and humid atmosphere. This practice is based on the fact that peripheral circulation is practically occluded by vasomotor response to lowering of the temperature, and thus desaturation of the skin and adjacent tissues is prevented with the unpleasant results of bends, itching, and similar sequelae.

As suggested above, the desaturation procedures described here do not proceed without occasional symptoms of decompression sickness, and this debility may appear for some hours after decompression because of bubbles from improperly desaturated nitrogen reservoirs. The subsequent lodgment of such bubbles in a critical point of the circulation will bring about the same symptoms as mentioned above and in the same range of severity. Thus it is necessary to keep decompressed individuals within reach of recompression equipment and to apply pressure and ventilative breathing of oxygen until remission of symptoms is complete.[67] The re-solution of bubbles is slow at recompression conditions because of the decreased bubble surface presented during recompression.[55] Unfortunately, these procedures often have been carried out on the worker's time, thus introducing a deterrent factor, and therefore many cases have been prolonged unnecessarily. In large part, the blame for poor decompression technique falls on the workers themselves, since they are often reckless in their haste to decompress and get off the job; and many have the ill-conceived notion that they have tough constitutions that will enable them to absorb such abrupt changes without permanent effects.[48]

Repeated exposure to bends symptoms, and perhaps even the mere repetition of decompression without discernible symptoms, will in time produce effects in those regions where bubble formation usually takes place.[53] In general, these chronic lesions are situated in the joints and in other tissues with poor circulation. They are thought to be the result of bubble trauma and repeated acute, or chronic, low-grade anoxia of the local tissues. They include: simulated arthritic effects of hip and knee joints; ankylosis of the incus, malleus, and stapes of the middle ear; thickening of the drum; and so forth.[53]

IV. Effects of Reduced Atmospheric Pressure on the Body

Ascent into the atmosphere simulates physically the conditions attending decompression from increased atmospheric pressures. It will be seen, however, that the ratio of pressure change permissible for the diver cannot be attained by the aviator. Thus, divers can work at 10 atm., a ninefold difference from their natural environment, yet at $1/2$ atm. there is serious disturbance for the aviator and few, even hardened and acclimated, men have ever attained $1/3$ atm. for more than passing intervals.[6] The difference in range is mainly due to the fact that the decrease in barometric pressure on ascent to altitude strikes at the only component of the body's energy-liberating mechanism that it is unable to store to any reasonable extent: oxygen. The following sections will deal with this and other limiting factors in a manner comparable to that of the preceding section on positive pressures.

A. DISCHARGE OF GASES FROM THE BODY: DECOMPRESSION

As in decompression from positive pressures, the reduction in environmental pressure causes a differential pressure to exist between the gases dissolved in the body and the ambient atmosphere. In this case, however, the body is usually in complete equilibrium with the inert gases and thus will be supersaturated as soon as any ascent is begun. Obviously, the body will lose part of all the gases dissolved in it, even oxygen if the rate of ascent is abrupt enough, and this efflux of gases is regulated by the access that the various parts of the body have to the exterior. Thus, the fatty tissues and those with poor vascularization will maintain a high differential pressure as compared with others more favorably endowed.

1. Rate of Ascent

At the present writing, the physical possibilities of rates of ascent can hardly be overestimated. Given a suitable initial change of acceleration, the practical limitations for the newer type of aircraft are actually beyond the capacity of the body for coping with the changes in the environment; for example, the little defensive rocket-propelled "viper" developed by the Germans for use against Allied bombers made a practically vertical ascent to over 30,000 feet in somewhat less than two minutes, and it is probable that this is not the maximum that can be attained for piloted aircraft. In any case, this rate of ascent induces a pressure differential of nitrogen of about 368 mm. Hg in a matter of minutes, discounting for the moment the loss of nitrogen in this time from some of the tissues. Thus, as will be seen below, ascents can be made in such time intervals that the normal routes of gas transfer cannot handle the amount of gas that tends to be freed from solution.

Catchpole and Gersh[69] have shown that for rabbits the critical rate of ascent lies somewhere near 7000 to 8000 f.p.m., even though the animals are breathing oxygen during ascent. At 4500 f.p.m., survival at 45,000 feet for 30 minutes indicated that serious effects were not incurred at this rate. In human decompressions to altitude more conservative estimates must be made, first, because of the difference in size and vascularization as compared with those of small mammals and, second, because of factors other than bubble formation. In decompression for experimental purposes where bends are to be avoided it has been the practice to ascend at about 2000 and 3000 f.p.m. In indoctrination ascents for fliers, the rate of ascent has been set as high as 5000 f.p.m. In the latter case, bends do occur in some instances and this routine has been used to assist in selection of air crews. The experience of the Army Air Corps[70] shows that ascent at maximum feasible rates of climb, about 5000 f.p.m., at the time of this writing produces a bends syndrome only above 18,000 feet, and the severity is dependent on contributing factors of exercise, cold, length of stay, and so forth.

[69] H. R. Catchpole and I. Gersh, *Naval Med. Research Inst., Research Project X-284, Rept. No.* VI (1945). Also in *J. Cellular Comp. Physiol.*, **27**, 15 (1946).

[70] War Dept., *Tech. Manual* 1-705 (1941).