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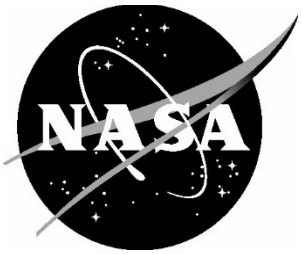

Assessment of Oxygen Toxicity and Neuro-vestibular Disturbances during Neutral Buoyancy Laboratory (NBL) Exploration Spacesuit Testing and Review of *Requirements Applicable to Personnel Participating in Diving, Hyper/Hypobaric Chambers, and Pressurized Suit Operations* (JPR 1830.6)

Compiled by:

Kristin M. Coffey, Office of the Chief Health and Medical Officer, KBR, Houston, Texas

Doug J. Ebert, Office of the Chief Health and Medical Officer, KBR, Houston, Texas

Joanne L. Kaouk, Office of the Chief Health and Medical Officer, UHCL, Houston, Texas

Kim Lowe, Office of the Chief Health and Medical Officer, Aegis Aerospace, Houston, Texas

Sarah D. Taoufik, Office of the Chief Health and Medical Officer, JES Tech, Houston, Texas

David R. Francisco, Office of the Chief Health and Medical Officer, NASA, Houston, Texas

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Introduction

NASA's Office of the Chief Health and Medical Officer (OCHMO) initiated a working group to investigate operations in the Neutral Buoyancy Laboratory (NBL) and determine the possible causes of adverse physiological effects that have been experienced by subjects operating exploration spacesuits during 1/6G simulations in the NBL training platform.
Note: The content discussed in this report, including the test parameters, symptomology, and recommendations are applicable only to the NBL and are not transferrable to spaceflight operations.

NASA Presenters

Dave Francisco – Technical Fellow for Human Spaceflight Standards, *Office of the Chief Health & Medical Officer*

Dr. Tyson Brunstetter – SANS Clinical Lead (Eyes/Vision), *Space Medicine Operations Division*

Zachary Fester – Space Suit Engineer, Advanced Suit Team

Jason Norcross – Senior Scientist, *Biomedical Research and Environmental Sciences*

Dr. Kristi Ray – Operational Flight Surgeon, *EVA and Human Surface Mobility Program*

Dr. Bob Sanders – Flight Surgeon & Program Medical Officer, *EVA and Human Surface Mobility Program*

Invited Reviewers

Dr. Jay Dean – Professor, *University of South Florida Morsani College of Medicine*

Dr. Bruce Derrick – Assistant Professor & Hyperbaric Medicine Specialist, *Duke University*

Dr. Frederik Kragerud Goplen – Professor & ENT Surgeon, *University of Bergen (Norway)*

Dr. Richard Moon – Professor and Medical Director, *Duke University Hyperbaric Center*

Dr. Neal Pollock – Research Chair in Hyperbaric and Diving Medicine, *Université Lavel (Quebec, Canada)*

Dr. Jason Porter – Associate Professor, *University of Houston College of Optometry*

Dr. Alexander Schill – Senior Research Scientist, *University of Houston College of Optometry*

Dr. Barbara Shykoff – Senior Research Scientist, *Naval Medical Research Unit Dayton*

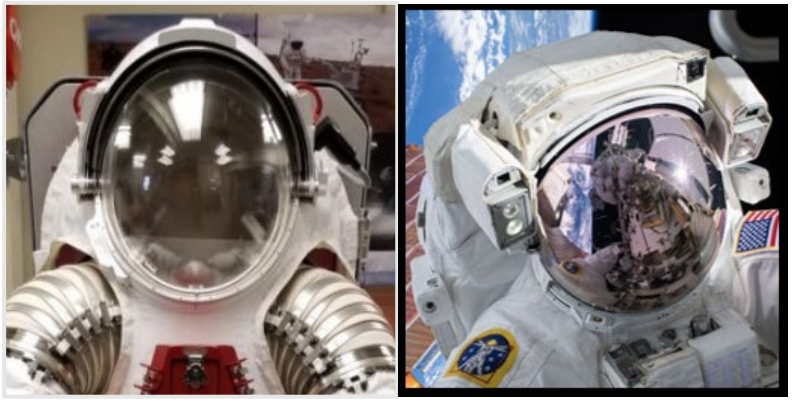
Dr. Alan Wyatt – Medical Co-Director, *Louisiana State University LCMC Hyperbaric Medicine Unit*

Contents

1 Background

1.1 NASA Suit Design and Elevated Suit Pressure Testing

The Exploration Extravehicular Mobility Unit (xEMU) is a dramatically different design than the International Space Station Extravehicular Mobility Unit (ISS EMU) with which Neutral Buoyancy Laboratory (NBL) subjects have much more experience. Of particular note, the visor has an elongated sphere curvature that is designed to facilitate a vertical field of view for surface operations compared to the spherical visor of the ISS EMU that is used for microgravity operations. The xEMU visor does not have a uniform curvature and produces visual distortion which is accentuated in the water environment of the NBL as opposed to the air or vacuum environment that it is designed for (see Section 2.3 Neuro-vestibular Disturbances for additional information).



xEMU helmet design (left) compared to EMU helmet design (right). Credit: NASA

Due to the need to test the new xEMU suit for future lunar surface operations, these sessions were performed at a depth of ~40-feet (bottom of the pool) compared to previous runs in the ISS EMU at ~16-feet (neutrally buoyant in the middle of the pool). The NBL runs for both the EMU and xEMU suit use a 46% O_2 nitrox with previous runs including 4.3 psid (pounds per square inch differential). The subject therefore had lunar weighouts instead of neutral buoyancy that is needed for simulated microgravity operations. There have been less than 29 individual subjects who had an NBL run in the xEMU suit with 20 runs occurring at 1/6g at ~40-feet depth and either 4.3 or 6.2 psid. Over the course of approximately 10 months, the individual who experienced the adverse effects had participated in three runs in the xEMU suit.

1.2 Summary of Symptomology from Mishap Report

Two subjects in the xEMU were both weighted to simulate 1/6g at an elevated suit pressure of 6.2 psid while on 46% O_2 nitrox at 40-foot depth which resulted in an oxygen exposure of approximately PO_2 (partial pressure of oxygen) 1.20 ATM (Atmospheres). This was the third run for one of the subjects in the xEMU suit and second run at these particular atmospheric parameters. The weighout of both subjects was prolonged (approximately 147 minutes), which

resulted in the affected subject of this report to have significant idle time (i.e., kneeling in static postures, standing forward lean). It was during this time that the subject reported some "stomach awareness" that did not improve. After approximately 30 minutes during this run, the subject reported to the medical officer (MO), while on a private loop, that they were experiencing some continued adverse symptoms and general dysphoria after moving from kneeling to a standing position. These issues did not resolve after increased activity and the subject reverted to a low activity state and also continued to have nausea. The second subject was not reporting any adverse issues at that time, however a decision was made to end the run for both subjects. There was a delay getting the affected subject out of the pool due to issues mating the suit with the donning stand, which added approximately 6.5 minutes to the anticipated egress operations. It was also noted that during ascent, the subject's symptoms worsened. During the process of extraction from the pool the subject appeared to have a syncopal event that lasted approximately 30 seconds. This event was not initially recognized by the divers while they were working to latch the subject to the donning stand, but was later noted upon video review. The presyncopal and syncopal events were not initially recognized because subjects typically will close their eyes when in discomfort, which was apparent to the NBL team. Once on the pool deck, there was a lack of improvement in symptoms and the MO determined that the subject was not able to egress nominally from the rear-access of the suit. Therefore, an assisted waist egress was performed and the subject was taken to the medical bay for evaluation and treatment. Upon suit egress, the subject was presyncopal with signs of nausea, paleness, diaphoresis, and weak radial pulse. Testing in the medical bay indicated normal ECG results and an abnormally low end tidal CO_2 of 20 mmHg, rising to 34 mmHg at 45 minutes post suit extraction. The subject appeared to have fully recovered after approximately one-hour following egress with no further reports of adverse symptoms.

Upon retrospective review, the subject's first run in the xEMU was noted to be at the lower 4.3 psid with an approximate PO_2 1.14 ATM. The subject had similar symptoms reported during that run (see Table 1 – Observations, Symptoms and Medical Data*). Information gathered from other subjects on their previous runs in the xEMU suit did confirm that there were previous reports of stomach awareness or nausea, but no documented vomiting. When this did occur, the subjects noted it was during tasks with and without provocative movements.

Table 1 – Observations, Symptoms and Medical Data*

Run #1 1/6g at 4.3 PSID	Run #2 1/6g at 6.2 PSID	Run #3 1/6g at 6.2 PSID
← less than 1-year →		
Weighout time: ~259 min** **Total time at depth: ~180 min** **Total pressurized time: ~346 min**	**Weighout time**: ~109 min** **Total time at depth: ~290 min** **Total pressurized time: ~379 min**	**Weighout time**: ~147 min** **Total time at depth: ~170 min** **Total pressurized time: ~218 min**
Corrective lens worn xEMU integrated comm system	Corrective lens worn CCA (Snoopy cap)	Corrective lens worn In-ear comms
Suit fit issue/crouch pain	Higher activity (shorter weighout duration), completing test objectives	Low activity (weighout duration), low met rate
Low activity (weighout duration), low met rate	Supine approx. 16 minutes during second subject's weighout	Excessive yawning
Some yawning	No symptoms reported	Hunched posture in suit that persisted throughout run
Reported feeling warm (confirmed not cooling flow or metabolic)		Neck fatigue
Dysphoria		Dysphoria
Stomach aware *(bottom of the pool)*		Stomach aware *(bottom of the pool)*
Leg shaking (twitching) *(bottom of the pool)*		Moving slowly in suit
Felt worse on ascent		Felt worse on ascent
Dry heaves observed		Twitching (few seconds) [possible myoclonic jerking/spasms] with paresthesia in the fingers while in donning stand being pulled out of water
Tingling in arms (exact time unknown, while in donning stand)		Weak radial pulse post run on pool deck while helmet off, likely brief syncopal event
Absent radial pulse post run on pool deck		Patient pale
Patient pale		Slow to respond verbally
Slow to respond verbally		Confusion
Nauseous		Nauseous
Diaphoretic (Sweaty)		Diaphoretic (Sweaty)
		Measured low CO_2 approx. ~10 minutes after helmet off

**Blue items are medical symptoms consistent between Run #1 and Run #3*
***Weighout time = elapsed time from when subject touched the pool bottom until weights were configured for 1/6 g simulation*

NBL Subject Test Runs Comparison

One subject – multiple runs over less than 1 year period

All runs with nitrox 46% @ 40 FFW

Run # 1
Full run minus 5 minutes due to stomach awareness

xEMU @ 4.3 psi
346 minutes total Pressurized time
259 minutes of weigh out time (up and down to floor during weigh out)
~173 Minutes at PO_2 of 1.14 ATM

~650 BTU/hr
Lower than typical

Symptoms Observed
- Some yawning
- Reported feeling warm
- Stomach aware
- Felt worse on ascent
- Dry heaves observed
- Absent radial pulse post run on pool deck
- Patient pale
- Slow to respond verbally
- Nauseous
- Diaphoretic (Sweaty)

Run #2
No symptoms reported – Higher activity

xEMU @ 6.2 psi
379 minutes total Pressurized time
109 minutes of weigh out time
~260 Minutes at PO_2 of 1.2 ATM

~1000 BTU/Hr
Typical NBL run

~15-minutes (estimated) on back during weigh out of other subject
< 400 BTU/hr

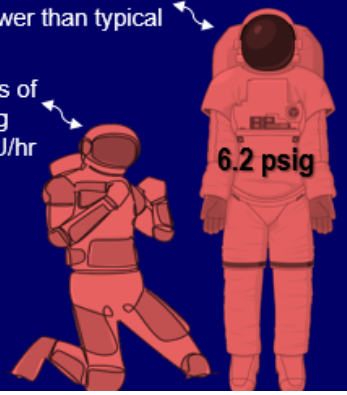
40 ffw

Run #3
Run terminated early due to not feeling well (after trying 10 minutes of increased metabolic rate)

xEMU @ 6.2 psi
218 minutes total Pressurized time
147 minutes of weigh out time
~175 Minutes at PO_2 of 1.2 ATM

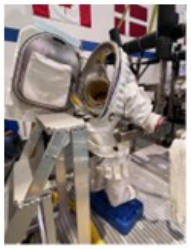
~650 BTU/hr
Lower than typical

15-minutes of kneeling
< 400 BTU/hr



Symptoms Observed
- Excessive yawning
- Stomach aware
- Moving slow in suit
- Felt worse on ascent
- Twitching while in donning stand being pulled out of water
- Weak radial pulse post run on pool deck while helmet off
- Patient pale
- Slow to respond verbally
- Nauseous
- Diaphoretic (Sweaty)

Timeline of Mishap Events

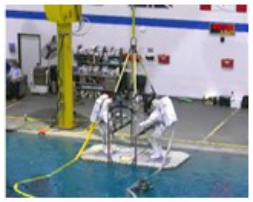


8:46 CDT
The subjects ingress suits and pressurization began

9:14 CDT
Target pressure reached

10:01 CDT
The subjects feet touch water

10:07 CDT
The subjects arrive at the bottom of the pool. Start of 0-g weigh outs



10:29 CDT
Completion of 0-g weigh outs Start of 1/6-g weight out on PGWS

11:30 CDT
Test team failed attempts to fit *affected* subject into the PGWS. Shifted their efforts to weigh out *unaffected* subject. *Affected* subject remained idle

11:45 CDT
Test team failed at getting the *unaffected* subject onto the PGWS and shifted their attention to getting the *affected* subject onto the PGWS

11:55 CDT
Test team failed attempts to fit subject into the PGWS. Shifted their efforts to weigh out *unaffected* subject. *Affected* subject remained mostly immobile

12:00 CDT
Affected subject remained mostly immobile in a kneeling position for about 15-20 minutes. The subject was yawning repeatedly, and the subject closed their eyes for about a minute

12:14 CDT
The test conductor announced the end of attempts to use the PGWS on the *unaffected* subject. The *affected* subject stood up from the kneeling position and immediately felt stomach awareness. Subject was more mobile with movements between standing to kneeling and picked up items from the ground

12:44 CDT
The subject walked into a simulated airlock and was standing for a few moments until requesting a PMC to share the symptoms. The subject wanted to emphasize feeling well enough to continue the run. The TD advised greater level of activity and reassess in 15 minutes

~13:05 CDT
Follow up PMC where the subject reported 'no worse, no better'. It was decided to end the run

13:07 CDT
TD announced to pull the subject. Upon ascent the subject experienced myoclonic spasms (<3s), syncope (<10s), paresthesia in both upper extremities (fingers)

13:19 CDT
Subject was on the pool deck and was experiencing confusion and syncope. The total time the subject was experiencing symptoms was 52 minutes

13:32 CDT
Subject was removed from the suit through waist extraction, placed on gurney and taken to medical

13:34 CDT
Subject arrived at medical

13:39 CDT
$EtCO_2$ measured at 20 mmHg

14:27 CDT
Recovered to baseline $EtCO_2$

1.3 Working Group Charter

Pre-defined goals of the working group included:

Goal 1: Assess the risks and provide potential options to mitigate the occurrence of oxygen toxicity to participants of exploration spacesuit testing in the NBL.

a) Identify the risks associated with temperature, pressure, depth, and activity level to reduce the risk of oxygen toxicity.

Goal 2: Assess and provide recommendations to mitigate neuro-vestibular issues experienced due to the helmet design of the exploration spacesuit combined with the submerged environment.

Goal 3: Assess and provide recommendations on procedures for monitoring biomedical data during a test run at the NBL.

a) Identify external team members responsible for crew monitoring, always ensure visual observation, establish communication protocol to monitor crew medical condition.

Goal 4: Potential testing that can be conducted beforehand to identify individuals who may be at elevated risk of experiencing symptomology related to neuro-vestibular issues.

Goal 5: Determine the level of risk acceptance for guidelines to diving and pressurized suit operations at the NBL; refer to and review JPR 1830.6.

a) Are NOAA guidelines too conservative? Are U.S. Navy guidelines applicable?
b) Are NBL test subjects considered diving at ‘wet’ or ‘dry’?
c) Guidance regarding limits of DCS risk vs. CNS oxygen toxicity risk

2 Overview of Primary Proximate Causes

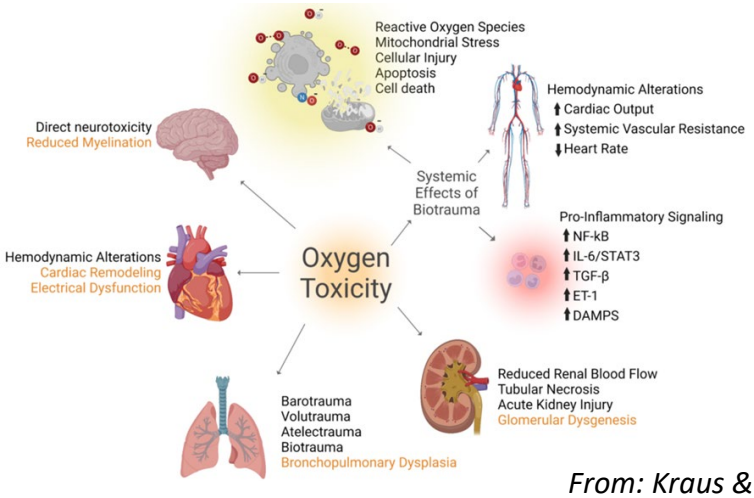
The following sections provide background information on the three identified proximate causes of the symptomology experienced by the subject participating in xEMU elevated suit pressure operations at the NBL, as described above. Other potential causes that were included in the investigation of the mishap but were ruled out as unlikely included suit sized improperly, suit pressure leading to discomfort, contaminated breathing gas, and decompression sickness.

2.1 Oxygen Toxicity

Oxygen toxicity is a condition resulting from the effects of breathing too much supplemental oxygen (O_2) or breathing O_2 at increased partial pressure. Oxygen toxicity is a risk in diving operations, including those used at NASA, when divers breathe O_2 at sufficiently elevated partial pressure. Partial pressure is increased by increased O_2 fraction (concentration) in the breathing gas, increased total pressure, or both. This excess supply of O_2 to body organs and tissues, if severe enough, can have harmful effects on the body. The extent of the toxicity is dependent upon both the O_2 partial pressure and the exposure time. The higher the partial pressure and the longer the exposure, the more severe the toxicity. It can present as central nervous system (CNS), eyes, or pulmonary toxicity and range from mild symptoms to loss of consciousness and seizure (Shykoff, 2013; Cooper, Phuyal, & Shah, 2023; Arieli et al., 2003). Pulmonary oxygen toxicity does not occur if O_2 partial pressure is 0.5 ATM or less. CNS oxygen toxicity very rarely occurs if O_2 partial pressure is less than 1.6 ATM, and is much less likely in dry than in submerged divers. Oxygen toxicity is of special concern to NASA when, through use of diving as an analog to spaceflight, subjects spend a significant amount of time training underwater in the NBL exposed to a variety of conditions that could make them vulnerable.

Pathophysiology

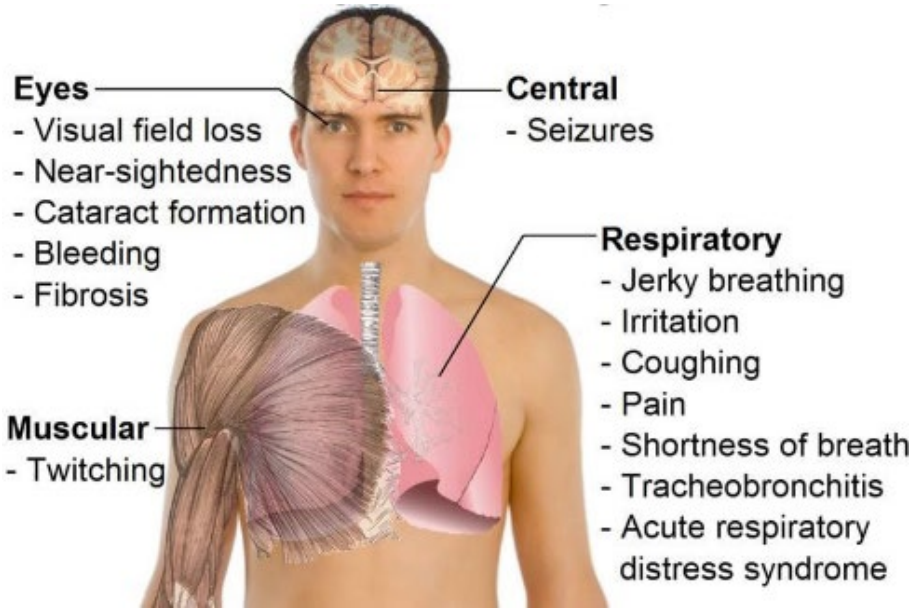
The cause of oxygen toxicity is not certain, however the leading theory is that increased O_2 concentration or partial pressure increases the levels of reactive oxygen species (ROS), and production of oxygen free radicals. These free radicals can disrupt the balance of oxidants and antioxidants in the body, potentially leading to cell and tissue damage affecting lipid membranes, proteins, nucleic acids, and pulmonary endothelial and alveolar cells, all potentially contributing to pulmonary or CNS symptomology (Cooper, Phuyal, & Shah, 2023; Arieli et al., 2003, Jing et al., 2024).



From: Kraus & De Miguel (2022)

Symptoms of Pulmonary & CNS Oxygen Toxicity

- Limb convulsions
- Hyperventilation
- Headache
- Visual disturbances
- Pallor
- Anxiety/Irritability
- Confusion
- Dizziness
- Muscle twitching
- Hearing disturbances
- Nausea
- Cough
- Weakness
- Choking sensation
- Non-cold shivering
- Attention loss
- Warm sensation in the face
- Convulsions



From: Diver's Alert Network (DAN) Dr. E.D. Thalmann

2.1.1 Pulmonary Oxygen Toxicity

Pulmonary toxicity is less of a concern for NASA divers than CNS toxicity as it is more often associated with longer duration oxygen exposure. "Pulmonary toxicity risk increases with exposure time, underwater exercise, and repeated exposure on same or consecutive days. Symptoms may have lag after exposure and include chest tightness, cough, discomfort, inflamed tracheal bronchial tree, and measurable pulmonary function loss. Pulmonary O_2 toxicity is not usually associated with serious health concerns but most commonly inflammation and discomfort" (Shykoff, 2025).

2.1.2 CNS Oxygen Toxicity

Brain and CNS decline caused by oxygen toxicity was first described by Paul Bert in 1878, named the Paul Bert effect. Bert, through dry dive experiments, showed that O_2 at high levels (PO_2 15-20 ATM) could lead to CNS toxicity and caused convulsions in many organic species and small mammals (Chawla & Lavania, 2011). Clinical signs of CNS toxicity may include visual changes

such as tunnel vision, tinnitus, nausea, facial twitching, dizziness, and confusion (Thompson & Paton, 2014). The time for the appearance of symptoms is inversely related to the oxygen pressure and may be as short as 10 minutes at pressures of PO_2 4-5 ATM (Luis & Syafaah, 2022). This may be followed by tonic colonic seizures and subsequent unconsciousness (Jing et al., 2024).

The U.S. Navy established limits for O_2 rebreather diving based on risks of CNS oxygen toxicity by 1986. However, documented human exposures to elevated O_2 partial pressures in the water continued. Decompression tables were developed for rebreather dives at O_2 partial pressures that were considered safe from CNS effects. Studies were conducted to assess pulmonary effects of long in-water exposures. Further, in-water rebreather training dives continued. The accumulated data indicate that diving below PO_2 1.6 ATM is not without risk of oxygen toxicity, but symptoms at these levels are rare and may have been caused by other contributing factors (Shykoff, 2025). The United States Navy diving manual also states CNS oxygen toxicity is usually not encountered unless the partial pressure of oxygen approaches or exceeds 1.6 ATM.

2.1.3 Contributing Factors to Oxygen Toxicity

Shykoff suggested that additional explanatory variables beyond PO_2 (below) must also be considered when looking at oxygen toxicity-like symptoms. One of the biggest challenges in identifying and predicting oxygen toxicity is that in addition to O_2 partial pressure and exposure time, multiple known risk factors can affect oxygen toxicity risk, and it can be difficult to identify true cause. Partial pressure of O_2 as well as exposure time are the commonly considered factors, but additional factors can also play an important part in CNS oxygen toxicity risk (Shykoff, 2013; U.S. Navy Diving Manual Revision 7, 2016), including:

- Increased metabolic rate, level of exercise/activity
- Increased sympathetic nervous system activity
- Hypercapnia (cerebral blood flow increases)
- Increased breathing resistance
- Individual susceptibility*
- Temperature — apparent incidence is higher in cold water; exposure to cold water causes peripheral vasoconstriction and increased O_2 delivery to the core, contributing to CNS oxygen toxicity symptoms
- Darkness of the environment
- The use of medications including those with vasodilatory effects (sildenafil/tadalafil) and those that can lower the seizure threshold (i.e., mefloquine, buproprion, etc.)
- Increased depth (gas density) — increased depth/density increases changes in pressure and resistance, contributing to oxygen toxicity

* Further investigation into the feasibility of individual CNS O_2 sensitivity testing found that various statistical analyses conducted by the U.S. Navy could not identify a reliable measurement of personal sensitivity, with variability of symptomology within the individual as well as many factors other than individual sensitivity contributing to the occurrence of O_2 toxicity, and the U.S. Navy has since abandoned oxygen tolerance testing (Harabin 1994; Walters et al., 2000). In sum, it is not currently recommended that the NBL pursue O_2 sensitivity testing for current operations.

Considering that the suited NBL subject in question was at a PO_2 1.19 ATM, the low incidence of oxygen toxicity at PO_2 <1.6 ATM, and the lack of additional oxygen toxicity contributory factors, these data do not support oxygen toxicity as the probable reason for the symptoms in the aforementioned case.

2.1.4 Safe Oxygen Partial Pressures

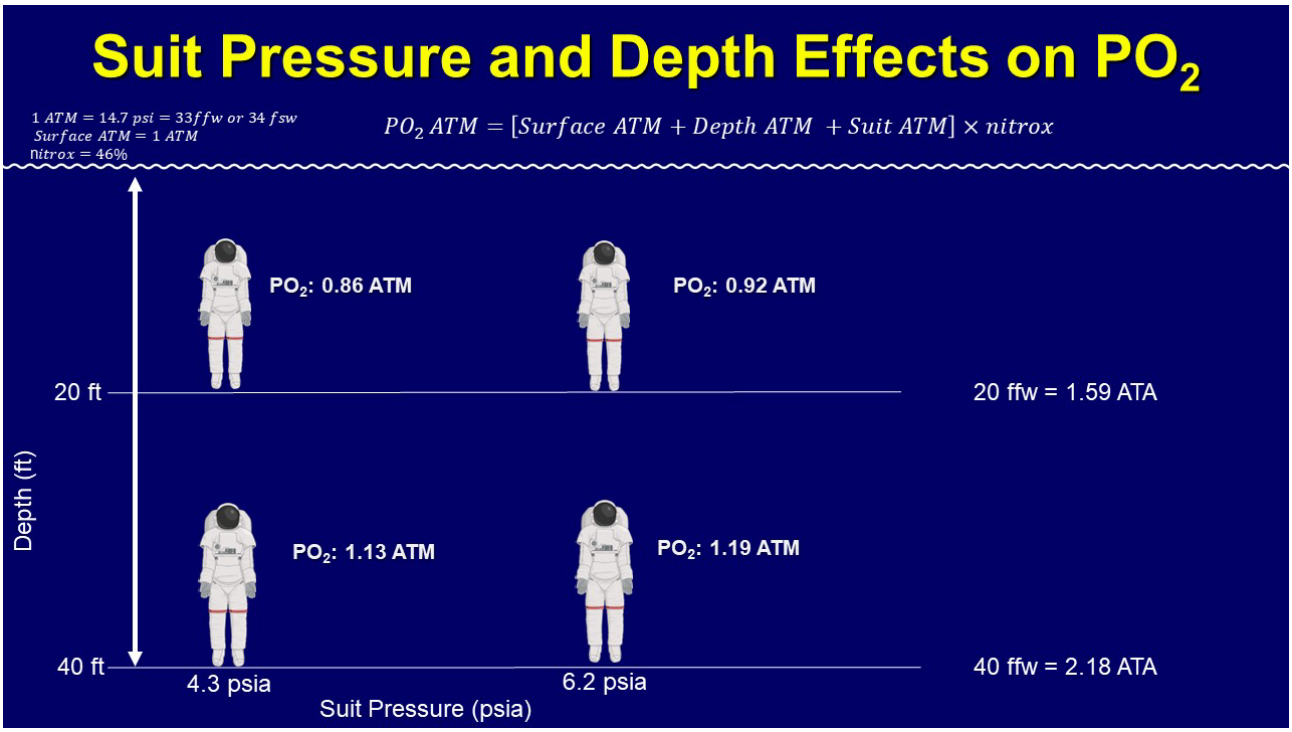
The partial pressure of oxygen drives oxygen transfer to and from blood and tissue, and determines its chemical activity in the body. As described by Dalton's Law, the partial pressure of oxygen in a gas is the product of the total pressure and the oxygen fraction in the gas. Thus, if a diver breathes a gas mix with constant oxygen fraction while descending, the inspired partial pressure increases.

At sea level, the pressure exerted by a column of air is 14.7 pounds per square inch (psi) and is equal to 1 ATM. For a diver, the water column exerts additional pressure, with 33 feet of sea water or 34 feet of fresh water adding an additional 14.7 psi relative to sea level for a total pressure of 2 ATM. If the diver wears a pressurized suit, total pressure in the inspired gas consists of the absolute pressure outside the suit plus the pressure differential, suit to water. An astronaut at the bottom of the NBL tank is exposed to a total pressure of atmospheric pressure + water column pressure + suit differential pressure.

The inspired O_2 partial pressure is the total pressure multiplied by the fraction of O_2 in the supplied gas. Atmospheric air contains 21% oxygen and 78% nitrogen (N_2), but nitrox mixtures are blended to reduce the risk of decompression sickness by providing proportionately less N_2 and more O_2. The O_2 partial pressure for an astronaut in the NBL is thus determined by depth in the tank, suit differential pressure, and nitrox mix (Shykoff, 2025).

2.1.5 Suit Pressure and Depth Effects on PO_2

Partial pressure of oxygen is dependent on the percent oxygen (nitrox mix), the dive depths, and the differential suit pressure. In the case of NBL dives, the depth of the dive will have more effect on PO_2 than suit pressure since suit pressure changes are a small fraction of pressure changes due to depth.



The National Oceanic and Atmospheric Administration (NOAA) set limits to manage the combined risks of CNS and pulmonary oxygen toxicity. The NOAA standard is a maximum PO_2 1.40 ATM for open circuit diving and PO_2 1.3 ATM for closed circuit diving (NOAA Diving Standards & Safety Manual, 2023). NOAA limits are not empirical, but are based on REPEX (Hamilton) and UPTD (Lamberstsen) models.

Table 2 – Oxygen Partial Pressure and Normal Exposure Time Limits for Working Dives from NOAA 2001 Diving Manual

Oxygen Partial Pressure Bar or ATM	Maximum Duration (mins) Single Dive	Maximum Total Duration (mins) for 24-hour Period
1.6	45	150
1.5	120	180
1.4	150	180
1.3	180	210
1.2	210	240
1.1	240	270
1.0	300	300
0.9	360	360
0.8	450	450
0.7	570	570
0.6	720	720

The United States Navy uses PO_2 1.3 ATM as the control point for O_2 partial pressure in its mixed gas closed-circuit rebreathers and emphasizes that very long exposures may increase diver risk for lung toxicity symptoms. Above PO_2 1.6 ATM is considered O_2 partial pressure above 1.6 ATM is considered to increase the risk of CNS oxygen toxicity above the negligible

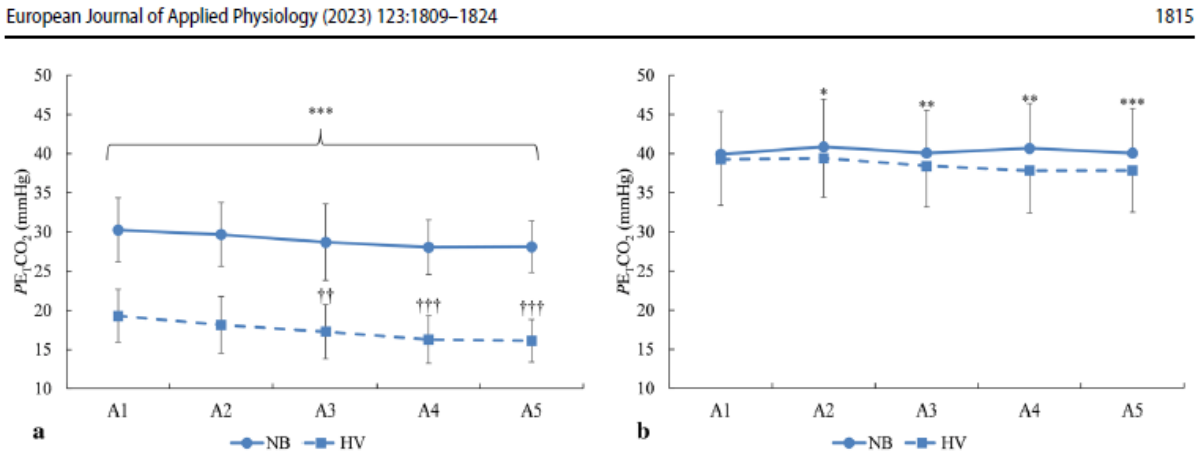
level (U.S. Navy Diving Manual Section 10-3 Revision 7, 2016). *Note that the Navy Dive Manual uses Bar or ATA nomenclature, but these are numerically equivalent to ATM.*

2.2 Hypocapnia and Metabolic Load

Although uncommon, hypocapnia (an arterial CO_2 partial pressure significantly below normal) is a possible condition experienced by the subject during the symptomatic dive at the NBL. While hypercapnia (an increased arterial CO_2 partial pressure) is where most suited spaceflight concerns lie due to limitations in CO_2 removal, the efficient CO_2 washout of the NBL's exploration extravehicular mobility unit (xEMU) system, combined with the subject's significantly low average metabolic rate of 668 BTU/hr in that run led the expert panel to rule out hypercapnia as the cause of the symptomology.

Hypocapnia is a condition described as blood and alveolar partial pressure decreasing below the normal reference point of 35mmHg (Sharma & Hashmi, 2023). Hypocapnia includes a variety of symptoms such as anxiety, shortness of breath, syncope, seizures, secondary hypocalcemia, and confusion. At the conclusion of the NBL run, the subject had an end-tidal CO_2 ($EtCO_2$) of 20mmHg which falls significantly below the reference value. It is also important to note that due to suit constraints, real-time in-suit $EtCO_2$ cannot be measured. Since the $EtCO_2$ values were taken approximately 20 minutes after the conclusion of the run, their relevance is limited, but continues to be a point of concern.

Hyperventilation is a well-established cause of hypocapnia, and one study found that even 15 seconds of forced vigorous hyperventilation, not due to metabolic needs, brought the $EtCO_2$ down by 20mmHg (Pernett et al., 2023). Different factors in the NBL can lead to hyperventilation such as anxiety, motion sickness, stomach awareness, and nausea. In attempts to alleviate these symptoms, the subject in this case had altered breathing patterns which could have unintentionally caused hypocapnia. It is not clear however whether the subject altered breathing patterns to increase or decrease ventilation.



Effects of hyperventilation on oxygenation, apnea breaking points, diving response, and spleen contraction during serial static apneas (Pernett et al., 2023)

Iscoe and Fisher (2005) proposed that a person at rest in a hyperoxic condition experiences hyperventilation leading to hypocapnia (Iscoe and Fisher, 2005). This was explained by the Haldane effect which suggests that oxygen-saturated hemoglobin has a lower capacity for CO_2 resulting in a decrease in bicarbonate and dissolved CO_2 in the blood. The hypocapnia causes vasoconstriction which paradoxically reduced oxygen delivery in tissues despite the high arterial oxygen content further decreasing the drive for respiration. This idea was later countered by more compelling evidence by Forkner it al., 2007 which proposed that there are many flaws with that argument:

- Hyperoxia does not reduce blood flow enough to offset the higher oxygen content which causes an enhanced oxygen delivery
- Acidosis caused by CO_2 accumulation in the tissues would counteract the vasoconstriction
- The Haldane effect accounts for small changes in PCO_2 in normoxia and hyperoxia
- There is not compelling evidence to conclude that hyperventilation caused by hyperoxia leads to significant arterial hypocapnia
    - Many studies show that a high O_2 administration (87-100%) caused no significant change in the arterial PCO_2
    - Studies show that even 100% O_2 administration up to PO_2 3 ATM caused does not lead to arterial hypocapnia.

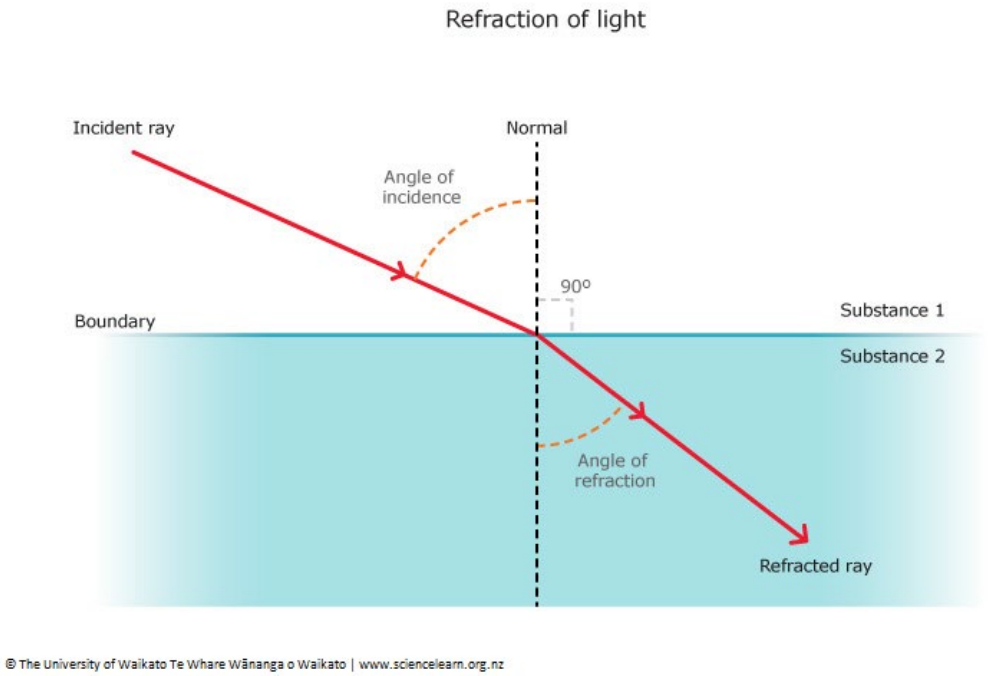
One method to prevent worsening hypocapnia is to raise the crewmember's metabolic rate. Metabolism is measured in British thermal units per hour (BTU/hr) and is an indication of the heat energy generated by the body per hour. As an individual's metabolic rate increases, the consequent CO_2 production increases which can in turn increase blood and alveolar pCO_2. During activity in an NBL run, the metabolic rate on average is about 1200 BTU/hr, but during the NBL mishap, the subject was idle resulting in a low metabolic rate that may have been too low to produce enough CO_2 to negate hypocapnia.

2.3 Neuro-vestibular Disturbances

Neuro-vestibular disturbances and associated physiological symptoms are fairly common among underwater divers. Vertigo, dizziness, nausea and vomiting are reported symptoms in divers attributed to various factors such as changes of pressure in the middle ear and audiovisual disturbances, including the perception of the body surroundings spinning and involuntary rhythmic movements of the eyes (Goplen et al., 2010; *Alternobaric Vertigo*, 2023).

Human vision underwater is altered in multiple ways, as described by Luria & Kinney (1975). Water transmits less total light energy than air due to scattering of the energy through particles suspended within water and absorption of energy by the water, both contributing to visual consequences. Additionally, light rays are refracted as they pass from water to air, which

displaces the image and creates distortions in size, distance, and direction of the observed object (*Refraction of light*, 2012).



From: *Refraction of light – Science Learning Hub New Zealand (2012)*

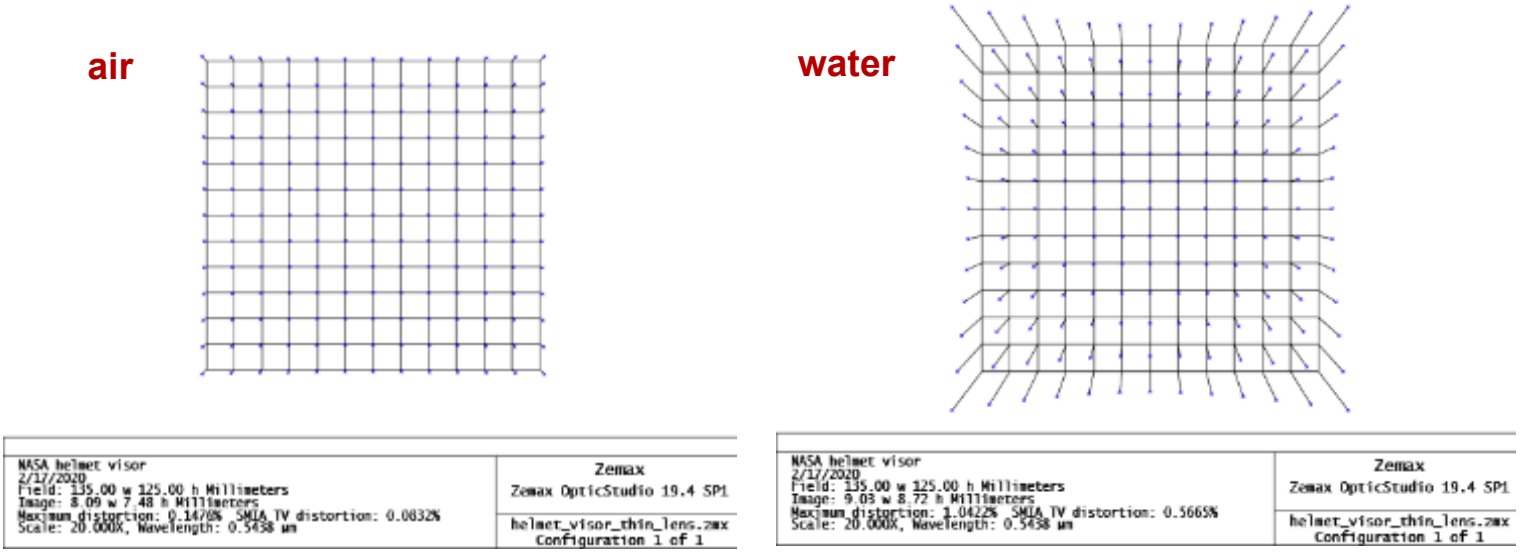
These changes in visual perceptions amongst divers may lead to symptoms similar to motion sickness, including stomach awareness, headache, and nausea/vomiting. The brain and inner ear receive the principal sensory signals that contribute to vestibular cues. When a person is stationary but the brain and inner ear sense motion from vestibular, visual, or somatosensory information, there is a conflict in the neuro-vestibular system that leads to symptoms of motion sickness. Similarly, if a visual system indicates movement but the vestibular system does not, visually induced motion sickness can occur (*Motion Sickness*, 2023).

2.3.1 Design of the EMU/xEMU Helmet

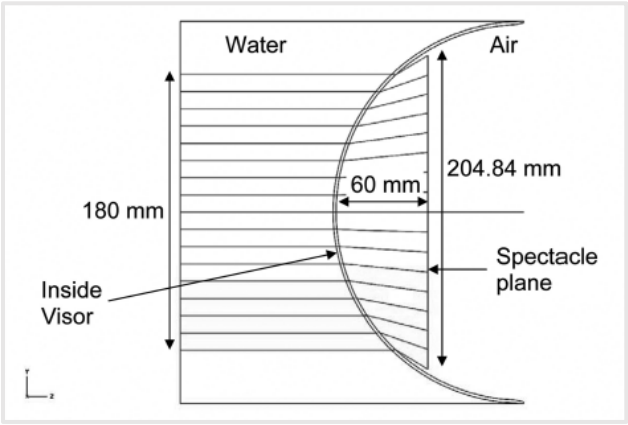
Commercial underwater diving helmets use flat glass, which minimizes the effects of underwater optical distortion caused by the refraction of light at the water/glass/air interface (Adolfson & Berghage, 1974).

Aerospace helmet visors are tested and used in air or in a vacuum, with the optical performance nearly identical between air and the vacuum of space (refractive index in vacuum = 1.00 vs. 1.0003 in air). However, the perceived optical qualities of helmet visors change dramatically when submerged in water, which has a refractive index of 1.33. This type of distortion experienced underwater does not occur in air or space, so the visual disturbances are only encountered in the suit while underwater.

ISS EMU suits used during NBL training have had a spherically curved helmet glass creating a uniform shift in refractive error at all viewing angles. The EMU visor's uniform curvature minimizes additional optical consequences of submersion (e.g., visual distortions), but the spherical helmet glass does induce a shift in refractive error underwater, which can be mostly counteracted by the use of prescription glasses at a specified diopter correction during NBL test runs (Porter, Gibson, & Strauss, 2013).



Square grid object Size: 13.5 cm wide x 13.5 cm tall, Distance from visor plane: 25 cm, Angular subtense: ~30 degrees x 30 degrees



Zemax model of the water/inside visor/air interfaces of the EMU helmet (wavelength = 550 nm). From: Porter, Gibson, and Strauss (2013)

The exploration spacesuit (xEMU) helmet design also utilizes curved glass but is curved elliptically, i.e., "football shaped", instead of spherically resulting in a completely different radius extending vertically versus horizontally. This design creates additional vertical field of view, which is important for future lunar surface operations. Like the ISS EMU visor, the submerged xEMU helmet visor induces a shift in refractive error which can be mostly mitigated

with prescription glasses. However, the elliptical shape of the xEMU visor induces a variable amount of visual distortions, depending on the angle of gaze through the visor. The distortion is least in the center field of view and increases away from the center of view, proportional to the arc of the ellipse. Therefore, submerged xEMU visors induce both static and dynamic optical distortions which cannot be mitigated by simple prescription glasses.

Modelled xEMU Visor Distortions. From Brunstetter et al., Vision/Optical Challenges of NBL Ops (2025)

Optical Consequence *when Submerged*	EMU Visor	xEMU Visor
Refractive error shift	+2.36 D	+2.40 D (or +1.82DS +1.16DC x 090)
Static visual distortion	N/A	Present
Dynamic visual distortion	N/A	Present

Submerged EMU and xEMU visor visual effects. From Brunstetter et al., Vision/Optical Challenges of NBL Ops (2025)

The shape and resulting visual distortion of submerged EMU and xEMU helmet visors in the NBL has led to some cases of subjects reporting stomach awareness and nausea. The resulting symptomology is likely attributed to motion sickness, driven by a mismatch between the visual scene and body/head motion. Motion sickness occurs when the subject is at rest with visual stimulation (i.e., virtual reality environment, simulators) or while a subject is moving with a stable visual scene (i.e., reading while in the car). It is hypothesized that dynamic distortions in the periphery of suited xEMU subjects in the NBL may lead to motion sickness due to the "unnatural swimming motion in periphery, with continuously variable image motion, size, and shape". These symptoms would be exacerbated by the subject's movement of the body/head/eyes, and movement of other objects within the subject's periphery. Additionally, the visual distortion and neuro-vestibular disturbances may be linked to a subject experiencing sopite syndrome, which is a condition characterized by persistent drowsiness, lethargy, and fatigue that occurs after prolonged exposure to motion (Lawson & Mead, 1998). It is important

to note that during the two NBL runs in which the mishap subject experienced nausea/stomach awareness, the subject did not utilize the traditional communications cap which restricts head movement in the helmet, rather the subject used either internally built speakers within the helmet or earbuds. During the second run in which the subject did not experience undesired outcomes, the communications cap was used. As with space motion sickness, minimizing head movements is thought to help control nausea and neuro-vestibular issues. During the two runs in which the affected subject suffered an undesired outcome, the head had a less restricted range of motion because the comm cap was not being used for audio communication and recorded video showed liberal movement of the head in the affected subject.

In a report of 14 surveyed xEMU subjects who have participated in NBL activities, 4 subjects reported cases of nausea with visual provocation; 1 subject reported nausea without visual provocation; and 1 subject reported visual provocation without nausea. The affected subject discussed in the mishap section above reported noticing visual distortion caused by the curved helmet glass but did not attribute any experienced visual provocation, stomach awareness, or nausea to the distortion.

3 Summary of Working Group Recommendations

The following section provides the main highlights of feedback and brainstorming from the working group, with additional context and conclusion statements from the meeting.

3.1 CNS Oxygen Toxicity

Based on the data presented from the external SMEs, there is a low occurrence of CNS oxygen toxicity at the levels operated at the NBL, and the data from some researchers on oxygen toxicity at low PO_2 ATM levels (1.2-1.3) does not factor in many other physiological parameters that can affect outcomes. The standard operating procedures of suited runs at the NBL is PO_2 1.19 ATM, it is believed that while not impossible, there is a very low risk of a NBL subject experiencing oxygen toxicity. Though not a preferred recommendation due to the working group's conclusion that oxygen toxicity is not a primary causative factor, it was discussed that NBL operations could eliminate any chance of CNS oxygen toxicity by performing dives at shallower depths in the pool through the use of elevated platforms, which would effectively lower the PO_2 ATM.

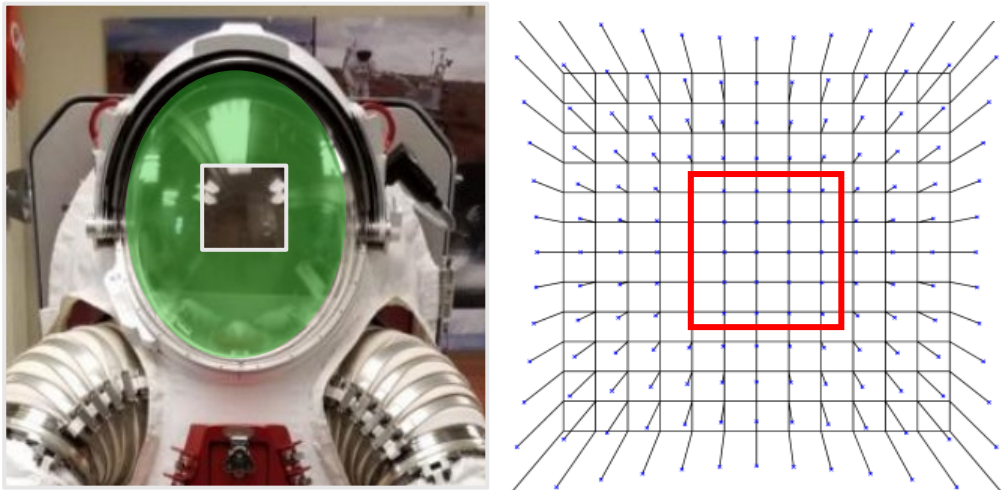
3.2 Neuro-vestibular Disturbances

During the working group, Dr. Tyson Brunstetter presented a potential mitigation strategy specific to the xEMU helmet design to prevent potential visual provocation and consequent neuro-vestibular symptomology.

One suggestion is to investigate the effectiveness of xEMU specific prescription glasses (similar to the "NBL glasses" utilized during EMU training operations), especially among subjects with presbyopia. These glasses may help to mitigate refractive error shift from the visor, improve

acuity and contrast sensitivity, and decrease eye strain and blurred vision. It is undetermined if the full prescription (i.e., sphere, cylinder, and axis) should be prescribed, or only the spherical average (i.e., +2.40 diopters), and whether the glasses would be as effective for mitigating vision disturbances in the xEMU helmet.

It was also proposed to test the effectiveness of an opaque or semi-opaque removable visor peripheral mask during periods of dynamic peripheral motion, such as during the period when the suited subject first enters the pool, when being hoisted to the surface for egress, and during periods of low activity when the subject is stationary but other objects (e.g., safety divers) are active and moving. Considerations must be taken for ensuring continuous visual monitoring of subjects if implementing this type of mitigation. It was also suggested that the xEMU helmet for the purposes of NBL activities be made into a more 'flight-like' configuration which would include the addition of sun shades on the sides of the helmet that would help to limit peripheral visibility when deployed.



Example of removable visor peripheral mask (left). Square grid object underwater with xEMU glasses - straight-ahead gaze ~30 degrees x 30 degrees - 100 cm distance from visor plane (right). From Brunstetter et al., Vision/Optical Challenges of NBL Ops (2025)

Additionally, the working group reviewers determined that it would be worthwhile to investigate the potential for re-designing the xEMU helmet with a flat glass visor for the purposes of NBL operations in order to reduce symptoms related to visual provocation.

The working group also discussed the usefulness of prophylactic treatment of nausea with anti-emetic medications such as meclizine, promethazine, or ondansetron. The panel agreed that it would not be worth the risk trade-off of subjects experiencing the common side effects such as drowsiness during suited NBL operations and the potential implications for performance. There are also the concerns for masking other potentially worse medical issues occurring during suited NBL operations. Appropriate medications should be available to treat symptoms as

needed but are not encouraged as standard practice. Other additional recommendation for further investigation is the use of scopolamine transdermal patches or Aprepitant.

Other discussed strategies for reducing neuro-vestibular issues were to inform subjects that they must alert medical staff at the early onset of symptoms to be given the opportunity to minimize body, head, and eye movements, and limit activities with increased velocities. It was noted that the CCA (Snoopy cap) worn during run 2 (without motion sickness) naturally restricted head movements. Restricting head movements within one's motion tolerance has been an effective way to mitigate motion sickness (Wood, 2011), and its plausible that this restriction of head movements contributed to the lack of symptoms during the successful run 2 and may be an effective strategy for future runs. Further investigation into whether visually induced motion sickness may be overcome with time, exposure, and adaption may help inform NBL operations early in the timeline to allow subjects to adjust to the environment when first entering the pool prior to beginning dynamic activities. It is also encouraged that NBL suited subjects participate in 'familiarization runs' prior to the higher demand runs in order to familiarize themselves with the environment and their personal symptoms and adjustment capabilities.

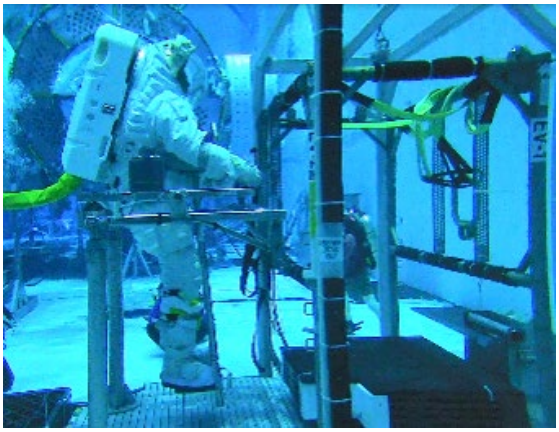
Finally, the expert review panel generally agreed that there is no known sensitivity testing that could be conducted on subjects prior to NBL activities to determine if they are more prone to experiencing neuro-vestibular disturbances.

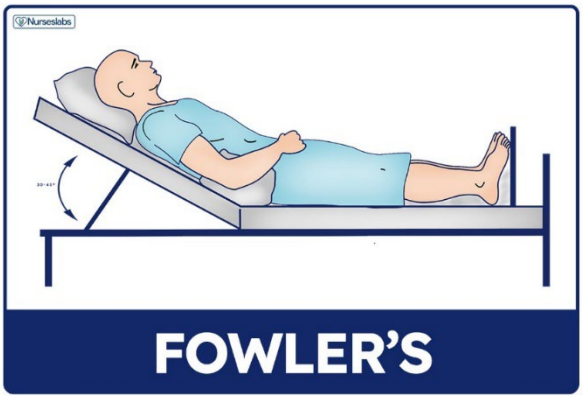
3.3 Mitigation Strategies for Orthostatic Intolerance

The expert panel reviewers discussed in length the potential for orthostatic intolerance (OI) being a potential cause of the symptoms experienced by the subject with the presenting issues. OI is defined by symptoms such as hypotension and lightheadedness (Stewart, 2013). The suited activities in the NBL are described as similar to being in a dry hypobaric chamber, with subjects being in upright postural positions for much of the activity. When experiencing symptoms of OI, remaining upright can exacerbate these feelings. Symptoms of early syncope may include yawning, and the observed facial twitching could be attributed to severe postural hypotension due to remaining upright while experiencing OI. When a suited subject in the NBL reports symptoms and the medical team makes the decision to end the run and remove the subject, the process of moving the subject to the donning stand to egress the pool requires the subject to remain standing, potentially increasing the severity of symptoms.

Based on these discussions, it was recommended by the panel that NBL operations investigate the potential for alternative extraction methods for subjects under duress, to include reduced egress time and allowing the subject to be extracted from the pool in a modified horizontal or Fowler's position. A semi-recumbent position would be appropriate when the airway is intact and a three-quarter prone or left lateral position would be appropriate if the airway is at risk of

being compromised, such as in the event the subject vomits in the suit. Additionally, having the ability to pronate the subject while awaiting extraction from the pool may help to alleviate symptoms.





Example Fowler's position. From: Nurseslabs

Partial Gravity Weighout Stand. Image Credit: NASA (2024)

It was discussed during the working group that a strategy to reduce potential symptoms related to hypocapnia or OI is to frequently 'check-in' with suited subjects and encourage continuous movement and providing a task as needed to reduce idle time to avoid decreased metabolic loads. This could include instructing the subject to move their feet and legs while standing in the suit awaiting extraction. Additionally, it was suggested that a medical pre-brief include instructions for subjects on appropriate breathing techniques to mitigate nausea.

3.4 Biomedical Monitoring during NBL Operations

Currently, standard operating procedures in the NBL include limited real-time monitoring of biomedical data during suited operations. A general discussion around recommendations for updates to protocol encouraged more biomedical monitoring capabilities should be implemented at the NBL, both real-time monitoring as well as pre- and post-run physiological data that would be useful for further investigating potential medical mishaps. The working group advised that multiple parameters would be needed to make informed assessments.

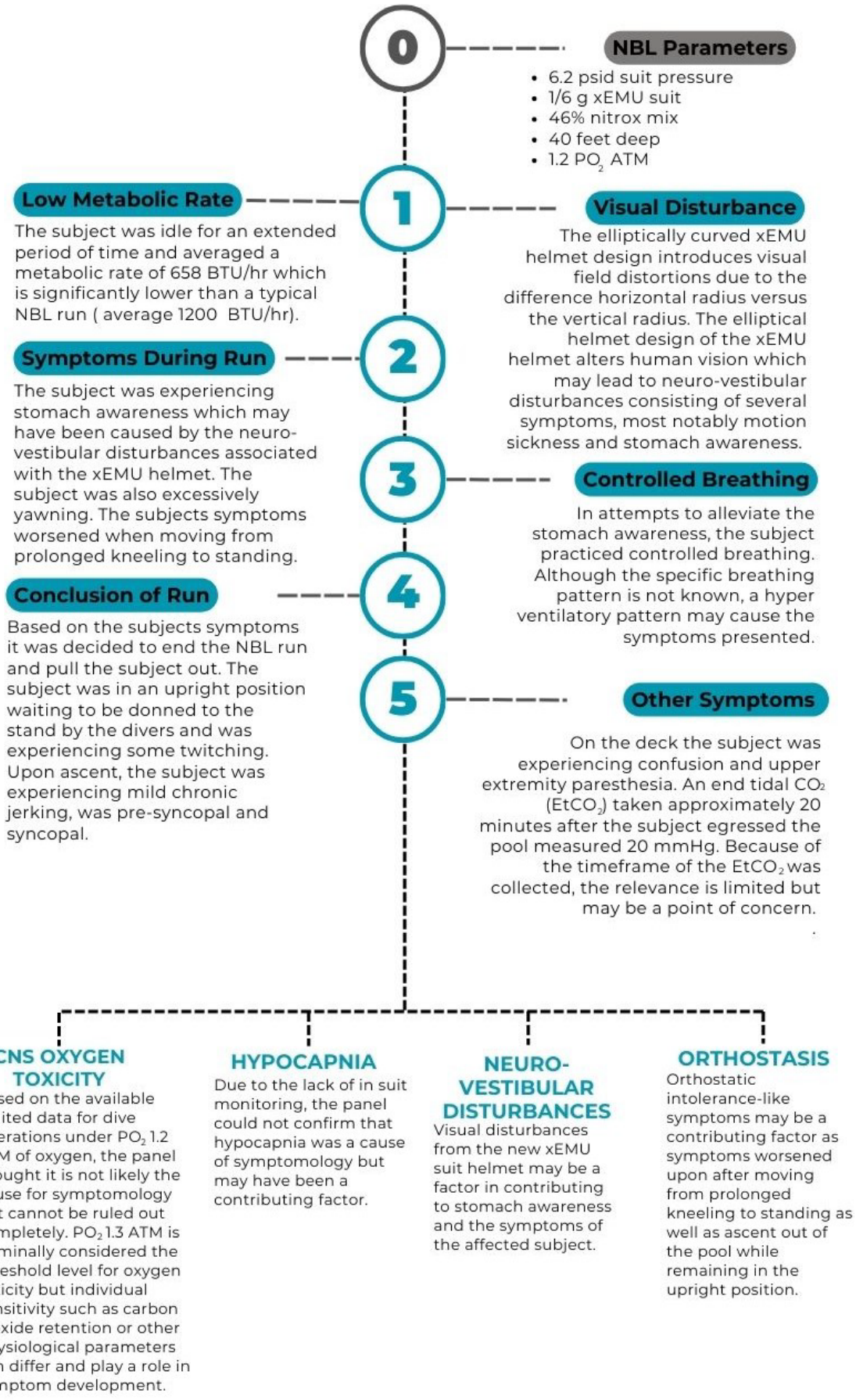
The following are some of the physiological data points that were suggested by the working group to consider for future real-time data monitoring at the NBL:

- Carbon dioxide (CO_2), particularly end tidal or expired CO_2
- Heart rate, or heart rhythm
- Metabolic rate/energy expenditure
- Blood pressure
- Respiratory rate and volume
- Skin temperature

Additionally, it was discussed that post-dive end tidal CO_2 is measured only if a subject experiences symptoms during an NBL suited run. It was suggested that a pre- and post-run end tidal CO_2 measurement be taken for all subjects to enable the assessment for hypocapnia or other CO_2-related factors, for both research purposes and to potentially better assess medical symptomology. It was also suggested that any biomedical data monitoring hardware would need to be thoroughly investigated for any potential interactions with the suit or subject during NBL test runs (ex., movement restriction due to a chest strap, placement of CO_2 sensors, etc.). There was broad recognition that a full suite of meaningful monitoring would be technically, operationally, and budgetarily very difficult.

4 Overview of Factors

The following graphic is a summary of the proposed factors implicated in the NBL case in discussion presented as potential explanations for symptomology. There are no available physiological data, thus the content is based on subject reporting, interviews with the NBL medical staff, discussions from the working group meeting, and limited video recording of the mishap.


0
NBL Parameters
• 6.2 psid suit pressure
• 1/6 g xEMU suit
• 46% nitrox mix
• 40 feet deep
• 1.2 PO_2 ATM
1
Low Metabolic Rate
The subject was idle for an extended period of time and averaged a metabolic rate of 658 BTU/hr which is significantly lower than a typical NBL run (average 1200 BTU/hr).
Visual Disturbance
The elliptically curved xEMU helmet design introduces visual field distortions due to the difference horizontal radius versus the vertical radius. The elliptical helmet design of the xEMU helmet alters human vision which may lead to neuro-vestibular disturbances consisting of several symptoms, most notably motion sickness and stomach awareness.
2
Symptoms During Run
The subject was experiencing stomach awareness which may have been caused by the neuro-vestibular disturbances associated with the xEMU helmet. The subject was also excessively yawning. The subjects symptoms worsened when moving from prolonged kneeling to standing.
3
Controlled Breathing
In attempts to alleviate the stomach awareness, the subject practiced controlled breathing. Although the specific breathing pattern is not known, a hyper ventilatory pattern may cause the symptoms presented.
4
Conclusion of Run
Based on the subjects symptoms it was decided to end the NBL run and pull the subject out. The subject was in an upright position waiting to be donned to the stand by the divers and was experiencing some twitching. Upon ascent, the subject was experiencing mild chronic jerking, was pre-syncopal and syncopal.
5
Other Symptoms
On the deck the subject was experiencing confusion and upper extremity paresthesia. An end tidal CO_2 ($EtCO_2$) taken approximately 20 minutes after the subject egressed the pool measured 20 mmHg. Because of the timeframe of the $EtCO_2$ was collected, the relevance is limited but may be a point of concern.
CNS OXYGEN TOXICITY
Based on the available limited data for dive operations under PO_2 1.2 ATM of oxygen, the panel thought it is not likely the cause for symptomology but cannot be ruled out completely. PO_2 1.3 ATM is nominally considered the threshold level for oxygen toxicity but individual sensitivity such as carbon dioxide retention or other physiological parameters can differ and play a role in symptom development.
HYPOCAPNIA
Due to the lack of in suit monitoring, the panel could not confirm that hypocapnia was a cause of symptomology but may have been a contributing factor.
NEURO-VESTIBULAR DISTURBANCES
Visual disturbances from the new xEMU suit helmet may be a factor in contributing to stomach awareness and the symptoms of the affected subject.
ORTHOSTASIS
Orthostatic intolerance-like symptoms may be a contributing factor as symptoms worsened upon after moving from prolonged kneeling to standing as well as ascent out of the pool while remaining in the upright position.

5 Conclusion

5.1 Background

The affected subject was an experienced operator in the NBL. The subject successfully completed EMU runs (at ~20 feet) for hundreds of hours, never experienced visual disturbance, sopite, or any other physiological issues. During the 3 xEMU runs at 40 feet, the subject spent 10 hours at PO_2 ATM between 1.14-1.2. Physiological symptoms occurred after 3 hours at PO_2 1.14 ATM of Run #1 and more severe symptoms after 2.5 hours of PO_2 1.2 ATM on Run #3. The subject did wear EMU/NBL corrective lenses and spent over 15 hours in the water with no adverse visual disturbance outcomes. Sopite, which may be related to the visual disturbances, occurred after 5 plus hours on Run #1 and after 3 plus hours on Run #3.

5.2 Summary

There is limited real time physiological data to make a definitive conclusion on what exactly caused the subject to experience physiological symptoms that led to the last run to be terminated early.

Oxygen toxicity was considered, based on available literature, which is limited for operations under PO_2 1.2 ATM, the overall thought was that it is not likely but cannot be ruled out completely. PO_2 1.3 ATM is nominally considered the threshold level for oxygen toxicity but individual sensitivity such as carbon dioxide retention may impact the threshold. Programs (such as Divers Alert Network and the U.S. Navy) have considered testing/screening but no longer perform testing due to feasibility, accuracy of data, and outcomes.

Individual human physiology plays a role, and any given individual can respond differently on runs with similar parameters.

Hypocapnia was also considered based on its impact due to potential hyperventilation from possible neuro-vestibular disturbances causing sopite syndrome. Hyperventilation coupled with a hyperoxic environment with low metabolic load may contribute to the observed symptoms. There is limited data on thresholds for metabolic loads, hyperoxia and hypocapnia. The subject averaged ~650 BTUs/hr during the runs with symptoms and closer to 1000 BTUs/hr on the run with no symptoms.

For future NBL runs, the parameters should remain the same with extra vigilance on subject monitoring.

The NBL should consider adding the ability to extract a NBL subject in the Fowler's position or modified prone if under duress.

Using medication to prevent neuro-vestibular symptoms was not recommended due the medication side effects of fatigue and affecting cognitive function.

If neuro-vestibular disturbances (visual issues/nausea, etc.) continue despite implementation of recommended countermeasures (e.g., use of "xEMU glasses," masking of the peripheral field during dynamic motion events, and minimizing body/head/eye movements and velocities if motion sickness symptoms are detected), redesign of the xEMU helmet with a flat front is recommended for NBL operations.

It is also important to note that there have been 20 NBL runs of the xEMU at 1/6 g, and only three exposures at the elevated suit pressure. It is suggested that careful and continuous monitoring of operations as additional xEMU NBL test runs occur is important to potentially identify any ongoing issues.

The working group review panel concludes that the symptomology which occurred during the mishap was likely a combination of multiple factors (such as the primary proximate causes described in this report, and/or other contributing factors). There is not enough supporting biomedical data available to establish a definitive diagnosis.

6 NASA JPR 1830.6: *Requirements Applicable to Personnel Participating in Diving, Hyper/Hypobaric Chambers, and Pressurized Suit Operations*

6.1 Background

Previously, NASA has maintained two separate documents for alternobaric operations: 1) **JPR 1830.3A – *Limitations applicable to Personnel Exposed to Diving*** and 2) **JPR 1880.4A – *Requirements and Limitations for Exposure to Reduced Atmospheric Pressures***. From a nitrogen absorption perspective there is no difference in the physiology and both documents had post-exposure limitations, but the JSC community was not applying a unified standard. To harmonize the requirements for alternobaric exposures across the agency, these documents are being combined into a single document: **JPR 1830.6 - *Requirements Applicable to Personnel Participating in Diving, Hyper/Hypobaric Chambers, and Pressurized Suit Operations***. Additionally, no-decompression limits and post exposure flight restrictions needed to be updated due to changes in industry practice and release of US Navy dive manual revisions. Updates were also required for prebreathe protocols and to add flexibility for Neutral Buoyancy Laboratory (NBL) operations to support exploration activities. Prior limits in **JPR 1830.3A** were never truly tested since all dives were treated as occurring at depth (40 feet) while in reality, even the deepest exposures averaged only 22 feet.

Treating dives and pressurized suit operations in a unified manner will improve community awareness of exposure risks and decrease chances for error and injury. The document is geared

to be practical for offshore/offsite operations and allows flexibility for other projects and pressure profiles that have not yet been conceived. Pressurized suit use is defined in the document as any pressure greater than 1 psid.

6.2 Major Changes and Associated Comments

- Decompression diving is no longer prohibited, but additional planning, review, and analysis is required.
- NASA no-decompression limits were updated:

  There are 200-300 exposures in NBL per year.

  The NASA Tissue Bubble Dynamics model was used to derive these limits, which were initially calculated where the model indicated "safe" bubble formation. Time points happened to be within 10 minutes of limits documented at two steps below USN table limits, therefore the USN table numbers at this more conservative time was used so the associated USN 'fly after dive' numbers could be used. These new NASA limits were deemed "conservatively appropriate" during the working group meeting.

- Surface Interval Times (SIT) after ambient environment pressure suit operations:

Combined Exposure Depth (feet)	NASA No-Decompression Limits (minutes)	US NAVY Limits
0-20	No Limit	No Limit
21-25	400	1102
26-30	240	371
31-35	190	232
36-40	135	163
41-50	80	92

For All Topside Ambient (1 ATA) Suited Operations (≤8.3 psid)				
		Surface Interval Time (SIT) before Flying		
Total Suit Pressure (psid)	Total Pressurized Time (minutes)	Air SIT (commercial) before 8K feet (hours	Air SIT (T-38 FL250) before 10K feet (hours)	Oxygen SIT (minutes)
1-0 - 4.5*	1-10*	-	-	-
4.51 - 8.9	1-10	3	6.5	20
	10-60	3	6.5	20
1.0 - 8.9	61-100	5	13	40
	101-400	16	24	120
	>400	24	26	180

*For short duration exposures to pressures of 4.5 psid or less, there is no restriction on flying, (i.e., no required surface interval)

- SIT after NBL diving operations using air:

Actual Depth + Suit Factor (feet)		Total Dive Time (minutes)	Surface Interval Time (SIT) before Flying		
			Air SIT (commerial) before 8K feet (hours)	Air SIT (T-38 FL250) before 10K feet (hours)	Oxygen SIT (minutes)
Do not forget Suit factor from table 3-2	0-20	1-60	3	6.5	20
		61-100	5	13	40
		101-400	16	24	120
		>400	24	26	180
	21-25	1-45	3	6.5	20
		46-80	8	16	40
		81-290	18	27	120
		291-400	24	28	180
	26-30	1-35	3	6.5	20
		36-60	5	13	40
		61-240	20	28	120
	31-35	1-30	3	6.5	20
		31-50	5	13	40
		51-190	20	28	120
	36-40	1-25	3	6.5	20
		26-45	8	16	40
		46-135	18	27	120
	41-50	1-20	3	6.5	20
		21-35	8	16	40
		36-80	16	24	120

- SIT after NBL diving operations using nitrox:

Actual Depth + Suit Factor (feet)		Total Dive Time (minutes)	Surface Interval Time (Sit) before Flying		
			Air SIT (commerial) before 8K feet (hours)	Air SIT (T-38 FL250) before 10K feet (hours)	Oxygen SIT (minutes)
Do not forget Suit factor from table 3-2	0-44	1-60	3	6.5	20
		61-100	5	13	40
		101-400	16	24	120
		>400	24	26	180
	45-51	1-45	3	6.5	20
		46-80	8	16	40
		81-290	18	27	120
		291-400	24	28	180
	52-58	1-35	3	6.5	20
		36-60	5	13	40
		61-240	20	28	120
	59-61*	1-30	3	6.5	20
		31-50	5	13	40
		51-190	20	28	120
	*Combined exposure depths greater than 61 fsw exceed the NASA limit of 1.35 ata pO_2				

Note that neither air nor nitrox tables account for repeat diving (i.e., there is no credit for surface time); total time underwater is used to calculate exposure.

The panel had suggestions to monitor signs and symptoms proactively so the data would be in hand for future questions about the protocol. Symptoms will obviously be recorded if they occur, but it would not be reasonable to collect bubble data on all operational runs, especially when these tables are specifically designed to not be pushing DCS limits. Risk acceptance for DCS in NBL operations is near zero. Up to 19.7% DCS risk is accepted for NASA hardware verification protocols but not for crew in nominal training operations such as the NBL.

There was also a suggestion to employ dive computer tracking of all pool profiles. The rationale is that dive tables provide rough guidance that may or may not reflect the true exposure of divers. Dive computer tracking would provide a better measure of true exposure, which could be important in assessing the decompression risk of nominal operations, as well as contingency scenarios of rapid subject removal from the pool. Despite early concerns that precision measuring would result in higher rates of decompression sickness in recreational and commercial divers, this has not been observed. More precise monitoring would also be useful for real-time assessment and event reviews.

- Suit factor needs to be added in to actual depth as indicated in tables:

Maximum Suit Pressure (psid)	Suit Example	Suit Factor (feet)
0.0 – 1.0	SCUBA, vent pressure	0
1.1 - 4.3	EMU, MACES, OCSS	+10
4.4 - 5.9	Orlan, Sokol	+14
6.0 - 8.3	Z2, Z2.5, xEMU, OCSS	+20

- An 8 psid suit pressure section was added:
    - CNS Oxygen toxicity
        - 82% of NBL runs violate the NOAA values if we use tables as written
        - With best fit curve (average PO_2) 18.6% of runs violate the NOAA lower limits
    - NASA limits inspired ppO_2 to PO_2 1.35 ATM (JPR 1830.6 2.3(b))
        - Effects have been reported at PO_2 1.2 ATM with prolonged exposure time (240 mins) (Arieli 2006).

- Z-2 suit with current limits/tolerances/umbilicals can reach a PO_2 1.36 ATM
- However, due to operational controls that are in place, NASA is not overly concerned about a seizure (or other symptoms) in the suit.
- There was a question about subjects reporting more fatigue during pool runs as opposed to using the Active Response Gravity Offload System (ARGOS) with similar activities; the panel members reported that there are known effects of this sort extreme/unreasonable fatigue after high oxygen exposure. Oxygen causes inflammatory response in lungs; there are also non-zero nitrogen bubble effects - overall these effects are considered "part of the job" by the panel.

o The following 8.0 psid suited requirements were originally established because there was no capability to reduce the partial pressure of oxygen in the breathing gas during the dive. This capability has recently been added and is called the Treatment Air System.

- *a. 8.0 psid operations should be planned for the beginning of the run, after weigh out, and other appropriate start up tasks.*
- *b. 8.0 psid suited nitrox operations should be conducted per the following guidelines:*
  - *(1) 8.0 psid operations should be planned for the beginning of the run, after weigh out, and other appropriate start up tasks.*
  - *(2) The planned depth profile should be 20 feet (allowing for inadvertent excursions to a depth of 25 fsw) and shall be limited to 375 minutes of on-gas time (355 minutes in water) when running split 8.0/4.0 psid operations with less than or equal to 120 minutes at 8.0 psid.*
  - *(3) Any split 8.0/4.0 psid run greater than 120 minutes at 8.0 psid operations shall be limited to a total run time of 240 minutes.*
  - *(4) Any 8.0 psid run deeper than 25 fsw shall be limited to 180 minutes (ref. NOAA oxygen toxicity limit).*
  - *(5) Planned excursions below 25 feet for a maximum cumulative time of 15 minutes are permissible, but no decompression limits will be determined by the deepest depth of the excursions (see Chapter 3).*
  - *(6) Inability to maintain a planned depth limit will result in reduced run time or test termination based on the judgement of the Test Director, Test Safety Officer, and Medical Officer.*

        - *c. An 8.0-psid suit can freely operate at 4.0 psid without depth restrictions, as long as the dive adheres to the above requirement for 8.0 psid operations and current EMU practices.*
    - Primary treatment of oxygen toxicity is accomplished by decreasing partial pressure of oxygen. Historically there has been no reliable method to treat but now NBL has the Treatment Air System which can change the breathing air composition to the subject with 23 seconds after activation.
        - Avoids decompression until subject/patient is safe
        - Avoids risk of arterial gas embolism (AGE)
            - Allows suited subject the time to regain consciousness at depth, prior to ascent
            - Same protocol used in hyperbaric chambers worldwide (max PO_2 2.8 ATM)
        - The panel discussed the option of removing the 8.0 psid limitations listed above given the new Treatment Air System; however, the panel believes that these are good considerations so should remain in the document.
    - There was discussion that CNS oxygen toxicity is a well characterized response to elevated oxygen with no sequelae. DCS has a much larger operational impact, although there will likely be a pause in operations with any incident (oxygen toxicity or DCS). There was also a discussion about what NASA considers an adverse event and how much attention it deserves as a mishap, as opposed to being classified as an expected negative response to the testing or training environment; NASA is very conservative in this regard.
    - The panel discussed that the concept of a "CNS clock" is not supported by data, even though every dive computer will calculate it this way. For pulmonary toxicity effects, the clock timing of exposure is a known factor. NOAA tables are primarily based on CNS effects but at the lower end of the table they incorporate pulmonary toxicity, so in a way these tables address both pulmonary and CNS effects. This is a known limitation of NOAA tables and specifically why NASA is not applying any time bounds to oxygen toxicity limits.

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