

TITLE: Recommending an Approach for NASA Toward Neurologic Decompression Illness

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ABSTRACT:

INTRODUCTION: Decompression illness, including decompression sickness and arterial gas embolism, may occur during extravehicular activity, with neurologic cases traditionally considered to pose a high risk of permanent disability or death. While developing lunar extravehicular activity denitrogenation protocols with human trials, National Aeronautics and Space Administration physicians uncovered subtle neurologic deficits in two test subjects. These findings challenged the conventional paradigm that neurologic decompression sickness is always serious and raised the question of whether mild cases could be accepted in the context of lunar extravehicular activity operations. **METHODS:** The literature was reviewed to characterize mild neurologic decompression illness in terms of epidemiology and clinical course in the absence of hyperbaric oxygen treatment. **RESULTS:** While a significant proportion of mild neurologic decompression illness cases may resolve without hyperbaric oxygen treatment, progression to severe status commonly occurs even in cases with initially mild manifestations. Furthermore, ground-level oxygen treatment has not always proven adequate to resolve deficits of neurologic decompression illness. **DISCUSSION:** Though mild neurologic decompression illness may resolve without hyperbaric oxygen treatment at higher rates than more severe forms, the rate of progression to functional impairment remains too high to accept in the setting of lunar exploration missions. Without a robust predictive model for deterioration, all neurologic decompression illness should receive prompt hyperbaric oxygen treatment regardless of initial presentation severity. The National Aeronautics and Space Administration should remain vigilant in their approach to neurologic decompression illness and strategize how to reduce the risk and impacts of even initially mild cases.

KEYWORDS: neurologic decompression illness, neurologic decompression sickness, Type II decompression sickness, lunar extravehicular activity

Introduction:

The history of hyperbaric medicine has been marked by ever-shifting classification systems for decompression sickness (DCS).¹ The most enduring classification places cases into Type I (pain only, often considered mild) and Type II (neurologic or cardiopulmonary, often considered serious) categories. Various investigators have also attempted to devise severity scores based on specific neurologic findings to predict outcomes with more precision than a simple mild-or-severe binary. To this day, what constitutes a mild or serious form of DCS, particularly regarding neurologic manifestations, remains a matter of considerable debate. According to the U.S. Navy Diving Manual, neurologic symptoms at “any level of the nervous system” would be considered evidence of a serious Type II DCS.² For a time, the U.S. Air Force labeled DCS with peripheral nervous system (PNS) involvement, indicated by “a little numbness or paresthesia in a limb,” as mild Type I, but they eventually reverted to categorizing all neurologic DCS as Type II.¹ While the Navy and Air Force label all neurologic symptoms as Type II DCS, the National Aeronautics and Space Administration (NASA) continues to separate neurologic cases into “Type I” (PNS involvement) and “Type II” (central nervous system (CNS) involvement).³

Accurately understanding DCS, especially its neurologic forms, has become increasingly crucial for NASA as preparations are made for lunar exploration missions. Extravehicular activity (EVA), colloquially referred to as “spacewalking,” is an integral mission component that presents a risk for decompression illness (DCI), which includes DCS and arterial gas embolism (AGE). Neurologic injury from either entity, even if not life-threatening, may be disabling enough to jeopardize mission objectives. While NASA has not reported any cases of DCS in astronauts performing EVA to date, future lunar missions are likely to pose a higher risk of DCS than previously encountered. Apollo-era EVAs precluded DCS risk by using a 100% oxygen cabin environment, which presents a fire risk that is no longer considered acceptable. In addition, DCS has been successfully prevented in the nitrogen-containing conditions of the International

Space Station through the use of lengthy prebreathe protocols. However, the concept of operations for planned lunar EVAs will require shorter prebreathe times, repetitive EVAs, and heavy use of the lower body, all of which increase the risk of DCS.⁴ Furthermore, in an exploration setting, capability for clinician diagnosis and definitive treatment with hyperbaric oxygen (HBO) will likely be limited or absent. As the risk of neurologic DCI varies depending on parameters like suit pressures and EVA durations, the appropriate level of risk tolerance toward neurologic DCI must first be agreed upon for NASA to develop appropriate lunar extravehicular protocols.

Currently, NASA's Spaceflight Human-System Standard, NASA-STD-3001, requires each program to "prevent Type II DCS" at a 95% confidence level.⁵ A key method for mitigating DCS risk is to use prebreathe protocols for denitrogenation. By varying the durations, pressures, and fractions of inspired oxygen (FiO_2) and investigating whether test subjects who undergo such prebreathe protocols develop DCS when performing simulated EVA, researchers can determine whether or not a given prebreathe protocol would satisfy the standard for DCS risk mitigation.

While studying rates of DCI under various lunar EVA denitrogenation protocols, NASA physicians uncovered two subtle cases of neurologic DCS. These findings challenged conventional conceptualizations of neurologic DCS severity and raised new questions concerning how NASA should mitigate neurologic DCI risk for lunar EVA.

Case 1

A test subject in their 30s participated in a study developing denitrogenation protocols for lunar EVA. At baseline, the subject was ambidextrous (i.e., writing with the left hand, throwing with the right hand) with a normal neurologic examination. The subject was scheduled to undergo multiple simulated EVAs

after achieving nitrogen saturation in a 28.5% oxygen environment at 9.6 psia. Prior to each EVA, the subject would denitrogenate with a 90-minute, 95% oxygen prebreathe and depressurize to the EVA condition of 4.3 psia (Figure 1). During one EVA, routine ultrasound studies revealed left-sided intracardiac bubbles at about 2 hours and 45 minutes after arriving at 4.3 psia, meeting criteria for test termination. (Upon further questioning, the subject noted that during the EVA they experienced “fullness” in the right 3rd and 4th fingers and that their knuckles did not crack as they usually would.) Descent on 100% oxygen was initiated. About 30 minutes after the bubbles were noted (3 hours and 17 minutes after arriving at 4.3 psia), a complete neurologic examination was performed that revealed nystagmus, left lower extremity (LLE) hip flexion strength 4+/5 (right lower extremity (RLE) hip flexion strength 5/5), LLE hyperesthesia (i.e., tingling with light touch), and a very slight right upper extremity (RUE) elbow flexion/extension weakness. HBO treatment with Navy Treatment Table 6 was initiated, and at repeat examinations 45 minutes after arriving at 60 fsw (1 hour and 33 minutes after the bubbles were noted, 4 hours and 18 minutes after arriving at 4.3 psia) and beyond, all symptoms and signs had resolved.

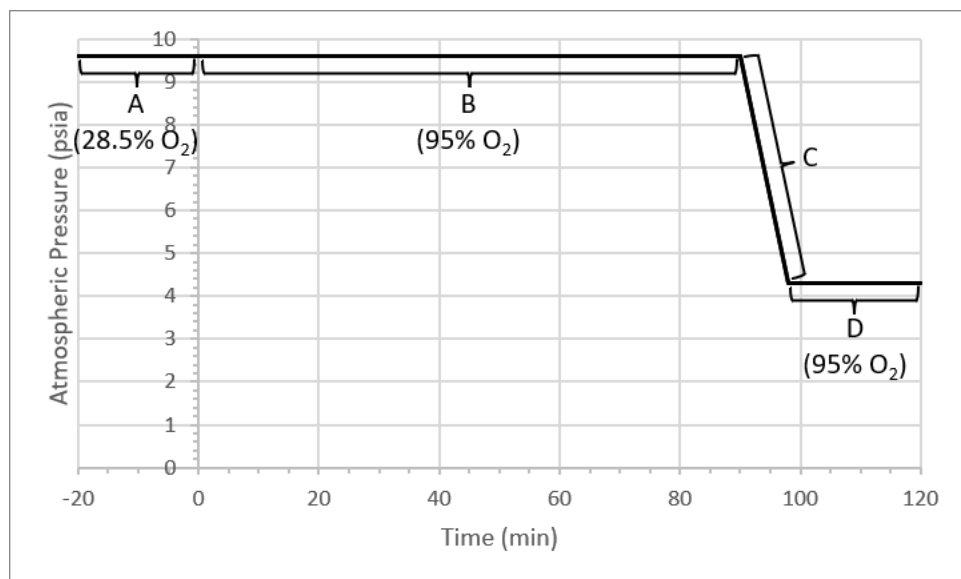


Figure 1. Case 1 atmospheric profile, first 120 min. A) The subject begins at the saturation condition of 9.6 psia, 28.5% O₂ for an indefinite amount of time. B) The subject undergoes a 90-min prebreathe using 95% O₂. C) The environment is depressurized over 8 min to 4.3 psia. D) The subject begins extravehicular activity at 4.3 psia, 95% O₂. Note: The full duration of extravehicular activity is not depicted.

Case 2

A test subject in their 30s participated in a study developing denitrogenation protocols for lunar EVA. At baseline, the subject was right-hand dominant with a normal neurologic examination. The subject was scheduled to undergo multiple simulated EVAs after achieving nitrogen saturation in a 28.5% oxygen environment at 9.6 psia. Prior to each EVA, the subject would denitrogenate with a 20-minute, 95% oxygen prebreathe and depressurize to the EVA condition of 5.2 psia (Figure 2). At about 7 minutes into one of these EVAs, the subject reported that they had experienced thigh and upper calf tingling during the EVA which resolved within about 2 minutes; later questioning revealed that these paresthesias were bilateral. At 90 minutes after arriving at 5.2 psia, the subject reported unilateral “cramping pain” in a knee and toe joint, meeting criteria for test termination for suspected DCS. Descent on 100% oxygen was initiated, during which the knee and foot pain resolved at around 9.2-9.4 psia. Once at site level (in Houston, Texas, approximately 13 feet above sea level),⁶ the patient was continued on 100% oxygen. Approximately 28 minutes after the lower extremity pain was reported (1 hour and 58 minutes after arriving at 5.2 psia), a complete neurologic examination revealed decreased deep tendon reflexes (DTRs) of 1+ throughout all extremities, RUE shoulder abduction/adduction strength 4+/5, and RUE elbow flexion/extension strength 4+/5 (left upper extremity (LUE) shoulder and elbow strength 5/5). About 1 hour later (1 hour and 33 minutes after the lower extremity pain was reported, 3 hours and 3 minutes after arriving at 5.2 psia), DTRs increased to 3+, and it was decided to convert to HBO treatment with Navy Treatment Table 6 given the presentation of CNS involvement. About 20 minutes after arriving at

60 fsw (2 hours and 57 minutes after the lower extremity pain was reported, 4 hours and 27 minutes after arriving at 5.2 psia), examination revealed resolved RUE weakness and stable DTRs at 3+, which remained unchanged during repeat examinations later that day. On examination the next day, DTRs had normalized to 2+, and 1-2 beats of horizontal nystagmus were noted bilaterally.

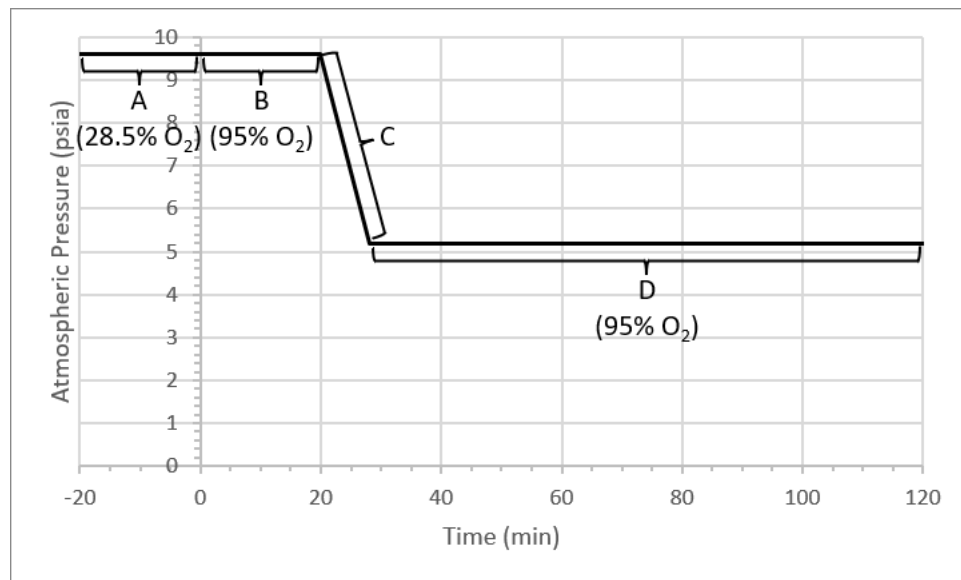


Figure 2. Case 2 atmospheric profile, first 120 min. A) The subject begins at the saturation condition of 9.6 psia, 28.5% O₂ for an indefinite amount of time. B) The subject undergoes a 20-min prebreathe using 95% O₂. C) The environment is depressurized over 8 min to 5.2 psia. D) The subject begins extravehicular activity at 5.2 psia, 95% O₂. Note: The full duration of extravehicular activity is not depicted.

These cases are notable for several reasons. First, the neurologic findings in each case were difficult to diagnose, even for clinicians. In Case 1, the test subject was entirely unaware of any of their symptoms; it was the ultrasound revealing left-sided bubbles that triggered the physical examination leading to the diagnosis of DCS. In Case 2, it was the joint pain, not the transient paresthesias, that triggered a diagnosis of DCS, and CNS involvement was recognized only when hyporeflexia transitioned to hyperreflexia. Given that astronauts in exploration missions would likely be unable to diagnose

asymptomatic cases of neurologic DCS or accurately localize a neurologic lesion, the utility of classifications distinguishing between PNS and CNS involvement was called into question. Second, the neurologic findings were exceedingly subtle. Whether such mild symptoms would pose a mission-impactful functional impairment was questionable. Assuming these manifestations remained stable without HBO treatment, perhaps even CNS involvement such as in Case 2 might be acceptable for astronauts to experience on exploration missions.

It became necessary to investigate whether mild forms of neurologic DCI exist that would be acceptable in exploration EVA (xEVA) contexts, and whether these mild forms of neurologic DCI could be distinguished by symptoms alone. In order to pursue answers to these questions, the following definition of mild neurologic DCI was proposed for the exploration context:

1. One of the following:
 - a. Unilateral paresthesia without objective sensory deficit
 - OR
 - b. 4+/5 motor strength asymmetry without functional impairment
- AND
2. No cognitive deficits

Figure 3. Proposed definition of mild neurologic DCI

This definition was applied to DCI instead of more narrowly to DCS because DCS and AGE could be especially challenging to distinguish in-mission, whether by astronauts or terrestrially based clinicians. Furthermore, in an astronaut with a patent foramen ovale (PFO), bubbles formed by DCS in the venous

system could cross into the systemic arterial circulation and produce findings consistent with AGE. PFO is a common anatomic variant, with a prevalence of approximately 24% in the general population.⁷

With this definition in mind, the following research questions were identified:

1. Does the proposed definition of mild neurologic DCI have clinical utility?
 - a. Do milder presentations of neurologic DCI differ from more severe cases in terms of natural history?
 - i. How often do milder presentations of neurologic DCI resolve without HBO treatment?
 - ii. How often do milder presentations of neurologic DCI deteriorate without HBO treatment?
 - b. Do milder presentations of neurologic DCI differ from more severe cases in their response to ground-level oxygen (GLO) treatment?
2. What proportion of neurologic DCI presentations meet the proposed definition of mild neurologic DCI?
3. Should development of xEVA pre-breathe protocols consider mild neurologic DCI a separate category from type I and type II DCS and allow for the occurrence of mild neurologic DCI on xEVA?
4. Should crewmembers who have experienced mild neurologic DCI after xEVA be treated as Type I DCS (i.e., automatically allowed to return to duty 24 hours after symptom resolution) or as Type II DCS (i.e., medical review and waiver from NASA's Aerospace Medicine Board (AMB) required prior to return to alternobaric duty) with regards to disposition?

In this literature review, the desired primary outcomes were the proportion of initially mild neurologic DCI presentations that resolve and the proportion that deteriorate without HBO treatment. A secondary outcome was the proportion of neurologic DCI presentations that meet the proposed definition of mild neurologic DCI. The aim of this investigation was to inform an appropriate risk posture for NASA toward mild neurologic DCI, including whether it is allowable during exploration EVA and what the disposition should be in the case that it does occur. It was hypothesized that, even with a mild initial presentation, there remains a high enough probability of neurologic DCI deteriorating into a disabling, life-threatening, or mission-compromising event that NASA should continue to reject neurologic DCI and strategize how to reduce its risk of occurrence and its impacts.

Methods:

A literature review was performed using databases including PubMed, Google Scholar, NASA Technical Reports Server (NTRS), and Defense Technical Information Center (DTIC) to accumulate available data relevant to the research aims, including articles that clarify the natural history of neurologic DCI in the absence of HBO treatment, its response to GLO treatment, and the epidemiology of its presenting symptoms. Although neurologic DCI that occurs from hypobaric exposure may behave differently from neurologic DCI that occurs after diving, the relative paucity in hypobaric DCI literature necessitated the inclusion of human data from all relevant settings (i.e., diving, hypobaric chambers, aviation, spaceflight). As the published literature was lacking in higher-quality evidence, such as randomized controlled trials, case reports were also included as appropriate.

Sources were excluded if any of the following criteria were met: non-English language, insufficient access to the publication to determine if inclusion criteria were satisfied, non-human animal study, or study focused on test results (e.g., laboratory, imaging) instead of clinical findings. Sources were included if

they provided insight into any of the following topics: natural history of mild neurologic decompression illness, proportion of neurologic decompression illness meeting proposed definition of mild neurologic decompression illness (Figure 3), and treatment response of mild neurologic decompression illness to GLO only. Date of publication was not factored into consideration when determining whether or not to include a given source.

Initially, a PubMed search was performed using the following MeSH (Medical Subject Heading) terms: ("decompression sickness/diagnosis"[MeSH Terms]) OR ("decompression sickness/complications"[MeSH Terms]) OR ("decompression sickness/classification"[MeSH Terms])) AND ((paresis[MeSH Terms]) OR ("paresthesia"[MeSH Terms]) OR ("reflex, abnormal"[MeSH Terms])). Given that some results cited Boussuges et al., 1996, a paper producing a severity scale that includes clinical course prior to HBO treatment, Google Scholar was then searched for Boussuges et al., 1996 and the articles that cited it.⁸ Finally, the NTRS and DTIC were searched using the terms “neurologic decompression sickness” and “neurologic decompression illness.” A total of 445 article abstracts was reviewed and narrowed down to 11 publications for inclusion (Figure 4, Table 1).

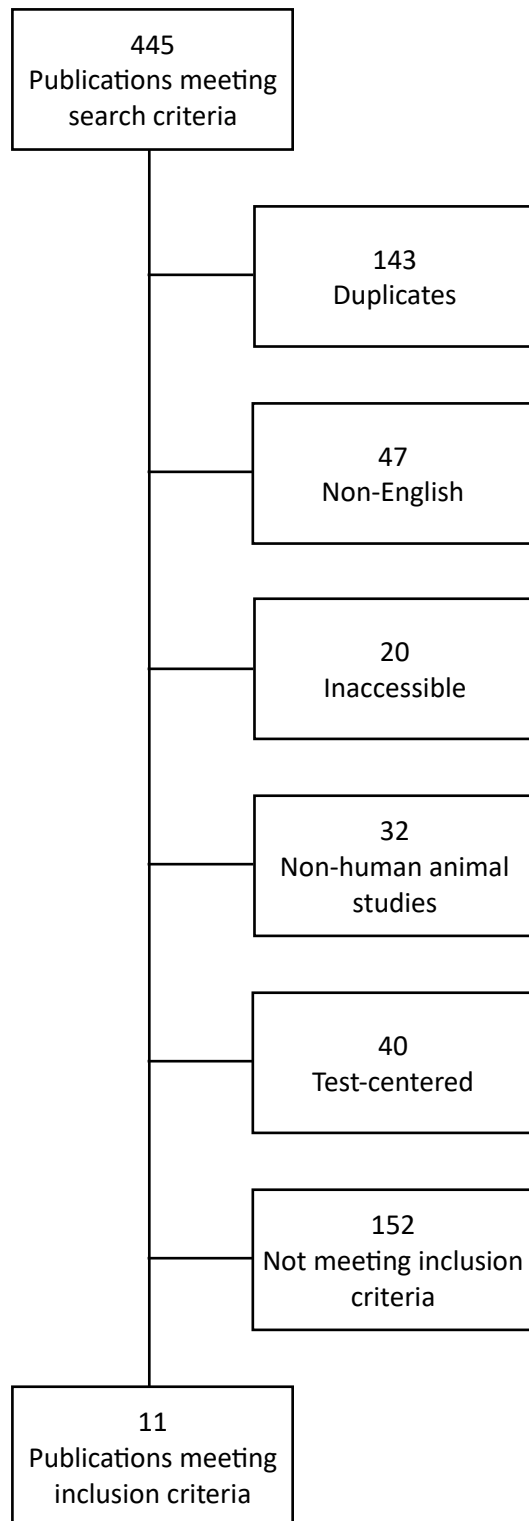


Figure 4. Literature review flowchart

Table 1. Summary of findings from publications included for review, categorized by research question

Q1.a.i. How often do milder presentations of neurologic DCI resolve without HBO treatment?			
Authors, Year	Clinical Entity (exposure)	Notable Findings	Caveats + Notes
Dick & Massey, 1985 (+ Mitchell, 2005)	Mild neurologic DCS (recreational divers)	10/11 (90.9%) resolved without HBO treatment	Different definition of mild neurologic DCS; unclear if cases resolved spontaneously or with GLO treatment
Monnot et al., 2019	Mild DCI (recreational divers)	54/61 (88.5%) resolved without HBO treatment	Different definition of mild DCI; not all cases were neurologic; some cases received GLO treatment; diagnoses made by self-reported symptoms on online survey
Linér & Andersson, 2010	Suspected cerebral AGE (positive airway pressure)	Case of R shoulder paresthesia and R neck numbness induced by glossopharyngeal insufflation under normobaric conditions; spontaneous resolution within 3-5 minutes	Initial presentation included objective sensory deficit
Q1.a.ii. How often do milder presentations of neurologic DCI deteriorate without HBO treatment?			
Authors, Year	Clinical Entity (exposure)	Notable Findings	Caveats + Notes
Boussuges et al., 1996	CNS or spinal cord DCI excluding cerebral AGE from pulmonary barotrauma (recreational divers)	7% of Cohort 1 and 6% of Cohort 2 deteriorated prior to HBO treatment	All cases eventually received HBO treatment; cases of all severity levels included
Pitkin et al., 1999	Neurologic DCI (Navy divers)	59/217 (27.2%) deteriorated prior to HBO treatment	All cases eventually received HBO treatment; cases of all severity levels included
Blatteau et al., 2011 (<i>Neurocritical Care</i>)	Spinal cord DCS (recreational divers)	66/269 (24.5%) deteriorated prior to HBO treatment	All cases eventually received HBO treatment; cases of all severity levels included
Butler & Pinto, 1985	PNS DCS (Navy diver)	Case of mild sensory symptoms in L finger that progressed to functionally impairing weakness requiring multiple HBO treatments	Initial presentation included objective sensory deficit
Q1.b. Do milder presentations of neurologic DCI differ from more severe cases in their response to GLO treatment?			
Authors, Year	Clinical Entity (exposure)	Notable Findings	Caveats + Notes
Dully, 1975	CNS DCI (hypobaric chamber)	Case of asymptomatic LUE and LLE hyperreflexia despite resolution of joint pain from GLO treatment	Presenting symptom of joint pain
Johnston, 2008	PNS DCS (sudden cabin depressurization)	Case of R finger numbness that resolved with 2 hours of GLO treatment	Initial presentation included objective sensory deficit
Q2. What proportion of neurologic DCI presentations meet the proposed definition of mild neurologic DCI?			
Authors, Year	Clinical Entity (exposure)	Notable Findings	Caveats + Notes
Blatteau et al., 2011 (<i>Diving and Hyperbaric Medicine</i>)	Spinal cord or cerebral DCS excluding cerebral AGE from pulmonary barotrauma (Navy divers)	17/59 (28.8%) with isolated limb paresthesia	Definition of spinal cord or cerebral DCS unclear but may require bilateral findings
Blatteau et al., 2011 (<i>Neurocritical Care</i>)	Spinal cord DCS (recreational divers)	140/279 (50.2%) with isolated paresthesia	Definition of spinal cord DCS requires bilateral findings or unilateral findings with associated back pain or bladder symptoms

AGE: arterial gas embolism; CNS: central nervous system; DCI: decompression illness; DCS: decompression sickness; GLO: ground-level oxygen;

HBO: hyperbaric oxygen; L: left; LLE: left lower extremity; LUE: left upper extremity; PNS: peripheral nervous system; R: right

Results:

Does the proposed definition of mild neurologic DCI have clinical utility?

Do milder presentations of neurologic DCI differ from more severe cases in terms of natural history?

How often do milder presentations of neurologic DCI resolve without HBO treatment?

Dick & Massey, 1985 defined mild neurologic DCI as scores of 1-3 on the severity scale they devised (Table 2). Their study graded 70 cases of neurologic DCI according to their severity scale and reported that 10 mild cases resolved on their own without recompression.⁹ Another paper clarified that these 10 reported cases were a subset of 11 mild cases not treated with HBO, corresponding with a 90.9% resolution rate (Figure 5).¹⁰

Table 2. Dick & Massey, 1985 Severity Scale⁹

Sensory Symptoms	
Grade	
1 Paresthesia:	Single limb or area
2 Paresthesias:	Multiple regions
3 Numbness:	Single region or limb
4 Numbness:	Two regions or limbs
5 Numbness:	Three or more limbs
Motor Symptoms	
Grade	
1 Weakness:	Single limb or muscle group
2 Weakness:	Multiple limbs or muscle groups
3 Paralysis:	Single limb or muscle group
4 Paralysis:	Two limbs
5 Paralysis:	Three or more limbs
10	Total possible score

The total score is calculated by summing the “Sensory Symptoms” and “Motor Symptoms” scores

Monnot et al., 2019 collected self-reported online survey data about dive-related injuries from 784 recreational divers. After reviewing the reported symptoms, the investigators coded 114 cases of

presumed DCI, which they graded by the Boussuges et al., 1996 severity scale (Table 3).⁸ Of those rated as mild, corresponding to a Boussuges score <7, 25/31 (80.6%) who received first-aid with GLO treatment and 29/30 (96.7%) who received no treatment experienced complete resolution of symptoms without HBO treatment, for an overall rate of 54/61 (88.5%) (Figure 5).¹¹ These data include all forms of mild DCI based on the Boussuges scale and are not limited to those with neurologic symptoms; for example, pain-only DCS cases were likely counted.

Table 3. Boussuges et al., 1996 Severity Scale⁸

Score		0	1	2	3	4	5	6
Repetitive dive	No	x						
	Yes			x				
Clinical course before HBO	Improvement	x						
	Stability				x			
	Deterioration						x	
Objective sensory disorder	No	x						
	Yes					x		
Motor impairment	No	x						
	Monoparesis, paraparesis, or tetraparesis					x		
	Paraplegia							x
	Hemiplegia				x			
Urinary disturbances	No	x						
	Yes						x	

The total score is calculated by summing the “Repetitive dive,” “Clinical course before HBO,” “Objective sensory disorder,” “Motor impairment,” and “Urinary disturbances” scores. HBO: hypobaric oxygen

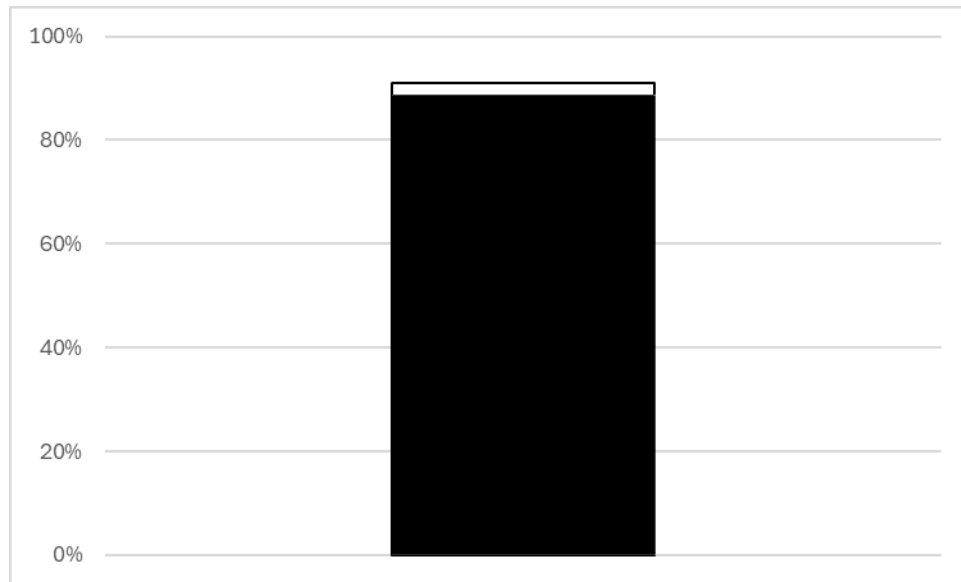


Figure 5. Rate of resolution of mild neurologic decompression sickness/mild decompression illness without hyperbaric oxygen treatment. The black bar represents a low estimate based on Monnot et al., 2019.¹¹ The white bar represents a high estimate based on Dick & Massey, 1985.⁹

Linér & Andersson, 2010 reported on an illustrative case of an 18-year-old male who likely sustained a cerebral AGE by practicing glossopharyngeal insufflation, a technique used by some breath-hold divers. Despite being in a normobaric environment, the lungs were pressurized to such an extent that the patient experienced paresthesias of the right shoulder and numbness of the right neck. Symptoms resolved spontaneously within 3-5 minutes.¹²

How often do milder presentations of neurologic DCI deteriorate without HBO treatment?

Three papers addressed rates of deterioration of neurologic DCS or DCI in the absence of HBO treatment. Boussuges et al., 1996 devised a severity scale in which one of the components was clinical course before HBO treatment (Table 3).⁸ Their study used two cohorts of divers at hyperbaric centers and scored the severity of each case of CNS or spinal cord DCI, excluding cases of suspected cerebral AGE from pulmonary barotrauma. Prior to HBO treatment, 7% of cases in Cohort 1 and 6% of cases in Cohort

2 deteriorated, while the remainder stayed clinically stable or improved.⁸ Similarly, the Boussuges scale has been used in other studies to grade neurologic DCI cases in Navy divers and spinal cord DCS cases in recreational divers at hyperbaric centers, where 59/217 (27.2%) and 66/269 (24.5%), respectively, were found to deteriorate prior to HBO treatment (Figure 6).^{13,14} All cases reported in these three papers ultimately received HBO treatment and were not limited to cases that could be considered mild.

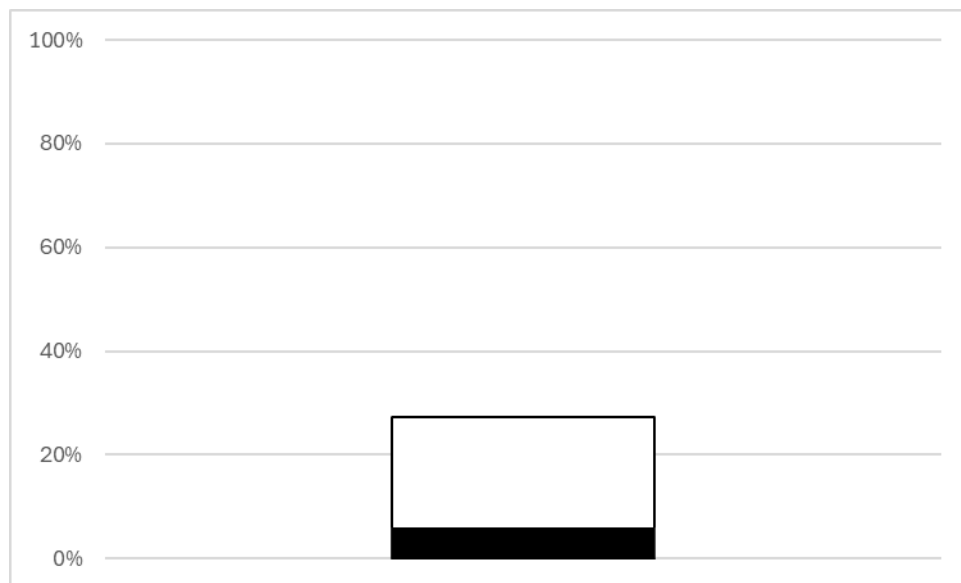


Figure 6. Rate of deterioration of neurologic decompression sickness/decompression prior to hyperbaric oxygen treatment. The black bar represents a low estimate based on Boussuges et al., 1996.⁸ The white bar represents a high estimate based on Pitkin et al., 1999.¹³

Finally, Butler & Pinto, 1985 reported on a case that demonstrates the possibility of mild neurologic cases deteriorating prior to HBO treatment. A 23-year-old male Navy diver developed numbness and paresthesia of left fifth finger 1 day after diving. The area of sensory involvement progressively spread, prompting presentation to a hyperbaric center. Despite receiving HBO treatments on day 6 and day 16 post-diving, the patient developed hand weakness on day 24 that eventually impaired his ability to open jars and perform other daily functions. A neurologic evaluation ruled out alternative etiologies and

concluded that the source of the symptoms was likely a lower brachial plexopathy or ulnar neuropathy due to DCS. Repeated HBO treatments improved symptoms, and residual symptoms eventually resolved completely within 4 months.¹⁵

Do milder presentations of neurologic DCI differ from more severe cases in their response to GLO treatment?

One published report described a case of pain-only DCS that was treated with GLO and found to have neurologic impairment that did not resolve with GLO. Dully, 1975 reported that a 37-year-old male Navy hospital corpsman participated in a 40,000 ft (12,000 m) hypobaric chamber flight and developed left ankle pain during descent. About 1 hour later, the pain persisted, prompting the patient to seek care and become hospitalized. On GLO, intravenous dexamethasone, and continuous intravenous normal saline, the patient's pain resolved by the next morning, but he was then found to have unilateral LUE and LLE hyperreflexia that fluctuated over the course of 4 days. LUE hyperreflexia was demonstrated again on exam weeks later.¹⁶

A different publication reported on a case of PNS DCS that did resolve with GLO treatment alone. The patient was a male riding a military aircraft when sudden cabin depressurization at 24,006 ft caused the effective altitude to increase from 7,001 ft to 24,006 ft. The patient used supplemental oxygen while the aircraft descended to below 10,000 ft within 5 minutes. Upon landing, the patient was found to have numbness of the tip of the right second finger. The symptoms resolved after 2 hours of GLO treatment.¹⁷

What proportion of neurologic DCI presentations meet the proposed definition of mild neurologic DCI?

Two papers reported proportions of spinal cord DCS cases that presented with isolated paresthesias without objective sensory or motor deficits.

Blatteau et al., 2011 (*Diving and Hyperbaric Medicine*) analyzed the presenting symptoms of Navy divers who were deemed to likely have spinal cord or cerebral DCS but not cerebral AGE due to pulmonary barotrauma. Cases were classified as presenting with “isolated limb paraesthesiae,” “objective sensory deficit,” or “motor impairment.” The authors found that 17/59 (28.8%) of cases presented with only paresthesias and no objective deficits (Figure 7).¹⁸

In a similar paper, Blatteau et al., 2011 (*Neurocritical Care*) classified cases of spinal cord DCS in recreational divers as presenting with “isolated paresthesia,” “objective sensory deficiency,” or “motor deficiency.” The investigators found that 140/279 (50.2%) of cases presented with isolated paresthesia (Figure 7). Their criteria defined spinal cord DCS as cases exhibiting bilateral neurologic findings and included unilateral cases only if they were associated with back pain or sphincter dysfunction.¹⁴



Figure 7. Proportion of central nervous system decompression sickness presenting with isolated paresthesia without objective sensory or motor deficit. The black bar represents a low estimate based on

Blatteau et al., 2011 (*Diving and Hyperbaric Medicine*).¹⁸ The white bar represents a high estimate based on Blatteau et al., 2011 (*Neurocritical Care*).¹⁴

Discussion:

This study has a number of limitations. Most notably, it was difficult to locate publications with patients that perfectly matched the proposed definition of mild neurologic DCI (Figure 3). Multiple case reports, for example, included patients with objective sensory deficits despite the proposed definition of mild neurologic DCI allowing only subjective sensory deficits (i.e., unilateral paresthesia).^{12,15,17} Moreover, illuminating the natural history of mild neurologic DCI required relying on other investigators' definitions of mild neurologic DCI, such as the severity scores created by Dick & Massey, 1985 and Boussuges et al., 1996.^{8,9} These severity scores categorized certain findings as mild (e.g., single limb paralysis) that exceed the limits of this study's proposed definition of mild neurologic DCI.⁹ In addition, the Boussuges study included non-neurologic DCI cases in their classification of mild and added points for characteristics such as "repetitive dive" that do not translate to specific physical examination findings or symptoms, potentially inflating scores compared to this study's mild neurologic DCI concept.⁸

Another limitation of the study is its heavy reliance on diving literature. The altitude DCI literature is relatively scarce, and the only relevant cases that were available were from the altitude chamber and cabin depressurization event. While interpreting the remainder of the literature, it is important to consider that DCI caused by diving exposures may behave differently from DCI caused by altitude exposures.

Also limited are this study's estimates of the rate of deterioration of mild neurologic DCI in the absence of HBO treatment. All the publications that reported clinical course prior to HBO treatment involved only

cases that eventually received HBO treatment.^{8,13,14} Furthermore, the cases that were included were not limited to those that were mild. Therefore, the estimates of deterioration might be biased in the positive direction given that the cases factoring into the estimates were likely more severe.

As for the data on response of neurologic DCI to GLO treatment, the primary limitation was a lack of adequate literature. A couple cases of relatively mild neurologic DCI were available to show that sometimes GLO treatment is sufficient to resolve findings while other times it is not.^{16,17} However, a rate of response to GLO treatment was not able to be estimated for either mild or severe neurologic DCI.

Finally, estimates of the proportion of neurologic DCI that is mild were limited by the cases included in the two publications by Blatteau et al., 2011. While it was possible to calculate a proportion that exhibited paresthesias only without any objective sensory or motor deficit, the inclusion criteria for those studies was patients with spinal cord or CNS DCS, not all neurologic DCI.^{14,18} According to one of the papers, most of the cases that were included had bilateral symptoms.¹⁴ It is difficult to predict whether estimates would be over-estimates or under-estimates.

Despite the above limitations, the reviewed literature does provide some insight into the nature of mild neurologic DCI cases. It remains possible to derive a moderately informed recommendation for NASA's approach to lunar EVA.

The data indicate that a significant proportion of neurologic DCS cases, approximately 88.5-90.9%, resolve without HBO treatment.^{9,11} Making the unlikely assumption that all the mild cases in Monnot et al., 2019 were neurologic without any pain-only presentations, the rate of spontaneous resolution increases to around 96.7%.¹¹ Even in this best-case estimate, though, at least 3.3% of such cases would

likely exhibit residual deficits in the absence of HBO treatment. The only consideration that may reduce this rate of residual deficits is the fact that the definition of mild neurologic DCS and mild DCI used in the reviewed literature include some cases with objective sensory and motor deficits that would be beyond this study's proposed definition of mild neurologic DCI, but the lack of clarity in the currently published data precludes further refinement of the estimate.

A significant proportion of neurologic DCS and DCI cases deteriorate prior to HBO treatment (6-27.2%).^{8,13,14} Admittedly, this figure is likely an overestimate given that the informing studies are based on cases at all severity levels instead of specifically mild cases and that all reported cases were ultimately treated with HBO, suggesting a probable bias toward more severe cases. Nevertheless, with the limited data available, there is little confidence to imagine that this rate of deterioration would meet the NASA standard of "preventing" serious Type II DCS "within 95% statistical confidence."⁵

The case reports provide further insight. Both the case of shoulder and neck sensory changes after glossopharyngeal insufflation and the case of progressively worsening left hand numbness and weakness would technically have fallen outside this study's proposed definition of mild neurologic DCI due to the presence of objective sensory deficits, but at least the latter would have still been considered Type I by NASA's current DCS classification paradigm given the ultimate diagnosis of peripheral neuropathy.^{12,15} While the cerebral AGE symptoms resolved spontaneously, showing that mild findings may sometimes improve without HBO treatment, the initially benign presentation of hand numbness deteriorated into a functionally impairing condition, one that required multiple HBO treatments to resolve. On the lunar surface, without the capability for HBO treatment, the prospect of such a seemingly mild case of PNS DCS turning into a mission-threatening or permanently disabling injury would support a posture that seeks to greatly reduce the risk of this outcome.

Similarly, while the case of the finger numbness after cabin depressurization resolved with GLO treatment, GLO treatment alone did not resolve the CNS findings in the patient experiencing lower-extremity pain after the altitude chamber run.^{16,17} In fact, the CNS findings, which were unrecognized by the patient, persisted for weeks. In a lunar exploration setting, it is unlikely that an astronaut would be able to detect such subtle neurologic findings, and with repeated EVA exposures the severity of the CNS injury would be likely to increase. It appears that asymptomatic neurologic findings are not guaranteed to resolve with GLO treatment.

Regarding how commonly neurologic DCI might present more mildly, approximately 28.8-50.2% of spinal cord DCS cases may exhibit presentations that could be considered mild according to this study's definition.^{14,18} However, because this number excludes PNS and cerebral cases, the true figure may be significantly different. While conventional wisdom would consider cerebral cases generally more severe and PNS cases generally milder, the case of the functionally impairing brachial plexopathy or ulnar neuropathy would suggest that even cases restricted to the PNS may not fall within this study's concept of mild neurologic DCI (Figure 3).¹⁵ Whether initially mild manifestations are more or less common than the above estimate, however, is unlikely to be highly relevant for NASA because of how commonly mild presentations of neurologic DCI may deteriorate into more severe cases.

Ultimately, the above data can be applied to the lunar exploration context to answer the following operational questions.

Should development of xEVA pre-breathe protocols consider mild neurologic DCI a separate category from type I and type II DCS and allow for the occurrence of mild neurologic DCI on xEVA?

The findings discussed do not provide enough confidence that mild neurologic DCI symptoms could be used to rule out progression to functional impairment in the setting of lunar exploration missions.

Without a robust predictive model for deterioration, all neurologic DCI likely requires prompt HBO treatment regardless of initial presentation severity. NASA should remain vigilant in their approach and strategize how to reduce the risk and impacts of even initially mild cases of neurologic DCI, including developing exploration EVA protocols with low risk of neurologic DCI of any initial severity level.

However, given the potential for severe outcomes of PNS DCS, which NASA currently defines as Type I, it is advisable that NASA revise its DCS classification system to reflect an understanding of how specific presenting symptoms correlate with outcomes.³

Should crewmembers who have experienced mild neurologic DCI after xEVA be treated as Type I DCS (i.e., automatically allowed to return to duty 24 hours after symptom resolution) or as Type II DCS (i.e., medical review and waiver from NASA's AMB required prior to return to alternobaric duty) with regards to disposition?

The data suggest that GLO, and even HBO treatment, has not always proven adequate to resolve mild neurologic deficits of DCI.^{15,16} Case reports have demonstrated that those whose symptoms have resolved may have persistent objective findings that are unrecognized by the patient.¹⁶ Therefore, it would be unadvisable to allow crewmembers experiencing mild neurologic DCI to participate in repeat xEVA as a default disposition, such as for "Mild DCS – Type I, Uncomplicated," even if their symptoms were to appear to resolve with GLO treatment.³ Rather, the disposition of all crewmembers who have experienced neurologic DCI should be the same as that for "Serious DCS Type II," where return to alternobaric duties is contingent on AMB review and waiver.³

In summary, the available evidence suggests that mild neurologic DCI may generally exhibit a more benign clinical course, yet there remains a significant proportion of mild neurologic DCI cases that deteriorate in the absence of HBO treatment. At this point, such findings warrant continued caution on NASA's part in preventing even mild neurologic DCI. However, future research can utilize this study's proposed definition of mild neurologic DCI and attempt to obtain a more definitive understanding of the natural history of this potential clinical entity (Figure 3). If more detailed models can be developed to predict outcomes of neurologic DCI based on initial presenting findings, they could potentially be used to further inform NASA's strategy.

Literature Cited:

1. Vann RD, Denoble PJ, Howle LE, Weber PW, Freiburger JJ, Pieper, Carl F. Resolution and severity in decompression illness. *Aviat Space Environ Med.* 2009 May;80(5):466–471.
2. U.S. Navy Diving Manual. Commander, Naval Sea Systems Command; 2018.
3. Human Health and Performance Directorate. Decompression sickness manual. 2022.
4. Conkin J, Pollock NW, Natoli MJ, Martina SD, Iii JHW, Gernhardt ML. Venous gas emboli and ambulation at 4.3 psia. *Aerospace Medicine and Human Performance.* Aerospace Medical Association; NaN/NaN/NaN;88(4):370–376.
5. Office of the Chief Health and Medical Officer. NASA Spaceflight Human-System Standard, Volume 2: Human Factors, Habitability, and Environmental Health [Internet]. 2023. Available from: <https://standards.nasa.gov/sites/default/files/standards/NASA/D//nasa-std-3001-vol-2-rev-d-signature.pdf>
6. Google Earth [Internet]. [cited 2025 Apr 11]. Available from: <https://earth.google.com/>
7. Koutroulou I, Tsigoulis G, Tsalikakis D, Karacostas D, Grigoriadis N, Karapanayiotides T. Epidemiology of patent foramen ovale in general population and in stroke patients: a narrative review. *Front Neurol.* 2020 Apr 28;11:281. PMID: PMC7198765
8. Boussuges A, Thirion X, Blanc P, Molenat F, Sainty JM. Neurologic decompression illness: a gravity score. *Undersea Hyperb Med.* 1996 Sep 1;23(3):151–155. PMID: 8931282
9. Dick APK, Massey EW. Neurologic presentation of decompression sickness and air embolism in sport divers. *Neurology.* 1985 May;35(5):667–671.
10. Mitchell S. Severity scoring in decompression illness. *SPUMS J.* 2005 Dec;35(4):199–205.

11. Monnot D, Michot T, Dugrenot E, Guerrero F, Lafère P. A survey of scuba diving-related injuries and outcomes among French recreational divers. *Diving Hyperb Med*. 2019 Jun 30;49(2):96–106. PMID: PMC6704004
12. Linér MH, Andersson JPA. Suspected arterial gas embolism after glossopharyngeal insufflation in a breath-hold diver. *Aviat Space Environ Med*. 2010 Jan;81(1):74–76. PMID: 20058741
13. Pitkin AD, Benton PJ, Broome JR. Outcome after treatment of neurological decompression illness is predicted by a published clinical scoring system. *Aviat Space Environ Med*. 1999 May;70(5):517–521.
14. Blatteau JE, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, Cochard G, Arvieux J, Henckes A, Lafere P, Germonpre P, Lapoussiere JM, Hugon M, Constantin P, Barthelemy A. Prognostic factors of spinal cord decompression sickness in recreational diving: retrospective and multicentric analysis of 279 cases. *Neurocrit Care*. 2011 Aug 1;15(1):120–127.
15. Butler FK, Pinto CV. Progressive ulnar palsy as a late complication of decompression sickness. *Annals of Emergency Medicine*. 1986 Jun;15(6):738–741.
16. Dully FE. Central nervous system involvement following Type I aviator's bends complicated by complacency. *Aviat Space Environ Med*. 1975 Sep;46(9):1186–1187.
17. Johnston MJ. Loss of cabin pressure in a military transport: a mass casualty with decompression illnesses. *Aviat Space Environ Med*. 2008 Apr;79(4):429–432. PMID: 18457302
18. Blatteau JE, Gempp E, Constantin P, Louge P. Risk factors and clinical outcome in military divers with neurological decompression sickness: influence of time to recompression. *Diving Hyperb Med*. 2011 Sep;41(3):129–134.