

Diving Disorders Requiring Recompression Therapy

20-1 INTRODUCTION

20-1.1 Purpose. This chapter describes the diagnosis of diving disorders that either require recompression therapy or that may complicate recompression therapy. While you should adhere to the procedures as closely as possible, any mistakes or discrepancies shall be brought to the attention of NAVSEA immediately. There are instances where clear direction cannot be given; in these cases, contact the Diving Medical Officers at NEDU or NDSTC for clarification. Telephone numbers are listed in Volume 1, Appendix C.

20-1.2 Scope. This chapter is a reference for individuals trained in diving procedures. It is also directed to users with a wide range in medical expertise, from the fleet diver to the Diving Medical Officer. Certain treatment procedures require consultation with a Diving Medical Officer for safe and effective use. In preparing for any diving operation, it is mandatory that the dive team have a medical evacuation plan and know the location of the nearest or most accessible Diving Medical Officer and recompression chamber. The Diving Medical Personnel should be involved in pre-dive planning and in training to deal with medical emergencies. Even if operators feel they know how to handle medical emergencies, a Diving Medical officer should always be consulted whenever possible.

20-2 ARTERIAL GAS EMBOLISM

Arterial gas embolism, sometimes simply called gas embolism, is caused by entry of gas bubbles into the arterial circulation which then act as blood vessel obstructions called *emboli*. These emboli are frequently the result of pulmonary barotrauma caused by the expansion of gas taken into the lungs while breathing under pressure and held in the lungs during ascent. The gas might have been retained in the lungs by choice (voluntary breathholding) or by accident (blocked air passages). The gas could have become trapped in an obstructed portion of the lung that has been damaged from some previous disease or accident; or the diver, reacting with panic to a difficult situation, may breathhold without realizing it. If there is enough gas and if it expands sufficiently, the pressure will force gas through the alveolar walls into surrounding tissues and into the bloodstream. If the gas enters the arterial circulation, it will be dispersed to all organs of the body. The organs that are especially susceptible to arterial gas embolism and that are responsible for the life-threatening symptoms are the central nervous system (CNS) and heart. In all cases of arterial gas embolism, associated pneumothorax is possible and should not be overlooked.

20-2.1 Arterial Embolism Development. Arterial gas embolism may develop within minutes of surfacing, causing severe symptoms that must be diagnosed and treated

quickly and correctly. Because the supply of blood to the central nervous system is almost always involved, unless treated promptly and properly by recompression, arterial gas embolism is likely to result in death or permanent brain damage.

20-2.2 Unconsciousness Caused by Arterial Gas Embolism. Gas embolism can strike during any dive where underwater breathing equipment is used, even a brief, shallow dive, or one made in a swimming pool. As a basic rule, any diver who has obtained a breath of compressed gas from any source at depth, whether from diving apparatus or from a diving bell, and who surfaces unconscious or loses consciousness within 10 minutes of reaching the surface, must be assumed to be suffering from arterial gas embolism. Recompression treatment shall be started immediately. A diver who surfaces unconscious and recovers when exposed to fresh air shall receive a neurological evaluation to rule out arterial gas embolism.

20-2.3 Neurological Symptoms of Arterial Gas Embolism. Divers surfacing with any obvious neurological symptoms (numbness, weakness, or difficulty in thinking) should be considered as suffering from an arterial gas embolism. Commence recompression treatment as soon as possible.

20-2.4 Additional Symptoms of Arterial Gas Embolism. Other factors to consider in diagnosing arterial gas embolism are:

- The onset is usually sudden and dramatic, often occurring within seconds after arrival on the surface or even before reaching the surface. The signs and symptoms may include dizziness, paralysis or weakness in the extremities, large areas of abnormal sensation, blurred vision, or convulsions. During ascent, the diver may have noticed a sensation similar to that of a blow to the chest. The victim may become unconscious without warning and may even stop breathing.
- If pain is the only symptom, arterial gas embolism is unlikely and decompression sickness or one of the other pulmonary overinflation syndromes should be considered.
- Some symptoms may be masked by environmental factors or by other less significant symptoms. A chilled diver may not be concerned with numbness in an arm, which may actually be the sign of CNS involvement. Pain from any source may divert attention from other symptoms. The natural anxiety that accompanies an emergency situation, such as the failure of the diver's air supply, might mask a state of confusion caused by an arterial gas embolism to the brain. A diver who is coughing up blood (which could be confused with bloody froth) may be showing signs of ruptured lung tissue, or may have bitten the tongue or experienced a sinus or middle ear squeeze.

20-2.5 Neurological Examination Guidelines. [Appendix 5A](#) contains a set of guidelines for performing a neurological examination and an examination checklist to assist nonmedical personnel in evaluating decompression sickness cases.

20-2.6 Administering Advanced Cardiac Life Support (ACLS) in the Embolized Diver. A diver suffering from an arterial gas embolism with absence of a pulse or respirations (cardiopulmonary arrest) requires Advanced Cardiac Life Support. Performing ACLS requires that special medical training and equipment be readily available. ACLS procedures include diagnosis of abnormal heart rhythms and correction with drugs or electrical countershock (cardioversion or defibrillation). Though patient monitoring and drug administration may be able to be performed at depth, electrical countershock must be performed on the surface.

If an ACLS-trained medical provider or a Basic Life Support–Defibrillation (BLS-D) provider with the necessary equipment can administer the potentially life-saving therapies within 10 minutes, the stricken diver should be kept at the surface until pulse and/or respirations are obtained. It must be realized that unless ACLS procedures—especially defibrillation—can be administered within 10 minutes, the diver will likely die, even though adequate CPR has been begun. If a Diving Medical Officer cannot be reached or is unavailable, the Diving Supervisor may elect to compress to 60 feet, continue Basic Life Support, and attempt to contact a Diving Medical Officer.

If ACLS becomes available within 20 minutes, the pulseless diver shall be brought to the surface at 30 fpm and defibrillated on the surface. (Current data shows there is 0-percent recovery rate after 20 minutes of cardiac arrest with BLS.) If the pulseless diver does not regain vital signs with ACLS procedures, continue CPR until trained medical personnel terminate resuscitation efforts. Never recompress a pulseless diver who has failed to regain vital signs after defibrillation or ACLS. Resuscitation efforts shall continue until the diver recovers, the tenders are unable to continue CPR, or trained medical personnel terminate the effort. If the pulseless diver does regain vital signs, compress to 60 fsw and follow the appropriate treatment table.

CAUTION **If the tender is outside of no-decompression limits, he should not be brought directly to the surface. Either take the decompression stops appropriate to the tender or lock in a new tender and decompress the patient leaving the original tender to complete decompression.**

20-2.7 Prevention of Arterial Gas Embolism. The potential hazard of arterial gas embolism may be prevented or substantially reduced by careful attention to the following:

- Proper, intensive training in diving physics and physiology for every diver, as well as instruction in the correct use of various diving equipment. Particular attention must be given to the training of scuba divers, because scuba operations produce a comparatively high incidence of embolism accidents.
- A diver must never interrupt breathing during ascent from a dive in which compressed gas has been breathed.
- A diver making an emergency ascent must exhale continuously. The rate of exhalation must match the rate of ascent. For a free ascent, where the diver

uses natural buoyancy to be carried toward the surface, the rate of exhalation must be great enough to prevent embolism, but not so great that the buoyancy factors are canceled. With a buoyant ascent, where the diver is assisted by a life preserver or buoyancy compensator, the rate of ascent may far exceed that of a free ascent. The exhalation must begin before the ascent and must be a strong, steady, forceful exhalation. It is difficult for an untrained diver to execute an emergency ascent properly. It is also often dangerous to train a diver in the proper technique. No ascent training may be conducted unless fully qualified instructors are present, a recompression chamber and Diving Medical Technician are on scene, and a Diving Medical Officer is able to provide an immediate response to an accident. Ascent training is distinctly different from ascent operations as performed by Navy Special Warfare groups. Ascent operations are conducted by qualified divers or combat swimmers. These operations require the supervision of an Ascent Supervisor but operational conditions preclude the use of instructors.

- Other factors in the prevention of gas embolism include good planning and adherence to the established dive plan. Trying to extend a dive to finish a task can too easily lead to the exhaustion of the air supply and the need for an emergency ascent. The diver shall know and follow good diving practices and keep in good physical condition. The diver shall not hesitate to report any illnesses, especially respiratory illnesses such as colds, to the Diving Supervisor or Diving Medical Personnel prior to diving.

20-3 DECOMPRESSION SICKNESS

Decompression sickness results from the formation of bubbles in the blood or body tissues, and is caused by inadequate elimination of dissolved gas after a dive or other exposure to high pressure. Decompression sickness may also occur with exposure to subatmospheric pressures (altitude exposure), as in an altitude chamber or sudden loss of cabin pressure in an aircraft. In certain individuals, decompression sickness may occur from no-decompression dives, or decompression dives even when decompression procedures are followed meticulously. Various conditions in the diver or in the diver's surroundings may cause absorption of an excessive amount of inert gas or may inhibit the elimination of the dissolved gas during normal controlled decompression. Any decompression sickness that occurs must be treated by recompression. The following paragraphs discuss the diagnosis of the various forms of decompression sickness. Once the correct diagnosis is made, the appropriate treatment from [Chapter 21](#) can be chosen based on the initial evaluation.

- 20-3.1 Initial Episode of Decompression Sickness.** A wide range of symptoms may accompany the initial episode of decompression sickness. The diver may exhibit certain signs that only trained observers will identify as decompression sickness. Some of the symptoms or signs will be so pronounced that there will be little doubt as to the cause. Others may be subtle and some of the more important signs could be overlooked in a cursory examination.

20-3.2 Differentiating Type I and Type II Symptoms. For purposes of deciding the appropriate treatment, symptoms of decompression sickness are generally divided into two categories. Type I decompression sickness includes skin symptoms, lymph node swelling and joint and/or muscle pain and is not life threatening. Type II decompression sickness (also called serious decompression sickness) includes symptoms involving the central nervous system, respiratory system, or circulatory system. Type II decompression sickness may become life threatening. Because the treatment of Type I and Type II symptoms may be different, it is important to distinguish between these two types of decompression sicknesses. Type I and Type II symptoms may or may not be present at the same time.

20-3.3 Type I Decompression Sickness. Type I decompression sickness includes joint pain (musculoskeletal or pain-only symptoms) and symptoms involving the skin (cutaneous symptoms), or swelling and pain in lymph nodes.

20-3.3.1 Musculoskeletal Pain-Only Symptoms. The most common symptom of decompression sickness is joint pain. Other types of pain may occur which do not involve joints. The pain may be mild or excruciating. The most common sites of joint pain are the elbow, wrist, hand, knee, and ankle. The characteristic pain of Type I decompression sickness usually begins gradually, is slight when first noticed and may be difficult to localize. It may be located in a joint or muscle, may increase in intensity, and is usually described as a deep, dull ache. The pain may or may not be increased by movement of the affected joint, and the limb may be held preferentially in certain positions to reduce the pain intensity (so-called guarding). The hallmark of Type I pain is its dull, aching quality and confinement to particular areas. It is always present at rest; it may or may not be made worse with movement.

20-3.3.1.1 Differentiating Between Type I Pain and Injury. The most difficult differentiation is between the pain of Type I decompression sickness and the pain resulting from a muscle sprain or bruise. If there is any doubt as to the cause of the pain, assume the diver is suffering from decompression sickness and treat accordingly. Frequently, pain may mask other more significant symptoms. Pain should not be treated with drugs in an effort to make the patient more comfortable. The pain may be the only way to localize the problem and monitor the progress of treatment.

20-3.3.1.2 Abdominal and Thoracic Pain. Pain in the abdominal and thoracic areas, including the hips and shoulders, may:

- Be localized to joints between the ribs and spinal column or joints between the ribs and sternum.
- Present a shooting-type pain that radiates from the back around the body (radicular or girdle pain).
- Appear as a vague, aching (visceral) pain.

Any pain occurring in these regions should be considered as symptoms arising from spinal cord involvement. Treat it as Type II decompression sickness.

- 20-3.3.2 **Cutaneous (Skin) Symptoms.** The most common skin manifestation of diving is itching. Itching by itself is generally transient and does not require recompression. Faint skin rashes may be present in conjunction with itching. These rashes also are transient and do not require recompression. Mottling or marbling of the skin, known as *cutis marmorata* (marbling), may precede a symptom of serious decompression sickness and shall be treated by recompression as Type II decompression sickness. This condition starts as intense itching, progresses to redness, and then gives way to a patchy, dark-bluish discoloration of the skin. The skin may feel thickened. In some cases the rash may be raised.
- 20-3.3.3 **Lymphatic Symptoms.** Lymphatic obstruction may occur, creating localized pain in involved lymph nodes and swelling of the tissues drained by these nodes. Recompression may provide prompt relief from pain. The swelling, however, may take longer to resolve completely and may still be present at the completion of treatment.
- 20-3.4 **Type II Decompression Sickness.** In the early stages, symptoms of Type II decompression sickness may not be obvious and the stricken diver may consider them inconsequential. The diver may feel fatigued or weak, and attribute the condition to overexertion. Even as weakness becomes more severe, the diver may not seek treatment until walking, hearing, or urinating becomes difficult. For this reason, symptoms must be anticipated during the postdive period and treated before they become too severe.
- 20-3.4.1 **Differentiating Between Type II DCS and AGE.** Many of the symptoms of Type II decompression sickness are the same as those of arterial gas embolism, although the time course is generally different. (AGE usually occurs within 10 minutes of surfacing.) Since the initial treatment of these two conditions is the same and since subsequent treatment conditions are based on the response of the patient to treatment, treatment should not be delayed unnecessarily in order to make the diagnosis in severely ill patients.
- 20-3.4.2 **Type II Symptom Categories.** Type II, or serious symptoms, are divided into three categories: neurological, inner ear (staggers), and cardiopulmonary (chokes) symptoms. Type I symptoms may or may not be present at the same time.
- 20-3.4.2.1 **Neurological Symptoms.** These symptoms may be the result of involvement of any level of the nervous system. Numbness, paresthesias (a tingling, pricking, creeping, “pins and needles,” or “electric” sensation on the skin), decreased sensation to touch, muscle weakness, paralysis, mental status changes, or motor performance alterations are the most common symptoms. Disturbances of higher brain function may result in personality changes, amnesia, bizarre behavior, lightheadedness, incoordination, and tremors. Lower spinal cord involvement can cause disruption of urinary function. Some of these signs may be subtle and can be overlooked or dismissed by the stricken diver as being of no consequence.

The occurrence of any neurological symptom is abnormal after a dive and should be considered a symptom of Type II decompression sickness or arterial gas embolism, unless another specific cause can be found. Normal fatigue is not uncommon

after long dives and, by itself, is not usually treated as decompression sickness. If the fatigue is unusually severe, a complete neurological examination is indicated to ensure there is no other neurological involvement.

20-3.4.2.2 **Inner Ear Symptoms (“Staggers”).** The symptoms of inner ear decompression sickness include: tinnitus (ringing in the ears), hearing loss, vertigo, dizziness, nausea, and vomiting. Inner ear decompression sickness has occurred most often in helium-oxygen diving and during decompression when the diver switched from breathing heliox to air. Inner ear decompression sickness should be differentiated from inner ear barotrauma, since the treatments are different. Staggers has been used as another name for inner ear decompression sickness due to the afflicted diver's difficulty in walking. However, symptoms of the staggers may be due to neurological decompression sickness involving the cerebellum. Typically, rapid involuntary eye movement (nystagmus) is not present in cerebellar decompression sickness.

20-3.4.2.3 **Cardiopulmonary Symptoms (“Chokes”).** If profuse intravascular bubbling occurs, symptoms of chokes may develop due to congestion of the lung circulation. Chokes may start as chest pain aggravated by inspiration and/or as an irritating cough. Increased breathing rate is usually observed. Symptoms of increasing lung congestion may progress to complete circulatory collapse, loss of consciousness, and death if recompression is not instituted immediately.

20-3.5 Time Course of Symptoms. Decompression sickness symptoms usually occur shortly following the dive or other pressure exposure. If the controlled decompression during ascent has been shortened or omitted, the diver could be suffering from decompression sickness before reaching the surface.

20-3.5.1 **Onset of Symptoms.** In analyzing several thousand air dives in a database set up by the U.S. Navy for developing decompression models, the time of onset of symptoms after surfacing was as follows:

- 42 percent occurred within 1 hour.
- 60 percent occurred within 3 hours.
- 83 percent occurred within 8 hours.
- 98 percent occurred within 24 hours.

20-3.5.2 **Dive History.** While a history of diving (or altitude exposure) is necessary for the diagnosis of decompression sickness to be made, the depth and duration of the dive are useful only in establishing if required decompression was missed.

NOTE **Decompression sickness can occur in divers well within no-decompression limits or who have carefully followed decompression tables.**

20-3.5.3 **When Treatment Is Not Necessary.** If the reason for postdive symptoms is firmly established to be due to causes other than decompression sickness or arterial gas embolism (e.g., injury, sprain, poorly fitting equipment), then recompression is

not necessary. If the diving supervisor cannot rule out the need for recompression, then commence treatment.

20-3.6 Altitude Decompression Sickness. Aviators exposed to altitude may experience symptoms of decompression sickness similar to those experienced by divers. The only major difference is that symptoms of spinal cord involvement are less common and symptoms of brain involvement are more frequent in altitude decompression sickness than hyperbaric decompression sickness. Simple pain, however, still accounts for the majority of symptoms.

20-3.6.1 Joint Pain Treatment. If only joint pain was present but resolved before reaching one ata from altitude, then the individual may be treated with two hours of 100 percent oxygen breathing at one atmosphere followed by 24 hours of observation. If symptoms persist after return to one ata from altitude, the stricken individual should be transferred to a recompression facility for treatment.

20-3.6.2 Transfer and Treatment. Individuals should be kept on 100 percent oxygen during transfer to the recompression facility. If symptoms have resolved by the time the individual has reached a recompression facility, they should be examined for any residual symptoms. If any decompression symptom had been present at any time or if even the most minor symptom is present they should be treated with the appropriate treatment table as if the original symptoms were still present.