

varying degree of pain. The mechanics of the openings to these spaces lends itself so well to relief of internal pressure that only with the most serious cases will more than fleeting sensations be experienced.⁷⁸

On the other hand, there is a more drastic effect to be expected from the expansion of the gases in the gastrointestinal tract. As in the case of decompression from high atmospheric pressures, there is expansion, but the effect caused by water vapor is considerably exaggerated. This is due to the fact that the vapor pressure of water is constant with the temperature of the body and since it is an increasingly larger part of the total pressure, as ascent is made, it causes an increasingly larger distension of the soft-walled gas pockets.¹⁰ Thus, on decompression from 10 atm. to 1 causes a volume increase of water-saturated air from 1 to 10.59, or a gain of a little more than half a volume over the dry expansion; while a decompression from 1 atm. to $1/10$ would result in a wet volume increase of 24.6 times, or a gain of 14.6 volumes over the dry expansion. Even at $1/6$ atm., 38,500 feet, there is a volume increase of 6.66 times rather than the fivefold dry expansion from one atmosphere. These considerations form the basis of the observation that at moderate altitudes flatus expansion can be readily handled by the normally induced movements of the smooth muscle of the gastrointestinal tract, but at an altitude of 30,000 feet or above the tendency of the smooth muscle to give way to gradually increasing internal pressures results in meteorism with all its attendant symptoms.

Besides the excruciating pain caused by these effects there is in addition a restriction of the movement of the diaphragm and thus a respiratory embarrassment. In any case, the prophylaxis of avoiding gas-forming foods and the treatment of early symptoms by massage and movement of the abdomen are soon put into practice by the individual who has the misfortune to experience this distress. According to an extensive series of uniformly conducted indoctrination ascents for Army airmen, Swann and Rosenthal⁷⁹ reported an incidence of gas pains severe enough to cause descent of 0.59 per cent; of these, less than 15 per cent occurred during ascent to 30,000 feet; 73 per cent occurred after arrival at the working level of 38,000 feet.

The most critical conditions for mechanical effects from decompression arise in the situation in which explosive decompression from pressurized cabins occurs. Under these conditions a volume change theoretically as great as 2.3 times the original volume was experienced in laboratory tests in 0.008 seconds without detectable harm. It is probable that the displacement of lung gases proceeded as indicated but the expansion of intestinal gases, if any, was somewhat less due to the tone of the muscles. Cabin pressurization, for the reasons discussed above,

⁷⁸ O. E. Reynolds, H. C. Hutchins, A. Y. Werner, and F. R. Philbrook, *U.S. Naval Med. Bull.*, 46, 845 (1946).

⁷⁹ H. G. Swann and T. B. Rosenthal, *A Survey of the Incidence of Decompression Sickness with Reference to Some Constitutional and Environmental Variants*. AAF School of Aviation Med., Randolph Field, Texas, August 8, 1944.

decreased at great altitudes because of the lack of a margin of safety in the case of decompression.¹¹ Breath holding is an obvious hazard that, although it has not occurred in these tests, might result in pulmonary emphysema, as in the submarine escape conditions cited earlier.⁵⁴

B. THE EFFECTS OF MAINTAINED LOW ATMOSPHERIC PRESSURE

1. Hypoxia

It is evident from the previous treatment of the physical nature of reduction of barometric pressure that the partial pressure of oxygen falls in direct proportion to that of the total pressure. Thus an ascent to $1/2$ atm. pressure will result in an ambient partial pressure of oxygen equal to 79.5 mm. Hg. From Figure 4 it is seen that the theoretical saturation of the blood with oxygen might be as great as 95 per cent if this were the actual partial pressure of oxygen at the lung capillaries. Because gaseous exchange takes place within the depths of the lungs, and because there is never a complete exchange of the contained gases of the lungs, the partial pressure of oxygen is subject to several factors that bring about a reduction in its magnitude. The first is a matter of dilution with the residual gas in the lungs and their large air passages; the second is an addition of water vapor to the air from the moist surfaces of the lungs. The former is a structural factor that varies from one individual to another; the latter varies only with the temperature of the evaporating surface within the lung. It becomes apparent now that the water vapor is a sort of constant that we must arbitrarily deduct from the total barometric pressure in order to find the effective pressure and that, moreover, it becomes a larger and larger part of the total pressure as we ascend. At $1/2$ atm.,¹¹ by analysis, the composition of the gases nearest the circulating blood, the so-called alveolar gas, has an oxygen partial pressure of only 36 mm. Hg, which would result in a saturation of the arterial blood of less than 70 per cent. It is this reduction in the saturation of the blood that is the primary factor in the disturbances attendant upon ascent to high altitudes, since it interferes with the adequate transport of oxygen to the tissues, particularly those of the central nervous system that have the highest rate of metabolism and the smallest reserve.

The reduction in partial pressure of the oxygen in the blood will excite the reflex mechanism of the carotid artery bulb, which excitation effects an increase in the rate of ventilation of the lungs, resulting in the removal of carbon dioxide (and perhaps some water vapor), with moderate improvement in the partial pressure of the oxygen in the lungs. While this response to lowered oxygen tension in the blood is purposeful at normal atmospheric pressure, it is rarely called upon and less effective than a parallel mechanism of the respiratory center in the medulla. The latter responds to the increase in carbon dioxide that ordinarily follows oxygen depletion in the body from muscular exertion. This mechanism is, however, also responsive to a decrement in the partial pressure