The time of onset of symptoms of decompression sickness is inversely related to the rate of ascent, as described previously,[69] and is further related to the product of altitude and duration of exposure at each altitude.[11] Thus, unless the precaution of thorough denitrogenation is taken before ascent, there is always the possibility of the formation of bubbles provided the altitude is great and the stay protracted. In view of these considerations it seems desirable to assume that bubble nuclei are always present or at least are being continuously formed in the various tissues of the body and that their chances of survival and growth are enhanced in proportion to the concentration of inert gases. The site of such activity may well be the loci at which the respiratory gases are handled normally by each tissue, thus accounting for the low incidence of patent bubbles in tissue cells, where gas exchange is diffuse, as compared with the high incidences in the blood, where the intensity of gas exchange is of a very high order.

Harvey's theory [84] that hydrostatic factors are basically responsible for the initiation of bubble nuclei is based on the observations that exercise and straining are accelerating factors in bubble formation and that the accompanying increase in carbon dioxide concentration is less effective. It is a well-documented fact that performance of muscular exercise is a predisposing factor for bends and is thus a variable in the determination of the time of onset of decompression sickness symptoms.[11,84,85,87]

The effect of temperature on the production of symptoms of decompression sickness has not been conclusively demonstrated at altitude,[11] but the fact that a predisposition to such symptoms in caisson workers subject to chilling can be shown[58] leads to the assumption of a conservative point of view. The maintenance of comfortable skin temperatures by electrical heating for the preservation of the highest degree of co-ordination in the performance of flight duties obviates, except in the case of accident, the need for consideration of the effects of cold on decompression sickness. The rationale of the effect of cold at altitude is based on the vasoconstricting action of cold in the skin, inducing an interference with the elimination of inert gases, and the production of shivering, which tends to exaggerate the effects of muscular tension over those normally found.

There are at least two other variables that have been found to correlate directly with the incidence of decompression sickness: age and linear density (i.e., pounds per unit height).[11] Similar relations have been determined for decompression from high-pressure atmospheres.[16,62] The manner in which age increases the incidence of symptoms is not clear except perhaps insofar as the circulatory resiliency of young individuals may minimize effects that would be reported as noticeable in older men. It would be more understandable if the differential were only in the severity of symptoms because the effects on the capillary vessels of older men would be expected to be more drastic. The basis of linear density is undoubtedly tied up with body fat and the accompanying difference in vascularization of the tissue. As shown by Gersh,[86] the fatty tissues are poorly ventilated because the effective ratio of capillary surface to tissue volume is low as compared with tissues such as muscle, which are known to be sites of extravascular bubbles.

6. Acclimatization

The repeated or continuous exposure of individuals to high altitudes has been studied mainly in connection with mountaineering and in connection with a few industries such as the Andean sulfur mining in Chile.[88] The effects noted were exclusively concerned with the reduction of the partial pressure of oxygen, since under the prevailing altitude levels decompression effects would not appear. The adjustments accomplished by the body under prolonged hypoxia of mild degree are mainly an increase in the number of red cells and their hemoglobin content, and in the tidal volume and rate of ventilation of the lungs, as well as changes in the circulatory efficiency: all directed to the more adequate transport of oxygen to the tissues.[11,84] Under high altitude conditions, that is, where great stress is placed on the organism even on an intermittent basis, the reactions invoked become less adaptive and in certain respects impose further stresses that may result in serious damage to the circulatory system, particularly the heart.[89] At intermediate levels some of these responses may attain, and recede from, a maximum even under continuing stimulation.[90] Ruff and Strughold laid great emphasis on these reactions and recommended the regular exposure of aviators to these conditions at mountain rest camps.[6] It is apparent, however, that the use of supplementary oxygen at the great heights attained by present aircraft largely does away with the desirability of such acclimatization. It would be of value at intermediate levels only for protracted sojourn, such as described above. These changes take place with the passage of time in normal individuals though not without other symptoms and temporary disabilities, cyclic breathing, nausea, syncope, etc., collectively known as mountain sickness.

C. CHARGING OF THE BODY WITH GAS IN DESCENT: RECOMPRESSION

1. Re-solution of Gases. Relief of Decompression Sickness

One of the most comforting aspects of exposure to decreased barometric pressures is the fact that most of the dangers involved in this condition can be readily eliminated by recompression to atmospheric pressure. The exceptions are cases where a shocklike condition has developed to the point of collapse or just short of it, and where the simple restitution of high partial pressure of oxygen will not correct the trauma to the peripheral vascular bed and the attendant hemoconcentration. Bends pain, chokes, and even serious central effects will respond rapidly to recompression if recompression is instituted early enough. The

[69] D. B. Dill, *Life, Heat, and Altitude*. Harvard Univ. Press, Cambridge, Mass., 1938.
[11] D. Altland and B. Highman, *Am. J. Physiol.*, **167**, 261 (1951).
[84] H. Marshall and H. Specht, *Am. J. Physiol.*, **163**, No. 3 (1950).