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,[30] 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[30] and argue strongly for thorough preemployment medical examinations in occupations involving atmospheric pressure changes.

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[43] 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 among 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 other than those controlling diffusion (i.e., invasion and evasion of gases)[15] 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[44] 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.

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

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.

3. Rate of Change of Pressure

The foregoing facts indicate that complete saturation requires a definite lapse of time, and thus on theoretical grounds alone short deep dives may be effected with less chance of sequelae due to excess inert gas than may long shallow dives. From experience and experimentation the relations of these pressure and time factors have been summarized in diving tables.[45,46]

The immediate effects limiting the rate of compression are mainly those of a mechanical nature on the ears, sinuses, and other isolated gas phases within the body. Gastrointestinal gases apparently play no role in compression but should be taken into account in view of the ultimate decompression. The effects on the ear and sinuses are not expressible in quantitative form because of the highly individual nature of the response.

The only indication that rapid compression may have some other limiting factor is in the observation of Bean[30] that the influx of gas into the lungs may cause a transient retardation of the CO_2 elimination and thus temporarily elevate the pCO_2 both in the alveoli and blood-tissue complex. Bean found complete arrest of alveolar gas outflow in human subjects during such rapid compression, and reports elevation of pCO_2 in catheterized lungs of dogs under anesthesia.

B. EFFECTS OF MAINTAINED POSITIVE ATMOSPHERIC PRESSURE

1. State of Saturation of the Several Body Tissues

The diving tables[46] indicate that saturation at any level is rarely attained in the course of the recommended bottom period since the time schedule for

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

[45] R. L. Fisher and W. A. White, Jr., *Naval Med. Research Inst., Rept.* No. **86** (1944).

[46] *Diving Manual, 1943.* Navy Dept., Bureau of Ships, U. S. Government Printing Office, Washington, D. C.