

for the development of local ischemia in the tissues, particularly in the skin. End⁵⁴ and Swindle⁵⁵ indicate that intravascular bubbles may be secondary to embolism caused by the agglutination of red cells following exposure to compressed air, but confirmation of this effect is lacking. In many instances, however, non-symptomatic accumulations of gas are found in large quantity, as for instance in the fascial layers beneath the skin and between the muscles. They produce a crepitation during manipulation. The question of whether intra- or extravascular bubbles are responsible for such occlusions is an academic point, but one from which future prophylactic measures will evolve.

Given the general mechanism of occlusion or embolism the whole gamut of symptoms presented by decompression sickness can be seen to have a common cause. In particular, the effects of central nervous system disorganization and breakdown are directly traceable to the anoxia of motor centers and the integrating mechanism of the cortex and medulla. Thus the reported "monoplegia, paraplegia, Ménière's complex, apoplectic deafness, vertigo, aphasia, hemiplegia, etc." are all manifestations of this basic mechanism, throughout the nervous system. In fact, the cause of death in the majority of cases is rather to be attributed to a cerebral embolic accident than to general anoxia from cardiac failure or a toxic effect of the exposure on the tissues.^{55,61}

The statistical distribution of symptoms of caisson disease indicates that peripheral effects predominate.⁵⁵ This may be related to the fact that peripheral reservoirs of fat unload their nitrogen slowly and therefore are subject to bubble formation, while only in case of gross supersaturation of the individual do chokes and central nervous system disturbances appear.

It is possible that the caliber of the vessels in the lungs and the brain play some part in this distribution, since it is generally estimated that the capillaries are larger here than in other tissues; thus only when large bubbles form either directly or by fusion from smaller bubbles will effects in these regions be expected.

3. Rate of Decompression

The problem of desaturating the tissues without causing bubble formation has been recognized since Bert's researches.⁴ The first attempts to circumvent this phenomenon were not very successful and were extremely time-consuming because it was thought that uniform, slow decompression would prevent bubbles from being initiated. This procedure, however, resulted in maintaining such a low differential pressure across the interface between tissue and environment that transfer of gas was slow, and bubbles formed despite all precautions. Haldane then instituted the technique of decompressing rapidly to such a pressure as to create a large differential pressure between tissues and environment but still small

enough to prevent bubble formation in the time used for desaturation. After desaturating at this level for an appropriate period the pressure was again reduced and a somewhat longer period of desaturation followed. By extension this procedure will bring the diver to the surface in considerably less time than with the former method and without as great a hazard. It was shown experimentally that the pressure could be safely reduced to one half at each stage, and the period of desaturation was calculated from composite data on the different rates of desaturation of various tissues of the body.

The method outlined above is essentially the one in use in all decompression procedures, but certain minor innovations have been applied by subsequent experimenters.⁶⁶ The Continental practice is a similar modification introduced by Bornstein,⁵⁸ in which the initial rapid reduction in pressure is made as in Haldane's method, but subsequently a continuous slow ascent is made. This method, according to its originator, maintains the gradient of pressure effected by one initial pressure drop throughout the decompression and further reduces the total decompression time.

The diving tables in use in this country are constructed on the stage basis of Haldane but include an ascent between stages of not more than 25 feet per minute.⁶⁶ Acceleration of the ascent schedule has been effected by the introduction of ventilative breathing of pure oxygen at the later stages when the total pressure is low enough to avoid effects of oxygen poisoning, but for economic reasons this method is used only in decompressions after recompression following bends or other serious decompression symptoms.⁶⁷ The use of helium in place of nitrogen has been advocated because of its lower solubility, but its principal contribution is the elimination of "nitrogen narcosis" in deep diving.^{40,64,65,68}

The practice in decompression for caisson workers is a continuous slow reduction to one half the working pressure. Attempts to decrease the incidence of bends by oxygen breathing have been made with some success.⁴⁸ Uniform decompression rates have been prescribed by law⁴⁷ in the past, but changes in the codes have been made to take advantage of more rapid methods, with due consideration for safety. In many instances there is an over-all limitation in the minimum period for decompression without strict interpretation of the initial rate of pressure reduction. Because of marked fog formation in this practice, and the lowering of the decompression chamber temperature during this procedure, serious consideration is given in Continental codes⁶⁸ to moderate recompressions following each stage of decompression, in order to warm the air slightly and thus avoid chilling the men who have been at hard work in a hot

⁵⁴ E. End, *J. Ind. Hyg. Toxicol.*, **20**, 511 (1938).

⁵⁵ P. F. Swindle, *Am. J. Physiol.*, **120**, 59 (1937).

⁵⁸ E. R. Boycott, G. C. C. Damant, and J. S. Haldane, *J. Hyg.*, **3**, 342 (1908).

⁶⁶ O. E. Van der Aue, W. A. White, Jr., R. Hayter, E. S. Brinton, R. J. Keller, and A. R. Behnke, *Experimental Diving Unit, Navy Yard (Washington, D. C.) and Naval Med. Research Center (Bethesda, Md.), Research Project X-443, Rept. No. I* (1945).

⁶⁷ A. R. Behnke, R. M. Thomson, E. P. Motley, *Am. J. Physiol.*, **112**, 554 (1935). E. End, *Am. J. Physiol.*, **120**, 712 (1937).