus, hypocapnia brings on peripheral sensory disturbances described as a tingling, especially of the hands, apprehension and considerable psychological changes which are precursors of a more drastic central nervous system response, often inducing strong autonomic stimulations resulting in vomiting. The latter is particularly hazardous in aviation because the presence of a mask will result in aspiration of the vomitus and complete asphyxia unless immediate aid is rendered.⁸

If the hypoxia caused by breathing ambient air at altitude is avoided by breathing oxygen, then normal carbon dioxide pressures will be maintained; but as higher altitudes are reached, the internal maintenance of carbon dioxide pressure through metabolic activity imposes a growing restriction that has been suggested previously; that is, the partial pressure of carbon dioxide tends to remain constant and thus becomes a larger and larger part of the total pressure and in effect prevents the entrance of sufficient oxygen into the lungs at critical levels.

3. Water Vapor of the Lungs

The considerations above of the physical effects of carbon dioxide in displacing oxygen at altitude are shared equally by water vapor that is constantly being evaporated from the lung surface. Traditionally, the assumption has been made that the gases in the lung are saturated with water vapor at body temperature, but on reflection it will be seen that the physics of this system cannot justify such a conclusion without modification, particularly at high rates of ventilation. The energy expended in evaporating water from the alveolar surface must produce a cyclic variation in temperature of the blood leaving the lung capillaries. It is also known that the saturation of the gases depends on the diffusion of water vapor throughout the space, and thus the ratio of surface to volume will influence the degree of saturation attained at any time. The facts of the case are rather stated as saturation at the temperature of the alveolar, bronchial, and tracheal gases, with respect to the time of exposure to the evaporating surface. Experimentally it has been shown that the degree of saturation of the whole lung is considerably less than calculated on the assumption of saturation at body temperature,⁸³ but there is little reason to doubt that at the alveolar surface the saturation is practically that imposed by the local temperature.

⁸⁰ W. O. Fenn, H. Rahn, and A. B. Otis, *Am. J. Physiol.*, **146**, 637 (1946).

⁸¹ W. P. Yant, J. Chornyak, H. H. Schrenk, F. A. Patty, and R. R. Sayers, *US Army Health Bull. No. 211* (1934).

⁸² G. A. Brown, C. L. Cronick, H. L. Motley, E. J. Kocur, and W. O. Klingman, *Aviation Medicine*, **7**, 157 (1945).

⁸³ G. P. Burch, *Science*, **102**, 619 (1945). L. H. Marshall and H. Specht, *Am. J. Physiol.*, **60**, 249 (1949).