### 4. Critical Altitudes

Practically, the gases in contact with the alveolar surface are diluted by both water vapor and carbon dioxide in the ratio of their combined partial pressures to the total barometric pressure. It is found experimentally that an altitude of about 41,500 feet is critical for hypoxic symptoms when pure oxygen is being breathed.[11] When pure oxygen is being inhaled, if one assumes a carbon dioxide pressure of 40 mm. Hg and a water vapor pressure of 47 mm. Hg, the pressure of oxygen at 41,500 feet can be no greater than 45 mm. Hg, equivalent to a hemoglobin saturation of not more than 80 per cent. This condition is attained when breathing ambient air at a level not strictly definable unless assumptions concerning the ventilation rate and respiratory quotient are made, but it is usually found that the critical level for unacclimatized individuals is at about 15,000 to 16,000 feet.[11]

The altitudes at which perceptible impairment, that is, objectively demonstrable impairment, is effected under the two conditions given above vary with the tests used to demonstrate it. Thus, effects on night vision are found at even low levels and necessitate the use of oxygen from the ground up for night flying even though low-level flying is to be carried out.[11] This finding from military experience should be one of the first to be applied to civilian flight, especially in passenger transport where hazard to the pilot is carried over directly to large numbers of individuals. At 10,000 to 12,000 feet the ability to adapt the eyes to night vision and the ability of discrimination of objects is cut to about one-half when breathing ambient air.[11] The addition of enough oxygen to raise the partial pressure to that at ground level will correct this deficiency. In general, the use of oxygen is to be recommended at 10,000 feet if operations are carried on for several hours. The exact time of onset of deficiency for different individuals varies greatly and prophylaxis is based on the most conservative interval for safety, a margin of 6 hours at 10,000 to 12,000 feet, 2 hours at 12,000 to 15,000 feet, and those above 15,000 feet have been found to be compatible with reasonable safety in selected groups of men, as in the military services,[11] but more recently the use of oxygen on all flights above 10,000 feet has been ordered.[70] Considerable caution should be exercised in transferring these values to civilian flying, especially to the pilots of private craft.

### 5. Decompression Sickness

In discussing the effects of ascent it was indicated that the onset of effects from inert gas due to the supersaturation caused by ascent to altitude was often delayed. This necessitates the discussion of these effects as they appear during sustained low pressure. The similarity of this condition to that found after decompressing to 1 atm. from high pressures is great enough to enable the same general approach to be carried over directly.



Because the terminology of these effects has been rather uncritical in its origin from compressed-air experience, workers in low-pressure studies have developed more specific interpretations of the names for the phenomena which occur. Thus the development of intravascular bubbles is called "aeroembolism," while extravascular bubbles are relegated to the category of "bends," which comprises both phenomena in compressed-air terminology except for bubbles in the lungs, which in both cases are called "chokes." Reference is made to these terms only because they appear in the literature of both fields and often constitute the only detail of symptomatology. In view of the fact that the theory that gas bubbles are the cause of the pain and other sequelae to decompression is connected with some reason by investigators,[11,30,84] the term "aeroembolism" has not been subject to some criticism. It is admitted, however, that in the fulminating cases of decompression sickness bubbles are found both intra- and extravascularly, and they certainly play a large part in the derangement of the body mechanisms whether primary or secondary in point of time.[69,84–87] The fact that recompression brings immediate relief lends great weight to the bubble theory of decompression sickness, whether of extra- or intravascular character.[11]

The appearance of bubbles in the vascular system has been shown to occur in animals at 45,000 feet with great regularity even in the presence of oxygen breathing throughout the ascent.[86] These observations indicate that the removal of inert gases from the body is not fast enough to prevent the formation of inert-gas free-gas phases and that once these bubbles are formed they continue to grow until they can be demonstrated as circulating or embolic entities and within body tissues. Attempts to demonstrate bubbles within cells, particularly those not containing fat inclusions, have been largely unsuccessful,[84,87] and even extravascular bubbles have not been observed in experimental animals after ascent to 45,000 feet,[86] despite the fact that rather large gas accumulations between muscle masses, under the skin, and near the joints have been demonstrated in human subjects.[84] Another disconcerting fact concerning the etiology of symptoms from decompression sickness is that bends pain can be reduced by the occlusion of arterial vessels leading to the region affected.[11] This is not compatible with the embolic theory or local ischemia unless the action involves the compression of vascular or other sensory nerves that are stimulated by the local effects of gas bubbles within the vessels. The fact that as little as 50 mm. Hg pressure over the tissues proximal to the site of bends pain will relieve the symptom indicates that the precise mechanism is not yet explainable by the mechanisms carried over from compressed-air studies.[11]

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