# Identifying the Subtle Presentation of Decompression Sickness

Kenneth Alea

**BACKGROUND:** Decompression sickness is an inherent occupational hazard that has the possibility to leave its victims with significant long-lasting effects that can potentially impact an aircrew's flight status. The relative infrequency of this hazard within the military flying community along with the potentially subtle presentation of decompression sickness (DCS) has the potential to result in delayed diagnosis and treatment, leading to residual deficits that can impact a patient's daily life or even lead to death.

**CASE REPORT:** The patient presented in this work was diagnosed with a Type II DCS 21 h after a cabin decompression at 35,000 ft (10,668 m). The patient had been asymptomatic with a completely normal physical/neurological exam following his flight. The following day, he presented with excessive fatigue and on re-evaluation was recommended for hyperbaric therapy, during which his symptoms completely resolved. He was re-evaluated 14 d later and cleared to resume flight duties without further incident.

**DISCUSSION:** The manifestation of this patient's decompression sickness was subtle and followed an evaluation that failed to identify any focal findings. A high index of suspicion with strict follow-up contributed to the identification of DCS in this case, resulting in definitive treatment and resolution of the patient's symptoms. Determination of the need for hyperbaric therapy following oxygen supplementation and a thorough history and physical is imperative. If the diagnosis is in question, consider preemptive hyperbaric therapy as the benefits of treatment in DCS outweigh the risks of treatment. Finally, this work introduces the future potential of neuropsychological testing for both the diagnosis of DCS as well as assessing the effectiveness of hyperbaric therapy in Type II DCS.

**KEYWORDS:** decompression sickness, fatigue, aviation, military, hyperbaric therapy, occupational hazard.

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Decompression sickness (DCS) is an inherent danger to flight crew that is characterized as an occupational hazard which has the potential to cause long and lasting effects, rending aircrew permanently disabled or even leading to death. Fortunately, the occurrence of DCS is not commonly seen by flight surgeons, but therein lays the risk of unrecognized symptoms and misdiagnosis. This delay in identification can lead to the difference between residual symptoms and full recovery. The purpose of this case study and topic discussion is to review a particular case in which local protocol led to the timely diagnosis and treatment of suspected DCS as well as provide background and possible protocols that may assist other flight surgeons in the evaluation and treatment of similar cases.

According to the U.S. Navy Aeromedical Reference and Waiver Guide, the aeromedical concern of decompression sickness is residual neurological/neuropsychological impairment resulting in safety of flight issues.[6] A multisystem disorder, decompression sickness results from exposure to rapid decrease in barometric pressure, leading to the escape of inert gases out of solution, such as nitrogen.[1] This leads to the formation of gas bubbles in tissue and venous blood that can lead to a litany of symptoms depending on the location of these bubbles. The freed gas bubbles can alter organ function by occluding blood vessels, distending/rupturing or compressing tissue, or activating inflammatory and clotting pathways.[2,4] Factors important to the severity of symptoms associated with DCS are the quantity and size of bubbles, location, and the type of reactions

From the U.S. Navy, Beaufort, SC.

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Address correspondence to: Kenneth Alea, M.D., Medical Department, U.S. Navy, 598 Geiger Blvd., Bldg. 598, Beaufort, SC 29904; kenalea@hotmail.com.



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caused.[9] These gas emboli have been found to modify vascular endothelium through adhesion molecule-mediated endothelial activation in addition to stimulating platelets.[9] Additionally, microparticles originating from vascular walls as a result of decompression stress also seem to be a factor. The relationship between these factors appears to be the cause of DCS rather than simply the formation of bubbles alone.[9] Henry's law explains the formation of bubbles in decompression sickness; it states that at a constant temperature, the amount of a gas that is dissolved in a liquid is directly proportional to the partial pressure of that gas.[4]

It is difficult to predict who is at risk for decompression sickness, but there are certain well-defined predisposing factors that can be helpful during diagnosis. Altitude is the most important predisposing factor for DCS. While no specific altitude has been identified as a clear limit below which people are considered safe from DCS, there is little evidence of DCS occurring in healthy individuals below 18,000 ft (5486.4 m). The largest prevalence of DCS cases occur at altitudes of 25,000 ft (7620 m) or higher.[3] A study conducted by the U.S. Air Force reported only 13% of DCS cases occurred below 25,000 ft (7620 m).[3] Repetitive exposures in relative rapid succession increases the risk of DCS. A rapid increase in altitude above 18,000 ft (5486.4 m), such as seen in a rapid decompression, increases the risk of DCS when compared to a gradual increase in altitude to the same level. Duration at altitudes above 18,000 ft (5486.4 m) or decreased ambient temperatures also contribute to possible DCS. According to Sheffield, approximately 75% of patients with DCS develop symptoms within 1 h and 90% percent within 12 h of the physiological event; only a small number become symptomatic more than 24 h after an insult.[4,11]

Clinically, DCS is characterized into Type 1 and Type 2 depending on the organ systems involved, with Type 2 being the more severe manifestation of the disorder. Type 1, most commonly known as the bends, usually results in pain due to its propensity to affect the musculoskeletal system, skin, and lymphatic system. Localized joint pain is the most common manifestation of DCS, occurring in 70% of patients.[1] Interestingly, the hips and knees are the most commonly affected joints in aviators, although any joint may be involved.[1] The reason for this is not known. Joint manipulation normally does not exacerbate pain and localized tenderness or evidence of inflammation is rarely seen.[4,10] Pruritus, mainly affecting the upper chest, may also be described, along with localized erythema that may become blotchy areas of cyanosis, which can be seen on examination of the skin.[4] Lymphatic involvement is rare, but can lead to pain, lymphadenopathy, and localized edema, usually with a peau d'orange effect and follicular depressions, again seen namely on the chest and torso.[4]

Type 2 DCS is a more severe manifestation of the disorder, with the potential for permanent damage and death, and is manifested by a wide range of signs and symptoms due to the arbitrary nature by which DCS affects the nervous system. While neurological symptoms have been described in up to 60% of sport divers, the military reports that neurological symptoms occur in only 10–20% of DCS cases.[1,4] The spinal cord is the most common site affected by Type 2 DCS, with symptoms mimicking spinal cord trauma. The lower thoracic and upper lumbar regions are the most common sites of spinal cord injury in DCS. Clinically, the patient may experience paresthesias and weakness, progressing to paraplegia, and sphincter dysfunction, with bladder involvement possible. Cerebral manifestations can also result in memory loss, ataxia, visual disturbances, and personality changes, as well as changes in speech and affect.[4] Pulmonary DCS, known as the chokes, can be seen in 5% of cases. Gas bubbles can obstruct portions of the pulmonary circulation, producing chest pain, wheezing, dyspnea, and pharyngeal irritation.[4] Disruption of right ventricular outflow coupled with acute right-sided cardiac failure, circulatory collapse, and death may also occur.[4] Severe fatigue that cannot be explained by the activity performed in flight may be an early indicator of DCS. While the etiology is unknown, a feeling of profound fatigue following possible rapid decompression deserves careful evaluation for other signs of DCS and may in and of itself justify treatment.[1]

## CASE REPORT

The incident pilot describes the following during the event that led to the generation of this case study and topic review. On 6 May 2014 at approximately 1455 and 35,000 ft (1,0668 m), during a functional check flight, a number of Integrated Caution and Warning Systems (ICAWS) annunciated. Immediately upon annunciation of these ICAWS, the incident pilot felt his cabin depressurize and began feeling symptoms similar to his Reduced Oxygen Breathing Device/hypoxia training. He manually turned the backup oxygen supply on and began a rapid descent to below 17,000 ft (5181.6 m) mean sea level and began to return to base. He landed uneventfully at base and taxied the aircraft back to the line. The elapsed times from annunciation of the ICAWS to both on deck and shut down were approximately 14 and 21 min, respectively. At 1+15 h after landing, he presented to a flight surgeon for evaluation.

At presentation, the incident pilot denied any current symptoms, but was transported to the base emergency room (ER) for further evaluation per local protocol. Upon arriving at the ER, oxygen therapy was initiated and a thorough evaluation was performed. Oxygen therapy was initiated approximately 1+45 following landing. History detailed the pilot had been flying at 35,000 ft (1,0668 m) with a set cabin pressure of 14,500 ft (4419.6 m) when depressurization occurred. The pilot was unable to determine how high cabin pressure reached during the decompression due to preoccupation handling other procedures. He described the immediate onset of hypoxic symptoms within seconds of receiving the first ICAW and reacted by rapidly descending to below 17,000 ft (5181.6 m) mean sea level and manually turning the backup oxygen supply on. He described his hypoxic symptoms as feeling light headed with a warm/flushed feeling with a slight degradation in concentration. The symptoms were identical to those experienced during Reduced Oxygen Breathing Device/hypoxia training and they

improved as soon as he descended below 17,000 ft (5181.6 m) and went on supplemental oxygen. The pilot denied any loud noise, rush of air, misting, or barotrauma to the ears at the time of decompression.

The physical exam, which consisted of a complete cardiopulmonary, dermatological, musculoskeletal, neurological, and mini-mental exam, demonstrated no focal findings. Following completion of the history and physical, a hyperbaric specialist was consulted and at the time no indications for hyperbaric therapy were present. Oxygen therapy was continued and prior to 2 h of therapy, oxygen was removed and the patient was assessed again. During the evaluation, the patient began to experience a unilateral temporal headache. Hyperbarics was consulted again given the development of this new symptom off therapy. The recommendation was to restart oxygen therapy and assess for resolution of the headache. When the headache resolved, the hyperbaric specialist believed that the headache was the result of a vaso-vagal phenomenon due to oxygen therapy and not explained by possible DCS. Oxygen therapy was discontinued following an addition 30 min, at which point the pilot was doing well and remained asymptomatic. When another physical failed to demonstrate any focal findings, the pilot was discharged with strict precautions and parameters for which he should seek medical attention if symptoms developed. Additionally, the patient was instructed to follow up with the squadron flight surgeon the following morning.

As instructed, the pilot followed up the next morning with the squadron flight surgeon. On evaluation, he described feeling light-headed, which he stated had been present since exiting the aircraft the day prior and that he had gotten dizzy while getting out of bed that morning. Given these symptoms, the squadron flight surgeon consulted hyperbarics and was instructed to transport the pilot for further evaluation and probable treatment. En route, the pilot complained of lightheadedness and fatigue. He described his fatigue as similar to the feeling of waking from a nonrestorative night of sleep. He was drowsy, but still able function and there was no evidence of altered mental status or deviations in personality. He was able to ambulate without assistance and without any gait abnormalities. Upon arrival at the hyperbaric treatment facility, he was further evaluated and the patient's fatigue was concerning enough for DCS to warrant hyperbaric therapy. Chamber treatment started roughly 21 h after landing. The pilot stated that his symptoms resolved approximately 3 h into the 6-h chamber ride. Upon release, he remained symptom free with no further complaints stemming from this incident. The pilot was evaluated by a Navy dive medical officer 2 wk post-incident in the presence of the squadron flight surgeon and cleared to return to flight status without limitations.

## DISCUSSION

Treatment of significant DCS includes fluids, 100% oxygen, and proper positioning of the patient.[4] It was once believed that the incidence of cerebral embolism could be reduced by placing the patient in Trendelenburg, taking advantage of the hydrostatic effect resulting from placing the head below the body's center of gravity.[7] While this was once standard practice to prevent cerebral gas embolization, more modern works by Edmunds, Pulley, and de Watteville recommend against placing the patient in Trendelenburg, citing that having the head lowered can exacerbate damage to the blood-brain barrier due to an increase in intracranial pressure.[7–9] Placing the patient in left lateral decubitus position is thought to encourage air to travel superiorly to a nonobstructing location by returning normal blood flow by positioning the right ventricular outflow tract inferior to the right ventricle.[4,13] This too has seemed to fall out of favor. Current recommendations suggest simply placing the patient in the supine position.[8] Locally, it is protocol that when a pilot initiates an in-flight emergency due to a rapid decompression, air traffic control notifies the base emergency room of the physiological event in flight. The emergency room responds via ambulance to the flight line, where the pilot is met at the aircraft and is transported directly to the ER while receiving 100% oxygen through his own $O_2$ mask, which is connected to an oxygen delivery system for evaluation in the ER. Definitive treatment includes hyperbaric oxygen therapy in a recompression chamber, initiated as quickly as possible.

Outcome, in part, is determined by how quickly hyperbaric therapy is initiated following onset of symptoms. Following Boyle's law, which states that at a constant temperature the volume of a gas varies inversely with the pressure to which it is subjected, hyperbaric oxygen treatment decreases the volume of air bubbles and also provides oxygenation to hypoxic tissue by increasing the dissolved oxygen content of arterial blood.[4] Favorable outcomes have been shown to occur in nearly 91% of Type 2 DCS patients, according to Chandy and Weinhouse: "Complete resolution of symptoms in Type 2 DCS is seen in almost 75% of cases while 16% of patients may have residual symptoms for up to three months. Delayed treatment has an adverse effect, with one study reporting a decrease in successful outcomes from 75 to 57% when treatment is delayed beyond 12 h. Recompression treatment should never be withheld even after a long delay because favorable results can still occur in this setting."[4] Unfavorable effects due to hyperbaric therapy are relatively rare; thus, when DCS is suspected but difficult to confirm, proceeding with hyperbaric therapy may be valuable given the possible ramification of untreated DCS.

Studies performed at the Navy Experimental Diving Unit published in 1988 demonstrated through neuropsychological testing that central nervous system (CNS) injury from Type II DCS may be more refractory to therapy than initially believed. In their studies, neurological and neuropsychological symptoms returned within hours to days following initial recompression, even though recompression initially suppressed all CNS symptoms. Even though neurological tests performed on their subjects failed to reveal any deficits, neuropsychological testing was able to reveal quantifiable evidence that the subject still suffered impairment.[5] These findings illustrate that the presentation of DCS in patients may be so subtle as to go undetected by current evaluation methods as well as the fact that, in some

cases, recompression therapy may need to be repeated due to the refractory nature of CNS insults from DCS. Lastly, their findings raise an interesting question regarding the potential role of neuropsychology in the future among the aviation community.

Thus the following protocol may serve as an effective approach for the evaluation and potential treatment of pilots subjected to rapid decompression and thus at risk for possible DCS:

1. First, the pilot should be placed on 100% oxygen as soon as possible.
   - Ideally, the pilot should go on oxygen as soon as he egresses the aircraft and be transported to the nearest point of medical care for evaluation.
2. Once at the treatment facility, the patient should be maintained on oxygen for at least 2 h and positioned in the supine position.
3. A thorough history and physical should be attained to assess risk for DCS as well as examine for signs and symptoms concerning for DCS.
   - It is important to note cabin pressure setting for the flight and if possible how high cabin altitude spiked during the decompression, as this is an important factor in predicting the potential for DCS acutely.
4. The physical should include a thorough neurology exam, including a cognitive assessment to evaluate for focal findings as these could be indicative of Type 2 DCS and would require coordination for hyperbaric therapy as soon as possible.
   - The Mini-Mental Status (MMSE) exam has long been a neuropsychological study of choice to determine cognitive impairment such as that seen in DCS. Recent studies have demonstrated that perhaps the Montreal Cognitive Assessment may be more sensitive in identifying mild cognitive impairment. Smith et al. demonstrated that the MMSE had a sensitivity of 17% while the Montreal Cognitive Assessment had a sensitivity of 83% in identifying mild cognitive impairment.[12] The specificity for the MMSE over the Montreal Cognitive Assessment was 100–50%, respectively.[12] A larger study performed more recently by Whitney et al. demonstrated that the Montreal Cognitive Assessment was 72% sensitive while the MMSE's sensitivity was 52% sensitive.[14] They were able to demonstrate that specificity was similar between the two tests, with MMSE being 77% specific while the Montreal Cognitive Assessment was 75% specific.[14]
5. Following completion of a thorough history and physical, the case should be presented to a hyperbaric specialist for further recommendations and, if indicated, coordination for hyperbaric therapy.
   - If hyperbaric therapy is indicated, then proceed as directed to the nearest facility with capabilities to provide the therapy.
6. If hyperbaric therapy is not indicated following evaluation and consultation, releasing the patient with strict precautions for seeking medical attention and follow-up the next day to assess for development of delayed symptoms which may require therapy, such as in the case shared above, is recommended.

As outlined by the Aeromedical Waiver Guide, pilots should be grounded for at least 3 d following Type 1 DCS and at least 14 d for Type 2 DCS with no evidence of residual effects. Documentation of a normal evaluation by a neurologist or dive medical officer should be obtained prior to returning a patient to flight status.[1]

In conclusion, the diagnosis of decompression sickness can be a difficult and sometimes confusing diagnosis to make. In the event that a pilot is suspected of potentially being affected by DCS in the absence of a definitive diagnosis, preemptive hyperbaric therapy is recommended, as the risk of undergoing hyperbaric therapy is minimal when compared to the potential outcome of untreated DCS. The establishment of a standard protocol adopted by medical personnel responsible for the treatment of pilots that is communicated to tenant squadrons specifies a structured approach for the evaluation of these patients as well as familiarizes the squadrons with the necessary steps required to ensure the safety of pilots in the event of a physiological event in flight, making them an active participant in pilot safety. Lastly, the aviation community should look further into the role of neuropsychological testing as a way to potentially diagnose DCS, but also to assess the effectiveness of hyperbaric therapy.

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*Author and affiliation:* Kenneth Alea, M.D., Medical Department, U.S. Navy, Beaufort, SC.

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