deleterious and painful when, as in the ear, the openings are not patent. In normal individuals this is rare, but it is a chronic condition with some individuals, generally associated with infection or malformations. Obviously, acute infection of the upper respiratory tract tends to produce this condition. When the openings of the sinuses are thus occluded an increase in atmospheric pressure will result in severe pain and varying degrees of trauma even with moderate pressure differences. This seeming disproportionality between cause and effect is due possibly to the rather large surface-to-volume ratio of most sinuses, allowing ready distension of large areas of capillaries. This distension leads to marked stimulation of deep pain receptors, with the observed sequelae of nausea, etc., due to autonomic discharge from this stimulation. As far as can be determined, reports from the older literature of mechanical trauma to the vascular system are to be attributed more to the various conditions enumerated above than to other gross hydraulic effects, as was formerly held.[41] It is true, however, that the blood is forced into the capillaries lining any such rigid-walled, isolated gas space and, depending on the tonic state, may result in hemorrhage. If we assume a normal capillary pressure of 40 mm. Hg, it takes only 1/19 atm. of positive pressure to double this value in the capillary bed of the ear or sinus. Clinical tests of capillary fragility are based on petechial hemorrhages in normal tissue with 50 mm. Hg pressure above normal![24]

Gases in the alimentary tract are, wherever located, subject to compression by the body tissues and for the most part do not cause any inconvenience beyond the displacement of the viscera, which in most cases can be of only a benign nature. As will be shown subsequently this does not absolve these gas entities from consideration since, unless passed as flatus, they expand on decompression.

There remains one more mechanical effect of compression which, while rare, is real and may produce drastic sequelae: this concerns the compression of the gases in the lungs. Offhand, no effect would be expected since almost continuous patency of the breathing passages is assumed. There must be recognized, however, the fact that, during rapid compression, closure of the glottis in swallowing or breath holding may result in compression of the lungs to such an extent that only with considerable difficulty can sufficient positive pressure be created by the thoracic musculature to release the glottal closure. Such cases have been observed on recompression from negative pressure, as will be discussed later. It is important, especially in experimental work, that warning against breath holding be promulgated during indoctrination and in the course of work while rapid compression is being effected.

Other effects of compression that are partly of a mechanical nature, such as the transient accumulation of carbon dioxide in the lungs and blood during inflow of gas on rapid compression of the atmosphere, as described by Bean,[20] are discussed under subsequent headings.

The extension of mechanical effects to conditions of pathology is not possible in this presentation, but reports of injury to pathological areas in the lungs on compression are found in the literature[20] and argue strongly for thorough pre-employment medical examinations in occupations involving atmospheric pressure changes.

[41] E. C. Hoff, *A Bibliographical Source-Book of Submarine and Compressed Air Medicine*. U. S. Government Printing Office, Washington, D. C., 1947.

2. Solution of Gases in the Body

As indicated in the section on the solubility of atmospheric gases, the capacity of the blood and tissues for taking up gases is determined by two general characteristics that respond to partial pressure. That is, both the simple physical solution of gases in fluids and the pressure-labile chemical association of certain of them with solutes in the fluids and their cells are concomitantly affected by changes in pressure. Relatively small quantities of gas are taken up by the watery portion of the blood as is seen from Table 2, but it must be kept in mind that all gases passing to and from the tissues pass through the solution phase in the course of their transfer between tissue and atmosphere. In addition, at every stage of the transfer of gases there is, inescapably, a series of mediums through which gas exchange takes place, each of which has its own coefficient of diffusion, permeability, partition, and the like, which set up the parameters of the tissue–gas relationship with external pressure.

Smith and Morales[42] describe at least four phases in the attainment of saturation of tissues with inert gases by way of the lungs. Their analysis gives a curve of uptake with respect to time that, instead of having a uniformly changing slope, is characterized by an inflection or flattening in what they call the second stage of gas transfer. At this time the blood is taking in gas at a uniform rate, since it is presented at the lung surface at a limited rate, in conformity with the speed of circulation. These transient phenomena are of interest here mainly in that they show the system to be first serially, then concomitantly, affected by gas pressure changes during an increase in pressure. It is evident, further, that by virtue of these limiting phases the degree of saturation or concentration of gases in the tissues is limited with respect to time.

Although the general nature of the uptake of gases in the different tissues of the body is similar, the effectiveness of the distributing system varies and this further affects the time required for saturation. The partition of gases between fatty, glandular, neural, and muscular tissues is obviously dependent at equilibrium on the solubility of the gases in the various materials comprising the cell structures, the protoplasm, and the inclusions, but at any time short of equilibrium the major factors may be considered to be the transport and access of the gases to the cells. These factors are usually spoken of as the vascularization of the tissue. Gersh[43] has shown that the ratio of capillary surface to the total cell volume of fatty tissues is 51.9 and of tissues poor in fat is 222.2. He indicates further that this disparity is due to the included fat alone, since the same ratio on the basis of volume of active protoplasm exceeds that found for muscle.

Essentially it is seen that the fat depots are large nitrogen reservoirs with restricted access and thus require a longer time for charging than do the lean tissues.

[42] M. F. Morales and R. E. Smith, *Bull. Math. Biophysics*, *6*, 141 (1944).

[43] I. Gersh and M. A. Still, *Naval Med. Research Inst., Research Project X-284, Rept.*, No. III (1944).