Helium is widely used in mixed-gas diving as a substitute for nitrogen to prevent narcosis. Helium has not demonstrated narcotic effects at any depth tested by the U.S. Navy. Diving with helium-oxygen mixtures is the only way to prevent nitrogen narcosis. Helium-oxygen mixtures should be considered for any dive in excess of 150 fsw.

3-9.2 Oxygen Toxicity. Exposure to a partial pressure of oxygen above that encountered in normal daily living may be toxic to the body. The extent of the toxicity is dependent upon both the oxygen partial pressure and the exposure time. The higher the partial pressure and the longer the exposure, the more severe the toxicity. The two types of oxygen toxicity experienced by divers are pulmonary oxygen toxicity and central nervous system (CNS) oxygen toxicity.

3-9.2.1 **Pulmonary Oxygen Toxicity.** Pulmonary oxygen toxicity, sometimes called low pressure oxygen poisoning, can occur whenever the oxygen partial pressure exceeds 0.5 ata. A 12 hour exposure to a partial pressure of 1 ata will produce mild symptoms and measurable decreases in lung function. The same effect will occur with a 4 hour exposure at a partial pressure of 2 ata.

Long exposures to higher levels of oxygen, such as administered during Recompression Treatment Tables 4, 7, and 8, may produce pulmonary oxygen toxicity. The symptoms of pulmonary oxygen toxicity may begin with a burning sensation on inspiration and progress to pain on inspiration. During recompression treatments, pulmonary oxygen toxicity may have to be tolerated in patients with severe neurological symptoms to effect adequate treatment. In conscious patients, the pain and coughing experienced with inspiration eventually limit further exposure to oxygen. Unconscious patients who receive oxygen treatments do not feel pain and it is possible to subject them to exposures resulting in permanent lung damage or pneumonia. For this reason, care must be taken when administering 100 percent oxygen to unconscious patients even at surface pressure.

Return to normal pulmonary function gradually occurs after the exposure is terminated. There is no specific treatment for pulmonary oxygen toxicity.

The only way to avoid pulmonary oxygen toxicity completely is to avoid the long exposures to moderately elevated oxygen partial pressures that produce it. However, there is a way of extending tolerance. If the oxygen exposure is periodically interrupted by a short period of time at low oxygen partial pressure, the total exposure time needed to produce a given level of toxicity can be increased significantly.

3-9.2.2 **Central Nervous System (CNS) Oxygen Toxicity.** Central nervous system (CNS) oxygen toxicity, sometimes called high pressure oxygen poisoning, can occur whenever the oxygen partial pressure exceeds 1.3 ata in a wet diver or 2.4 ata in a dry diver. The reason for the marked increase in susceptibility in a wet diver is not completely understood. At partial pressures above the respective 1.3 ata wet and 2.4 ata dry thresholds, the risk of CNS toxicity is dependent on the oxygen partial pressure and the exposure time. The higher the partial pressure and the longer the

exposure time, the more likely CNS symptoms will occur. This gives rise to partial pressure of oxygen-exposure time limits for various types of diving.

3-9.2.2.1 ***Factors Affecting the Risk of CNS Oxygen Toxicity.*** A number of factors are known to influence the risk of CNS oxygen toxicity:

Individual Susceptibility. Susceptibility to CNS oxygen toxicity varies markedly from person to person. Individual susceptibility also varies markedly from time to time and for this reason divers may experience CNS oxygen toxicity at exposure times and pressures previously tolerated. Individual variability makes it difficult to set oxygen exposure limits that are both safe and practical.

CO_2 Retention. Hypercapnia greatly increases the risk of CNS toxicity probably through its effect on increasing brain blood flow and consequently brain oxygen levels. Hypercapnia may result from an accumulation of CO_2 in the inspired gas or from inadequate ventilation of the lungs. The latter is usually due to increased breathing resistance or a suppression of respiratory drive by high inspired ppO_2. Hypercapnia is most likely to occur on deep dives and in divers using closed and semi-closed circuit rebreathers.

Exercise. Exercise greatly increases the risk of CNS toxicity, probably by increasing the degree of CO_2 retention. Exposure limits must be much more conservative for exercising divers than for resting divers.

Immersion in Water. Immersion in water greatly increases the risk of CNS toxicity. The precise mechanism for the big increase in risk over comparable dry chamber exposures is unknown, but may involve a greater tendency for diver CO_2 retention during immersion. Exposure limits must be much more conservative for immersed divers than for dry divers.

Depth. Increasing depth is associated with an increased risk of CNS toxicity even though ppO_2 may remain unchanged. This is the situation with UBAs that control the oxygen partial pressure at a constant value, like the MK 16. The precise mechanism for this effect is unknown, but is probably more than just the increase in gas density and concomitant CO_2 retention. There is some evidence that the inert gas component of the gas mixture accelerates the formation of damaging oxygen free radicals. Exposure limits for mixed gas diving must be more conservative than for pure oxygen diving.

Intermittent Exposure. Periodic interruption of high ppO_2 exposure with a 5-15 min exposure to low ppO_2 will reduce the risk of CNS toxicity and extend the total allowable exposure time to high ppO_2. This technique is most often employed in hyperbaric treatments and surface decompression.

Because of these modifying influences, allowable oxygen exposure times vary from situation to situation and from diving system to diving system. In general, closed and semi-closed circuit rebreathing systems require the lowest partial pressure limits, whereas surface-supplied open-circuit systems permit slightly higher

limits. Allowable oxygen exposure limits for each system are discussed in later chapters.

3-9.2.2.2 ***Symptoms of CNS Oxygen Toxicity.*** The most serious direct consequence of oxygen toxicity is convulsions. Sometimes recognition of early symptoms may provide sufficient warning to permit reduction in oxygen partial pressure and prevent the onset of more serious symptoms. The warning symptoms most often encountered also may be remembered by the mnemonic VENTIDC:

V: Visual symptoms. Tunnel vision, a decrease in diver's peripheral vision, and other symptoms, such as blurred vision, may occur.

E: Ear symptoms. Tinnitus, any sound perceived by the ears but not resulting from an external stimulus, may resemble bells ringing, roaring, or a machinery-like pulsing sound.

N: Nausea or spasmodic vomiting. These symptoms may be intermittent.

T: Twitching and tingling symptoms. Any of the small facial muscles, lips, or muscles of the extremities may be affected. These are the most frequent and clearest symptoms.

I: Irritability. Any change in the diver's mental status including confusion, agitation, and anxiety.

D: Dizziness. Symptoms include clumsiness, incoordination, and unusual fatigue.

C: Convulsions. The first sign of CNS oxygen toxicity may be convulsions that occur with little or no warning.

Warning symptoms may not always appear and most are not exclusively symptoms of oxygen toxicity. Muscle twitching is perhaps the clearest warning, but it may occur late, if at all. If any of these warning symptoms occur, the diver should take immediate action to lower the oxygen partial pressure.

A convulsion, the most serious direct consequence of CNS oxygen toxicity, may occur suddenly without being preceded by any other symptom. During a convulsion, the individual loses consciousness and his brain sends out uncontrolled nerve impulses to his muscles. At the height of the seizure, all of the muscles are stimulated at once and lock the body into a state of rigidity. This is referred to as the *tonic phase* of the convulsion. The brain soon fatigues and the number of impulses slows. This is the *clonic phase* and the random impulses to various muscles may cause violent thrashing and jerking for a minute or so.

After the convulsive phase, brain activity is depressed and a *postconvulsive (postictal) depression* follows. During this phase, the patient is usually unconscious and quiet for a while, then semiconscious and very restless. He will then usually sleep on and off, waking up occasionally though still not fully rational. The