decompression increases throughout with increased stay at the working level. The phenomenally long decompressions necessary for even short stays at great depths prove to be a limiting factor in the amount of useful work that can be effected, even under the best possible conditions; for example, a 20-minute dive of 300 feet on compressed air requires a total time of 3 hours.

The limitations on the stay at working level are, however, legion. Although electric heating of suits to maintain the body temperature and improvement in construction of the suits to increase mobility and reduce the fatigue of maneuvering the diving rig have been effective in improving the performance of divers, the length of stay at the feasible work depths is still short. The resistance to movement and the difficulty of working against water movements is so telling on the strength of divers that effective work cannot be carried out for the recommended periods except under very favorable conditions of weather and currents.[47]

In caisson work the periods of work are longer in general, and as a rule the pressures encountered are limited by the nature of the operations. Pressure depths equivalent to about 100 feet probably are not often encountered and a work period of 1.5 hours twice a day has been used at such levels.[47] Since the conditions for performing useful work are in most cases better for caisson workers than for divers, some difference is understandable. On the other hand, the caisson worker is undoubtedly more nearly saturated than is the diver under these practices and thus more subject to sequelae if proper decompressions are not carried out. It has been the practice in this country for caisson crews to work in divided shifts.[42] The incidence of bends reported by Jones *et al.*[48] is 1.27 per thousand decompressions after a 1.5-hour shift but, because of the reluctance of workers to report mild cases or to have treatment on their own time, there is some question whether complete coverage was obtained. It was found, in tests of prophylaxis, where attention was directed to reporting all cases and where no stigma was attached to such reporting, the incidence was 2.05 per thousand despite the complete lack of any severe cases. Other groups showed incidences of 2.49 to 2.24 per thousand when no treatment or supervision by interested personnel was maintained. The latter higher incidence is undoubtedly related to the extremely casual manner in which decompression is carried out at the insistence of the men themselves.

2. *Effects on the Circulation*

In an academic sense, the blood can carry in physical solution enough oxygen to supply tissue respiration when the inspired partial pressure of oxygen is equivalent to 3 atm.,[16] whereas while breathing 1 atm. of air, less than 1 per cent of the oxygen carried by the blood is in physical solution. The full oxygen capacity of the hemoglobin is attained, however, with only slight increase in

[47] *Occupation and Health*. International Labour Office, Geneva, 1930.

[48] R. R. Jones, J. W. Crosson, F. E. Griffith, R. R. Sayers, H. H. Schrenk, and E. Leva, *J. Ind. Hyg. Toxicol.*, **22**, 427 (1940).

alveolar pO_2. Therefore, it would seem that oxygen transport per se is more than adequate in compressed air atmospheres.

The situation with regard to CO_2 is by the same token not so favorable. This follows from the fact that the carbon dioxide from the tissues is less readily taken up by the hemoglobin under these conditions and a rise in venous pCO_2 occurs accompanied by a fall in pH with the attainment of the buffer capacity of the blood. Behnke *et al.*[49] showed that the change in pH at 4 atm. of oxygen amounted to 0.03 in the venous blood. As indicated above a further temporary enhancement of this effect may follow rapid compression.[30]

In general the pulse pressure and circulation are not uniformly changed by increase in atmospheric pressure, but when very high partial pressures of oxygen are obtained, complicating effects from the impaired carbon dioxide transport cause some changes. It is probable that dilation of cerebral and peripheral vessels is effected.[30]

3. *Oxygen Poisoning*

At from 3 to 4 atm. of oxygen, or corresponding oxygen pressure in air, certain motor disturbances of cerebrocortical origin develop. The syndrome is called "oxygen poisoning" and may show only minor twitches but usually progresses to full-blown convulsions within a short time. This is an acute condition related qualitatively to symptoms known to affect individuals maintained at a partial pressure of oxygen above about 600 mm. Hg for periods of several days.[50] The primary effects at normal atmospheric pressure seem to be a pulmonary involvement resulting in substernal distress and coughing with a clinical picture of lung congestion, while the acute conditions brought on by exposure to high partial pressures of oxygen over short intervals at increased atmospheric pressures affect the central nervous system, causing seizures, loss of consciousness, narrowing of the visual fields through scotomata, and so forth, which overshadow the appearance of less spectacular symptoms. The modus operandi of oxygen poisoning is in considerable dispute at the present time since both CO_2 and O_2 are known to have destructive effects on isolated systems at increased pressure.[30] Studies tend to show that irreversible or incompletely reversible changes take place in all intracellular reactions and particularly in the central nervous system, and there is good reason to believe that the normal gas-handling systems involving enzymes are intimately affected.[30] It has been shown recently that oxygen toxicity is not initiated by interference with the transport of carbon dioxide from tissues to lungs.[51]

4. *Inert Gas Effects*

From the experimental work on deep-sea diving and salvage it was reported

[49] A. R. Behnke, L. A. Shaw, C. W. Shilling, R. M. Thompson, and A. C. Messer, *Am. J. Physiol.*, **107**, 13 (1934).

[50] H. Becker-Freyseng and H. G. Clamann, *Luftfahrtmedizin*, **7**, 272 (1942).

[51] C. Lambertsen *et al.*, *J. Appl. Physiol.*, **6**, No. 6, 358 (1953).