

gas during rapid decompression will cause discomfort only in proportion to the expansion ratio effected in the routine followed. Since in most cases this involves the Haldane or Bornstein stage method of decompressing to half the starting pressure,^{14,53} the expansion will be some two times (greater by the effect of water vapor¹⁰) the volume at equilibrium. In most cases this is a moderate stimulus to the smooth muscles of the gastrointestinal tract and results in the expulsion of the gas.

A more serious consequence of mechanical nature is the rupture of the lungs during even moderate decompressions because of the failure to keep the respiratory passages open.⁵⁴ This rupture occurs mainly in cases of submarine escape where a tendency to hold the breath is the natural result of apprehension and unfamiliarity with the unusual environment. The incidence in caisson workers and divers is undetermined, but the individual should be warned against breath holding during decompression.

2. Bubble Formation

The rate of removal of a gas from a fluid must keep pace with the tendency to desaturation resulting from a fall in total pressure if the formation of a free gas phase, i.e. bubbles, is to be prevented. The physiology of bubble formation may be best evaluated by a consideration of the mechanism based on the kinetic theory of gases. The molecules of a liquid may exist in the gaseous state and while in the gaseous state they have a higher average energy content than their fellows in the liquid state. Presumably, then, the average energy of gas molecules dissolved in a liquid composed of other molecules becomes equal to the average energy of the liquid molecules. A bubble of gas will not form unless sufficient energy is applied to enough gas molecules in a restricted locus to result in displacement of the liquid molecules. The chances for the spontaneous localization of such energy quantities are very slight and it is generally assumed that minute gas or vapor spaces large enough to permit initiation of bubbles are always present at liquid-solid interfaces under natural circumstances.⁵⁵⁻⁵⁷ These spaces have been termed "gas nuclei." (Harvey⁵⁸ has been instrumental in popularizing this term but it is probably less confusing to call the spaces *bubble nuclei*.)

The data gathered from many experiments on degassing by Kuper,⁵⁶ Piccard,⁵⁶ Harvey *et al.*,⁵⁹ and Dean⁵⁷ indicate that a free-gas phase forms readily

⁵⁴ I. B. Polak and B. H. Adams, *U. S. Naval Med. Bull.*, **30**, 165 (1932).

⁵⁵ J. B. H. Kuper, *Rept., Aviation Medicine Unit, Ind. Hyg. Research Lab., Nat. Inst. Health*, Bethesda, Md. (1942).

⁵⁶ J. Piccard, *Proc. Staff Meetings Mayo Clinic*, **16**, 700 (1941).

⁵⁷ R. B. Dean, *J. Applied Physics*, **15**, 446 (1944).

⁵⁸ E. N. Harvey, A. H. Whitely, W. D. McElroy, D. C. Pease, and D. K. Barnes, *J. Cellular Comp. Physiol.*, **24**, 23 (1944).

⁵⁹ E. N. Harvey, D. K. Barnes, W. D. McElroy, A. H. Whitely, D. C. Pease, and G. W. Cooper, *J. Cellular Comp. Physiol.*, **24**, 1 (1944).

only if a space is present into which the gas may diffuse. Relatively enormous supersaturations are possible in the absence of such bubble nuclei. It is probable that under normal life conditions nuclei exist in profusion, specifically at such points as on substances active in the transfer of gases, such as hemoglobin and other respiratory pigments. The question of the ultimate locus of bubble formation is one that is being actively investigated at this time, and until definitive evidence is in hand no further analysis can profitably be made.

From the work of Gersh⁶⁰ it is indicated that a gross correlation of the distribution of bubbles with that of fat tissues is found in decompressions from positive pressure. It seems that these loci by virtue of their relatively high gas content have at least the requisite condition for maintaining patent bubbles whether or not they offer the highest incidence of nuclei among the various tissues of the body. In point of time it is evident, however, from these data that bubbles in the vessels appear in greater number than bubbles in fat, at least in the early stages of this condition.

The evolution of gases from solution in the various tissues of the body on decompression gives rise to the main body of sequelae and forms the basis of the occupational disease of workers in compressed-air atmospheres. The essential factors that on decompression the body fluids are supersaturated to a varying degree depending on the pressure attained and the time of exposure. If the supersaturation is great enough in the face of a continuous loss of the gas through exhalation and respiration, then discrete gas phases will form, both intra- and extravascularly. These accumulations of gas then act to disturb the normal functions of the tissues in which they appear in proportion to their number and size. Thus, in the vascular system bubbles are found to form largely in the venous vessels and they are carried to the right heart and lungs where they accumulate and are responsible for the symptoms known as the "chokes."⁶¹ This is a disability of varying degree, depending upon the extent to which the circulation of the lungs is interfered with. In general, it is considered a serious condition and recompression is instituted as quickly as possible. The appearance of bubbles in the blood is usually accompanied by appearance of bubbles in the various tissues, the fatty tissues being most subject to bubble formation.^{62,63} While the cause of the symptoms of "bends" and other peripheral symptoms, such as pruritus, tingling sensations, and joint and tendon pain, is still a moot point, there is little doubt that the presence of the bubbles constitutes one of the most reasonable mechanisms

⁶⁰ G. E. Gersh, G. E. Hawkinson, and E. H. Jenney, *Naval Med. Research Inst., Research Project X-284, Rept. No. V* (1944). Also in *J. Cellular Comp. Physiol.*, **26**, 63 (1945).

⁶¹ G. E. Gersh, *Naval Med. Research Inst., Research Project X-284, Rept. No. X* (1945). Also in *J. Cellular Comp. Physiol.*, **26**, 101 (1945).

⁶² G. E. Gersh, G. E. Hawkinson, E. N. Rathbun, and A. R. Behnke, *Naval Med. Research Inst., Research Project X-284, Rept. No. II* (1944). Also in *J. Cellular Comp. Physiol.*, **24**, 35 (1944).

⁶³ G. E. Gersh, *Naval Med. Research Inst., Research Project X-284, Rept. No. IX* (1945).
⁶⁴ E. Warner, *ibid.*, *Rept. No. IV* (1944).