



DEPARTMENT OF VETERANS AFFAIRS
Veterans Benefits Administration
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Director (00/21)
All VA Regional Offices and Centers

Training Letter 07-04
In Reply Refer To: 211

SUBJ: Medical Consequences of Diving

Purpose

The purpose of this training letter is to provide information on the disabilities that may result from diving. We want to ensure that VA decision makers are informed of the medical consequences of diving so that claims from veterans who were divers in service are properly developed and adjudicated.

Who to contact for additional information

If you have questions about the content of this letter, please contact Rhonda F. Ford (Rhonda.Ford@va.gov, 202-461-9739).

/s/

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Enclosure

Medical Consequences of Diving

A. Introduction

This letter is meant to provide information on the disabilities that may result from diving, so that veterans who were divers will be assured of receiving a thorough and informed consideration of their claims for benefits. The potential medical consequences of diving, which are quite diverse, have not been a focus of VBA training, and there is only limited information available on this subject in general medical textbooks.

This letter summarizes the short- and long-term medical effects of diving and provides a brief summary of pertinent technical diving information. Consult the selected references listed at the end of the letter for more detailed information about the historical, technical, and medical aspects of diving.

B. Diving techniques

Diving techniques include free (breath-hold) diving, snorkeling, SCUBA (Self Contained Underwater Breathing Apparatus) diving, surface supplied air and mixed-gas bounce diving, and saturation diving. This letter focuses primarily on the potential long-term disabling effects of saturation and non-saturation (bounce) diving, including both the residuals of decompression sickness (DCS) and other long-term problems. Under certain circumstances, any type of diving may result in decompression sickness, barotraumas (tissue damage due to increased pressure), and pulmonary overinflation syndromes (trapping of gases in lungs, with potential for rupture of alveoli leading to arterial gas emboli). These conditions are discussed below.

1. **Descent**

As a diver descends, increasing pressure causes increasing amounts of gases in the lungs to diffuse into the body. Increased oxygen can be partially metabolized by the body, but the nitrogen (or other inert gas) that is absorbed remains in the body until removed by pulmonary gas exchange during ascent.

2. **Ascent**

When a diver ascends and the pressure decreases (decompression), the gas that was absorbed on descent diffuses back out of the tissues and is exhaled. However, if the ascent is too rapid, gas bubbles form in tissues and blood vessels, and may result in mechanical blockage of blood vessels to vital organs, compression of nerves, pressure pain within joints, and disruption of other tissues, resulting in symptoms of decompression sickness (DCS). DCS is treated by recompression in a hyperbaric (high pressure) chamber, which reduces the size of bubbles and creates a favorable diffusion gradient that allows a slow, controlled release of the gas from body tissues without allowing the reformation of bubbles.

Deeper dives have a greater risk of DCS. What is considered to be a deep dive varies with the type of equipment used, the gas used, and other factors. While 100 feet would be considered a deep dive in recreational diving, a deep dive in commercial and military diving is often 300 feet or more. A diver may need to stop and wait for a period of time several times during the ascent to allow gas diffusion out of the tissues to take place slowly, in order to decrease the risk of DCS. Oxygen may be used during decompression to facilitate the elimination of inert gas.

Flying shortly after diving increases the risk of DCS because the ascent to altitude (with decreased pressure) allows residual nitrogen in body tissues to diffuse out as gas bubbles, as on ascent from the dive.

3. Saturation diving

Saturation diving is a special diving technique developed by the U.S. Navy in the late 1960s that permits divers to work underwater at great depths for days, weeks, or even months at a time. Examples of its use are for salvage work and for construction projects. Saturation means that a diver has been at a depth long enough (typically 24 hours or longer) for tissues to have absorbed the maximum amount of gas possible for that particular depth, so that gas equilibrium has been achieved. Once saturation has taken place, additional time at depth, whether a day, a week, or a month, will not increase the required decompression time. Although not used as commonly as it once was, because ROVs (remotely operated underwater vehicles) and AUVs (autonomous underwater vehicles) now carry out many routine underwater tasks, saturation diving is still used for certain tasks.

Saturation divers live for long periods of time in a chamber pressurized to the desired depth, and work in an extremely harsh, physically demanding environment. While many of the medical effects described in this letter can result from non-saturation diving, some of the effects are more common and more severe in saturation divers.

4. Bounce diving

In military and commercial diving, a bounce dive commonly refers to any non-saturation dive. Often multiple bounce dives of different depths and duration are conducted in a single day, and repeated decompressions are necessary. Both bounce diving and saturation diving are considered to be stressful types of diving.

In recreational diving, a bounce dive is a descent of short duration (usually less than ten minutes) with an almost immediate ascent to keep the time needed for decompression to a minimum.

5. Advantages of saturation diving

One advantage of saturation diving is that the diver will need only a single decompression, which takes place in a pressurized chamber, rather than the multiple decompressions that would be required if multiple shorter dives (bounce dives) were used. Decompression is an extremely long process that may last days or weeks after deep dives of over 200 ft, so the need for only a single decompression saves considerable time and is therefore more efficient. In addition, saturation diving diminishes the overall risk of DCS for a project because there is only one gradual decompression rather than multiple short decompressions.

C. DCS (Caisson disease)

1. **Time of onset, predisposing factors, and treatment** - Symptoms of DCS may appear while still ascending, or almost immediately after surfacing (with 95% occurring within three hours), and nearly all appear within 24 hours after the dive ends. DCS has been divided into various descriptive types (see below) depending on the particular symptoms, but some divers experience multiple types, and it is the specific organs affected rather than the designation of type that is important for rating purposes. Many of the signs and symptoms resolve rapidly, but some persist for weeks or months, and some are associated with permanent injury or disability.

Predisposing causes of DCS include technical factors such as inadequate decompression, too rapid ascent, and flying too soon (12-24 hours) after diving; personal factors such as smoking, fatigue, and obesity; and environmental factors such as cold water, heavy physical labor, rough seas, and heated diving suits. However, DCS may occur even when all factors are favorable.

Divers with all types of DCS are treated with oxygen combined with recompression in a pressurized chamber, and then undergo gradual decompression while breathing oxygen so that bubbles do not reform. If treatment is begun within minutes of symptom onset, most symptoms resolve, but it is not known whether subtle injury may persist.

2. **Type I DCS**

The musculoskeletal system and skin are primarily affected in this type, with signs and symptoms including:

- a. mild to severe pain (“ the bends”), mainly in the joints of the arms and legs
- b. itching, mottling, and rashes of skin

3. **Type II DCS**

The brain, spinal cord, and organs of special sense are primarily affected in this type. Spinal cord damage occurs more often than brain damage.

Signs and symptoms include:

- a. Central nervous system (CNS) DCS - weakness or paralysis (hemiplegia, paraplegia, quadriplegia), decreased sensation, paresthesias, peripheral neuropathy, speech difficulty, headache, bladder and bowel sphincter abnormalities, change in mental status (such as odd behavior, confusion, loss of alertness), and other findings, such as extreme fatigue. However, any type of neurologic deficit may occur.
- b. Labyrinthine DCS (“the staggers”) - nausea, vomiting, vertigo, nystagmus, loss of equilibrium, tinnitus, and hearing loss.
- c. Visual disturbances – scotomas, diplopia, vision loss
- d. Pulmonary DCS (“the chokes”) - respiratory symptoms, such as burning substernal chest pain, cough, shortness of breath, and respiratory distress
- e. Thrombus formation and nitrogen emboli to any organ – can result in stroke, myocardial infarction, etc., depending on the organs affected
- f. Hypovolemic shock – tachycardia and postural hypotension
- g. Most extreme cases – coma and death within hours

4. **Arterial gas emboli (AGE)** (generally appears within minutes after surfacing) When gas emboli, which are inert gas bubbles that form in the venous blood during decompression, enter the arterial circulation, the emboli can then lodge in coronary and cerebral arteries and in other arteries throughout the body with potentially catastrophic results.

One source of gas emboli entering the arterial circulation is overinflation of the lungs due to excessive pressure. This can cause rupture of pulmonary alveoli, which allows gas from the lungs to enter the arterial circulation.

Another source of arterial emboli is patent foramen ovale. Almost 30% of people retain a patent foramen ovale, an opening between the left and right atria of the heart that is present in the fetus but that closes by the age of one year in most people. While generally a benign finding, it provides a connection between the right and left sides of the circulation, and therefore may allow gas emboli to enter the arterial circulation.

The effects of arterial gas emboli depend on which arteries are affected; stroke, seizures, other CNS effects, and myocardial infarction, for example, may occur. As with other manifestations of DCS, some of the effects of AGE may be transient, but some may cause permanent disability.

5. Long-term and late effects of DCS

Although most symptoms of DCS resolve with treatment, residual permanent damage sometimes occurs. Repetitive cases of DCS are believed to cause cumulative damage.

- a. Dysbaric osteonecrosis is a degenerative disease of bone due to the death of an area of bone tissue (avascular bone necrosis, bone infarct), and is thought to be due to nitrogen embolization. It is believed to be caused by the cumulative effects of unrecognized DCS. It is most common in the shoulders and hips, but may involve any bone. Infarcts in the shaft of a bone are usually asymptomatic and of no clinical significance, but those that are close to a joint may result in persistent pain and severe, disabling arthritis that develops months or years later. Pathologic fractures may also occur.
- b. Neurologic abnormalities include partial or complete paralysis, peripheral neuropathy, etc. (see Type II DCS manifestations, above). Common residual neurologic symptoms include varying degrees of muscle weakness, sensory deficits or paresthesias, and coordination difficulties. Cognitive deficits are also possible.
- c. Chronic skin conditions
- d. Hearing loss, vertigo, tinnitus, dysequilibrium
- e. Residuals of arterial emboli, with serious examples being myocardial infarction and stroke.

D. Toxic effects of gases in diving

1. **High pressure nervous (or neurological) syndrome (HPNS)** - This condition may appear during diving at great depths (over 600 fsw (feet of salt water)) using a mixture of helium and oxygen. Symptoms include dizziness, nausea, vomiting, tremors, incoordination, fatigue, somnolence, myoclonic jerking, stomach cramps, decreased intellectual performance, and disturbed sleep. These neurological effects limit the depth of human diving. It has been found that adding a small fraction of nitrogen to the gas mixture and using a proper compression rate may reduce the serious symptoms. Whether or not there are long-term residual effects from HPNS is still unclear.

2. **Nitrogen narcosis (“the narcs,” “the rapture of the deep”)**

Nitrogen narcosis results from increased pressure of nitrogen that occurs after a depth of about 100 fsw. It resembles the effects of alcohol intoxication, with euphoria, confusion, irrational behavior, paranoia, and hallucinations. While it resolves upon ascent, it may be deadly, because it may result in coma, or the CNS effects may lead to poor judgment, resulting in fatal accidents.

3. **Oxygen toxicity**

Oxygen toxicity of the CNS may occur when oxygen in high concentrations is inhaled under pressure. It is characterized by muscle twitching, seizures, and vision and hearing problems. There may also be pulmonary effects such as cough and substernal burning with prolonged exposure. Death may result from extreme exposures. Risk of CNS oxygen toxicity increases with increasing depth, increased oxygen fraction, and increased oxygen exposure time. It can begin to occur at a depth of slightly over 170 fsw when breathing air and can occur at depths as shallow as 20 fsw when using an oxygen rebreather.

4. **Carbon dioxide toxicity** may occur when there are closed (rebreather) diving system malfunctions that allow carbon dioxide to accumulate in the diver's breathing gas. Normally, in rebreather systems the carbon dioxide is removed by a scrubber before fresh oxygen is added and the gas breathed out is recycled. Carbon dioxide toxicity can affect respiration and cause headaches or other CNS effects, including unconsciousness.

5. **Atmospheric contaminants** such as carbon monoxide, hydrocarbon fuel vapors, or vapors from paints, solvents, or welding fumes may lead to either acute toxic effects, or subtle, unrecognized long-term toxicity.

To avoid most of these toxic reactions, deep divers use various mixtures of oxygen, nitrogen, helium, and sometimes hydrogen, with the percentage of oxygen used depending on the depth. In military and commercial diving, great care is taken to reduce known atmospheric contaminants, but unrecognized contamination is a constant risk.

E. Other potential long-term effects of diving

1. **Ear and hearing effects**

The diving environment exposes the auditory system to intense pressure changes, noise, physical injury, and contaminated water. This may result in:

- Chronic otitis externa

- Hearing loss from noise exposure – one of the most common symptoms of divers

- Perforated tympanic membrane – may occur from barotrauma from a too rapid descent

- Round or oval window rupture - The round and oval windows are membrane-covered openings between the middle ear and the inner ear, and the rupture of a window may result in sudden hearing loss, tinnitus, and vertigo. Symptoms may resolve or may require surgery.

2. **Neurologic (cognitive) effects**

There is conflicting medical information about whether or not long-term neurologic effects can result from diving (with and without DCS). Standard neurological examinations and tests ordinarily do not reveal any abnormalities. However, some studies have found that divers have neurological complaints, and

subtle abnormalities have been found on special testing. Other studies have shown mild abnormalities on testing but no clinical abnormalities. A concern about the studies is that there are no large-scale controlled studies.

The most prominent symptoms reported have been difficulties in concentration and problems with long and short term memory. One study conducted neuropsychological testing in 64 divers before and after deep diving. Some of the divers showed hand tremor, impaired spatial memory, reduced finger coordination, and autonomic nervous system abnormalities after the dives, and these findings did not improve after one year.

A study of 156 Norwegian divers compared with 100 nondiving controls reported the following symptoms believed to be related to DCS: fatigue, mood lability, irritability, difficulty concentrating, memory problems, and autonomic nervous system symptoms. Physical findings included tremor, a modified Romberg sign (a test of balance), and decreased sensation in the feet.

Another study reported that abnormal neurologic exams were associated with exposure to air and saturation diving and a history of DCS. The changes were considered mainly minor and not sufficient to affect the quality of life.

The risk of neurological injuries is believed to be related to DCS, anoxia (from near drowning), and gas toxicities, but no consensus exists as to whether or not diving per se causes brain damage, as many studies show no late effects.

3. Alterations of liver enzymes

These have been reported but have not been associated with clinical illness.

4. Pulmonary effects

Airflow obstruction due to airway narrowing has been reported, but to date, this change has not been found to affect the diver's health. A few divers may have significant pulmonary injury.

5. Hypothermia

Divers may spend many hours working in cold water. Any of the effects of non-freezing cold injury may be seen, with the hands and feet being most likely to be affected.

6. Thermal injuries

Heated suits used to avoid cold exposure may, at times, result in burns. In hot environments, heat stress or even heat stroke may occur.

7. PTSD

Potential stressors include experiencing near-death situations in accidents, witnessing the serious injury or death of co-workers, explosions, etc. In one study, 43% of divers said they had experienced the death of a diving friend, and

over 80% reported having been in dangerous situations that were difficult to put behind them.

8. Delayed arthritis

This includes osteoarthritis, discussed above under dysbaric osteonecrosis, or osteoarthritis secondary to compression arthralgia. Compression arthralgia or compression pains is a condition of painful joints (knees, shoulders, fingers, back, hips, neck, ribs, and occasionally low back) due to increased external pressure. It occurs during rapid compression and may result from interference with joint lubrication. This, in turn, may affect joint function and potentially lead to arthritis, especially after repeated dives.

F. Rating implications

The only current diagnostic code specific to diving residuals is 5011, Bones, caisson disease of. The condition is rated as arthritis, cord involvement, or deafness, depending on the severity of the disabling manifestations. All of these conditions may be separately evaluated under appropriate diagnostic codes, as they are not likely to have overlapping or duplicative signs and symptoms. However, this letter points out that numerous other disabilities may result from diving. In order to identify their origin as diving-related disabilities, all could be rated under diagnostic code 5011, hyphenated with the appropriate diagnostic code used for the actual evaluation.

Many of these conditions will be reported in the service medical records, but some, such as bone infarcts and arthritis, do not appear immediately and may not be reported in service. In other cases, symptoms may have been mild or subtle and went unreported or unnoticed. With a history of diving in service, and evidence of a bone infarct or arthritis, especially of the hip or shoulder, a diving etiology should be considered. The same is true of many other residuals, such as hearing loss, other ear or labyrinthine abnormalities, neurologic abnormalities, and skin abnormalities.

Many of the veterans who did saturation diving in the early days (at least from the 1960's, 70's, and possibly 80's) worked under different circumstances from those doing diving today, as new techniques and safety measures have been progressively developed. Therefore, it is possible that early divers experienced more complications than more recent divers. The lists below are some of the disabilities that you may encounter in a veteran with a history of diving, but this is not an exclusive list of all possible disabilities that may result from diving.

Musculoskeletal

- Bone infarcts (Dysbaric osteonecrosis)

- Osteoarthritis, particularly of shoulders and hips, occurring months to years after diving, due to DCS

- Arthritis of joints following compression arthralgia, mainly in saturation divers (initial pain occurs mainly in knees, shoulders, fingers, back, hips, neck, and ribs)

Chronic skin conditions (hands are a common site)

- Eczema
- Infection

Ear

- Chronic otitis externa
- Hearing loss
- Perforated eardrum
- Vestibular problems
- Tinnitus

CNS complications of DCS, including arterial emboli

- Stroke
- Seizures
- Paralysis or weakness
- Peripheral neuropathy
- Autonomic symptoms
- Any other neurologic deficit

Other long-term neurologic abnormalities

While there are some studies showing long-term neurologic effects and other studies showing none, the studies continue, and raters should be aware of the possibility of neurologic damage in veterans who were divers, whether or not they experienced DCS. Most commonly reported are cognitive effects (short-term memory loss and difficulty concentrating), but other neurologic abnormalities have also been described.

Visual disturbances

- Scotomas
- Diplopia
- Vision loss up to complete blindness, either cortical (due to effects on brain) or arterial (due to effects on ophthalmic or retinal arteries)

Pulmonary problems – obstruction to airflow, almost always with minimal or no clinical findings, but some divers may have clinically evident restrictive lung disease

Cardiac problems – myocardial infarction residuals, arrhythmias

Cold injury – any of the non-freezing cold injury residuals

Thermal injuries

PTSD

Medical Opinions: In claims from veterans who were divers, most issues necessitating a medical opinion will be similar to medical opinions needed in any case. The procedures in M21-1MR, III.iv.3.A.9 should be followed. However, when questions arise about the relationship of a claimed condition to diving, particularly when the claimed condition is not addressed in this paper, or when especially complex or unusual questions arise, consultation with medical diving experts may be needed. These questions should be submitted to the Director of Compensation and Pension Service (211) for an opinion.

G. Selected references for further information

US Navy diving manual (2005), at <http://www.supsalv.org/pdf/Diveman.pdf>

The Merck Manual (18th edition, 2006)

Bennet and Elliot's Physiology and Medicine of Diving, 5th edition, A.O. Brubak, T.S. Neuman editors, Saunders Publishers, 2003

Bove' and Davis' Diving Medicine, 3rd edition, A.O. Bove, editor, Saunders Publisher, 1997

Undersea and Hyperbaric Medical Society (UHMS), <http://www.uhms.org>

Long-term effects of diving – <http://www.medscape.com/viewarticle/408486>

Medical problems - <http://www.diversalertnetwork.org/medical/articles> and <http://www.osha.gov/SLTC/commercialdiving/more.html>

Brain damage in divers - <http://www.bmj.com/cgi/content/full/314/7082/689>

Decompression syndrome - http://www.e-med.co.uk/diving/decompression_sickness.php and http://findarticles.com/p/articles/mi_g2601/is_0004/ai_2601000406

Technical information about diving and equipment - http://en.wikipedia.org/wiki/Saturation_diving

History and background of saturation diving - <http://www.divingheritage.com/saturationkern.htm>

Norwegian studies of North Sea divers, including chronic neurologic effects and PTSD - <http://www.nui.no/adm/dokument/Ross.pdf>

Saturation diving - http://seagrants.wisc.edu/madisonjason11/diving_saturation.html

High pressure nervous syndrome - <http://scuba-doc.com/HPNS.html>

SCUBA diving medical effects - <http://www.scuba-doc.com/LTE.htm>

Chronic neurological effects - <http://scuba-doc.com/chrneur.htm>

Compression arthralgia - <http://www.scuba-doc.com/cmprarth.html>