Guideline

Revised guideline for central nervous system oxygen toxicity exposure limits when using an inspired PO_2 of 1.3 atmospheres

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Abstract

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Technical and scientific divers breathing gases delivering hyperbaric pressures of inspired oxygen may be at risk of developing cerebral oxygen toxicity which can manifest as a seizure with little or no warning. The principle preventative strategy is adherence to time limits based on inspired PO_2 levels promulgated in 1991. These limits had their origins in US Navy studies of exposures to higher inspired PO_2s than are typically utilised by modern divers. Indeed, the duration limits for inspired PO_2s in the range typically utilised by technical divers (≤ 1.3–1.4 atm) have relatively little experimental provenance. Contemporary technical dives often involve decompression durations that result in breaches of these limits, and anecdotally, this common occurrence seems associated with a low risk of cerebral oxygen toxicity. A committee of experts recently sought experimental evidence that might support an adjustment to the recommended duration limits for typical technical dives. Such evidence exists only for an inspired PO_2 of 1.3 atm, which is a common default in use of constant PO_2 rebreather devices. The (1991) limit for a single exposure to an inspire PO_2 of 1.3 atm is 180 min with a 24-hour maximum of 210 min. Recent studies provide reassurance that dives with an inspired PO_2 of 1.3 atm consisting of up to 240 min of working dive activity followed by up to 240 min of resting decompression are associated with an acceptably low risk of cerebral oxygen toxicity. This recommendation was promulgated and endorsed at a recent workshop convened by the National Oceanographic and Atmospheric Administration (NOAA) involving technical and scientific divers.

Introduction

Breathing oxygen at inspired partial pressures higher than incurred when breathing air (FiO_2 20.9%) at one atmosphere absolute (atm abs)* (101.3 kPa) may result in toxic effects. Three organ systems are recognised as potentially affected; the brain (central nervous system [CNS] oxygen toxicity); the lungs (pulmonary oxygen toxicity), and the eyes (hyperoxic myopia).[1] CNS toxicity may manifest as a tonic-clonic seizure with little or no warning and, if occurring underwater, this creates a significant risk of drowning. Pulmonary oxygen toxicity causes a progressive

***Footnote:** Although DHMJ policy is to use SI units for pressure, the use of atmospheres and atmospheres absolute is so inculcated into the language of oxygen utilisation in diving, that these latter units will be primarily used for the purposes of this paper.

inflammatory pneumonitis with early symptoms like dry cough, chest discomfort, and reduced lung volumes on spirometry. As its name implies, hyperoxic myopia causes a myopic shift in ocular refraction. The exact mechanisms of all three forms are incompletely characterised, but a central theme is increased oxidative stress and free radical production. Provided no secondary event supervenes (such as drowning after a CNS toxic seizure) the CNS and pulmonary forms seem fully reversible, with the possibility of some residual effects related to hyperoxic myopia.

All three forms of oxygen toxicity are dose-dependent, where dose is a function of inspired PO_2 and time, but the circumstances under which each form may occur differ somewhat. CNS oxygen toxicity only occurs with inspired PO_2s in the hyperbaric range, that is, at inspired PO_2s greater than 1.0 atm. It can occur in a single exposure, and risk may accumulate over multiple closely spaced exposures. Pulmonary oxygen toxicity can occur with very long sub-hyperbaric exposures, but develops more quickly at hyperbaric pressures. It can occur in a single substantial exposure, and risk may accumulate over multiple closely spaced exposures. Hyperoxic myopia only occurs with hyperbaric exposures. It is not known to occur with single exposures but rather develops gradually over multiple exposures. It is most commonly seen in patients having prolonged courses of daily hyperbaric oxygen treatment, but also occasionally in technical divers participating in long expeditions with multiple dives breathing oxygen at hyperbaric pressures.

Although pulmonary toxicity symptoms may be seen in long dives or dive series, and although troublesome symptoms need to be managed, there are no reports of this becoming incapacitating in the water or producing long term effects in divers. Divers have reported myopic change at the end of prolonged expeditions, with the effects being fully reversible in the vast majority of cases.

The management of oxygen exposure during diving has a primary focus on controlling the risk of CNS oxygen toxicity with its predilection for causing a dangerous oxygen toxicity seizure. This was the focus of the present workshop, and further perspectives on relevant diving activities and CNS toxicity itself are provided below.

Technical and scientific diving

The term 'technical diving' is applied broadly to a suite of techniques primarily aimed at allowing a diver to dive deeper or stay longer or both. These techniques are often employed by recreational, scientific, and exploration-oriented divers. Detailed review of 'technical diving' methods can be found elsewhere.[2]

Using gas mixes containing helium, diving beyond 50 m is now common, and even depths beyond 100 m are not uncommon. Extreme exponents have visited shipwrecks deeper than 200 m and caves deeper than 300 m. The decompressions prescribed for these increasingly deeper dives are frequently longer than four hours and may exceed 12 hours. A ubiquitous strategy to minimise the decompression obligation is to breathe as much oxygen as is considered safe because high fractions of inspired oxygen reduce inert gas absorption during time at depth and accelerate inert gas washout during decompression. This creates a tension between maximising the inspired PO_2 to reduce decompression duration and risk of decompression sickness (DCS) versus managing the risk of oxygen toxicity. Increasingly long decompressions often result in violation of long-standing guidelines for maximum safe oxygen dose prescribed to avoid CNS oxygen toxicity.

It is increasingly common for deep dives of long duration to be performed using underwater breathing apparatus referred to as 'rebreathers'. These recycle exhaled gas (thereby minimising gas consumption) through a soda-lime canister to remove exhaled carbon dioxide (CO_2) and replace metabolised oxygen to maintain a relatively constant inspired PO_2 chosen by the diver (often referred to as the PO_2 'setpoint'). There are varying beliefs and practices around the choice of setpoints. Broadly speaking, some divers choose to leave it constant throughout a dive whereas others strategically vary it based on perceived risk, for example, lowering it during the deep working phase of the dive when the risk of oxygen toxicity poses a greater life threat and increasing it during the resting decompression phase of the dive when the greatest decompression advantage can be gained.[3] A common (but not universal) default PO_2 setpoint chosen by rebreather manufacturers is 1.3 atm.

Central nervous system oxygen toxicity

As mentioned above, CNS oxygen toxicity can result in a seizure. The mechanism is uncertain, though in general terms it seems likely that through intermediary pathways, oxidative stress either suppresses GABAergic transmission or enhances excitatory (e.g., glutamatergic) transmission, or both. Although premonitory symptoms such as visual changes, nausea, tinnitus, confusion, and muscle twitching (especially the diaphragm and facial muscles) may precede a seizure, this is not always the case. A seizure may occur without warning.

A curious feature of CNS oxygen toxicity is that for a given inspired PO_2 the risk seems higher in immersed divers than for dry occupants in a hyperbaric chamber. This risk difference may be explained by the tendency of exercising divers exposed to an elevated work of breathing to experience a perturbation of normal respiratory control which reduces CO_2 responsiveness and causes CO_2 retention.[4] This is more likely if the respired gas density is inappropriately high ($> 6\ g{\cdot}L^{-1}$) which contributes to an increased work of breathing.[5] Failure of a rebreather's CO_2 scrubber and CO_2 rebreathing is another mechanism for CO_2 retention. Carbon dioxide retention promotes cerebral

vasodilation and increased blood flow which increases brain tissue oxygen tension in the absence of any change in the inspired PO_2.[6] It follows that hard work and elevated work of breathing are considered significant risk factors for CNS oxygen toxicity during diving. Other precipitating events can include diver error (such as mistakenly breathing a hyperoxic gas at an inappropriate depth) or equipment failures (such as an oxygen addition valve in a rebreather failing in an open position). Although some of these risk factors are understood, there is also a significant component of unexplained inter- and intra-individual variability in risk.[7]

RISK REDUCTION AND MITIGATION

The primary means of risk reduction for CNS oxygen toxicity has been adherence to published inspired PO_2-time exposure limits. The most widely promulgated compilation of such limits is published in the 1991 National Oceanographic and Atmospheric Administration (NOAA) diving manual[8] (Table 1) which are still the standard taught to modern divers. Concomitantly, NOAA also created the 'CNS clock', to track cumulative oxygen exposure for a dive or a day of diving. In particular, this approach allowed the diver to account for varying inspired PO_2s over the course of a dive by summing 'CNS percent' contributions. Each contribution is calculated as:

$$\text{CNS percent} = (\text{time at } PO_2/\text{time limit for that } PO_2) \times 100$$

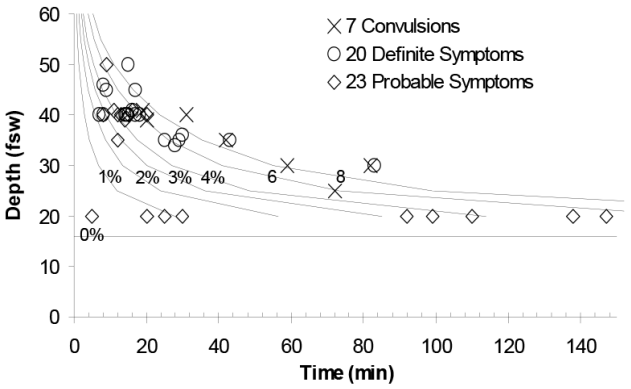
A CNS percent of 100 means that the diver has reached their maximum exposure limit for a dive. For example, based on the limits in Table 1, a CNS percent of 100 could be reached by a 180-min exposure to an inspired PO_2 of 1.3 atm or by an exposure of 90-min at 1.3 atm followed by 60-min at 1.5 atm (each of the latter two exposures being 50% of their respective limits, summing to 100%). A daily exposure percentage is calculated in the same way using the 24-hour day limits in Table 1. Most desktop decompression software programs used for generating decompression plans include CNS clock calculations for the dive.

There are a number of important points about the nature and provenance of the NOAA limits promulgated in Table 1. First, the table includes limits for lower inspired PO_2s that will not result in CNS oxygen toxicity. This reflects a goal for these limits to cover the risk of both pulmonary and CNS toxicity. Unfortunately, as the inspired PO_2 decreases, the transition from limits with primary relevance to CNS toxicity to those with a primary focus on pulmonary toxicity is undefined. Second, the experimental provenance of these limits is undescribed. However, if one searches the relevant literature for related information it becomes apparent (as described by Vann and Hamilton)[9] that "*these limits were based on best judgement from extensive experience, not on the statistical analysis of quantitative data*". This characterisation refers primarily to the limits around inspired PO_2s of most relevance to modern divers (less than 1.6 atm). There was, in fact, a moderate amount of testing at inspired PO_2s of 1.6 atm and above under the auspices of the US Navy.[10] For example, Vann[11] reported a large dataset of 773 working oxygen dives between depths of 20–50 ft (6–15 m, inspired PO_2 1.6–2.5 atm), and summarised outcomes for a

Table1
Table of oxygen exposure limits as published in the NOAA 1991 diving manual

Inspired PO_2 (atm)	Max duration (min) Single exposure	Max duration (min) 24 h day	Exceptional exposure (min)
2.0			30
1.9			45
1.8			60
1.7			75
1.6	45	150	120
1.5	120	180	150
1.4	150	180	180
1.3	180	210	240
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	

Figure 1
Statistical model of CNS oxygen toxicity risk (convulsions, definite and probable symptoms) reported by Vann and Hamilton[9] and based on US Navy testing data; fsw – feet of seawater



subset of that data including risk isopleths for a composite outcome of convulsions, definite or probable symptoms in the 2008 technical diving conference proceedings as shown in Figure 1.[9]

The 0% risk isopleth in Figure 1 is depicted as 16 ft or 1.5 atm oxygen. In fact, Vann and Hamiton reported that, informed by a model developed by the US Navy,[12] the 0% risk isopleth was, for practical purposes, considered to lie at 1.3 atm (132 kPa), and that this was the basis for the choice of 1.3 atm as the setpoint for US Navy mixed gas rebreather diving. Accordingly, one could then ask why the Navy (and subsequently NOAA) imposed a limit for 1.3 atm at all? In answering this question, Vann and Hamilton[9] cite a personal communication from Dr Ed Flynn stating that (despite the perception of little or no CNS risk at an inspired PO_2 of 1.3 atm) "*the Navy also imposed an arbitrary time limit of 240 min at 1.3 atm because of potential onset of pulmonary oxygen toxicity*". Despite this, in the same publication, Vann and Hamilton also caution that '0% risk' for CNS toxicity is an uncertain parameter, and describe several anecdotes that may represent seizure events among technical divers breathing an inspired PO_2 of 1.3 atm.[9]

There are other preventative strategies unrelated to inspired PO_2 and duration *per se*. Intermittency, referring to periodic switching to a lower inspired PO_2, typically an inspired PO_2 equivalent to breathing air at the same depth (and often referred to as 'air breaks'), is accepted to be effective in reducing the risk of CNS oxygen toxicity. However, there is no widely accepted or proven regimen in diving applications. A single five-minute air break in the middle of a 75-minute dry hyperbaric oxygen treatment at 2.5 atm was reported to more than halve the risk of oxygen seizures in human patients.[13] Another preventative approach embraces strategies to reduce the risk of CO_2 retention. These include: minimising work at depth, for example, through use of diver propulsion vehicles; maintaining respired gas density below 6.2 $g \cdot L^{-1}$;[5] and using underwater breathing apparatus with a low breathing resistance.[14] Strategies to mitigate the drowning risk should a seizure occur are also often employed by technical divers. There is some evidence to support the efficacy of mouthpiece retaining devices[15,16] and full-face masks[17] in protecting the airway in unconscious divers. Combined with use of a buddy system, these devices may make an underwater seizure more survivable. Conducting long decompressions in dry habitats[18] potentially lowers the risk of a seizure, and also improves survivability should one occur.

The modern context and workshop goals

To summarise the present situation, technical divers and other groups such as scientific divers conducting prolonged dives are working with restrictive oxygen exposure limits that are essentially untested for the outcome of greatest concern (CNS toxicity) in the range of inspired PO_2s commonly used (< 1.6 atm). As technical diving has progressed to deeper longer dives, divers are inevitably forced to ignore these limits.[19] Comments from rebreather divers who informally discussed the matter with author MM in related on-line communications reflect this situation (Table 2). As implied in comments in Table 2, there is abundant anecdote suggesting that when operating at the inspired PO_2s most commonly used by technical divers (~1.3–1.4 atm), CNS oxygen toxicity events are vanishingly rare, even when the current limits (Table 1) are substantially exceeded. Given this unsatisfactory situation of ignoring (or being unreasonably constrained by) limits that have little face validity and lack an evidential base, the context area experts among the author group for this report convened a workshop to evaluate whether it was possible to revise the current limits on an evidential basis. Importantly, this group contained strong representation from NOAA whose limits were under consideration.

The goals for the project were to evaluate relevant evidence that might allow an objective evidence-based revision of the present PO_2-time limits, and to run a workshop whose participants could have input into refining a set of statements/guidelines arising from the author committee's preliminary deliberations. This workshop took place on 29 March 2025.

Evidence supporting change

It is germane that author FGM has knowledge of and access to US Navy testing data that is relevant to this subject area. In preliminary discussions within the author group, it became clear that there are essentially no data that extend our ability to prescribe evidence-based duration limits for inspired PO_2 to avoid CNS toxicity other than at an inspired PO_2 of 1.3 atm. Since an agreed goal among authors was not to replace guesswork with more guesswork, consideration for revision was therefore limited to an inspired PO_2 of 1.3 atm. Fortunately, as previously mentioned, this corresponds with

Table 2

Representative commentary from current technical divers illustrating attitudes to current inspired PO_2 time limits; the last two boxed comments reflect the dilemma facing advanced technical diving instructors who find themselves teaching limits that they sometimes ignore, and may even exceed during training course dives

"To be honest we haven't worried about CNS toxicity for years. We do a 125 m dive on a 1.2 atm setpoint and end the dive with over 200% CNS. I've done this many, many times with little to no symptoms. The worst I ever felt was feeling a bit nauseous. We do conduct air breaks every 30 minutes."
"The [CNS limits] are something that run the risk of being ignored by deep tech divers, purely because it is unavoidable, regularly exceeded, and has little consequence or known issues. I am a big fan of using conservative limits, but they need to be based on some logic, studies, data or more modern evidence instead of what we currently have now; blindly sticking to old recommendations with no reason, is not a good strategy."
"We basically disregard the CNS clock. Pulmonary toxicity is really our only concern, and while uncomfortable it is not really a great danger, just something to be managed."
"I regularly exceed my CNS clock on our advanced 3–4-hour long CCR course dives, which can range from 130% plus on class dives. I've had my clock up to 680% on exploration dives, which have been up to 14 hours long. While I definitely teach the CNS clock, I am also very open about sharing our accumulated experience, which seems to prove that these numbers are overly conservative."
(In respect of teaching students) *"I present: here's the clock, and here's our experience. We want people to follow reasonable guidelines."*

a common default PO_2 setpoint adopted by many rebreather manufacturers, and is a pragmatic, useful setting for real-world rebreather dives.

The NOAA 1991 limit for an inspired PO_2 of 1.3 atm was 180 minutes for a single exposure and 210 minutes for any 24-hour day (Table 1). A PO_2 of 1.3 atm is currently the maximum setpoint allowed for NOAA rebreather operations, with the same exposure duration limits to those in the 1991 manual.[20] The question posed in this panel workshop was whether the published 1.3 atm duration limits remain appropriate. An abundance of community experience alluded to above and limited but relevant research data indicate that relaxation of the 1.3 limits is likely appropriate.

The US Navy single exposure duration limit for 1.3 atm inspired PO_2 is currently 240 minutes for up to four consecutive days.[21] Research trials conducted at the Navy Experimental Diving Unit (NEDU) in Panama City, FL have also included seizure-free studies out to an eight-hour (480 minute) duration. These data provide significant new information about the risk of CNS oxygen toxicity associated with diving at pressures of oxygen commonly seen in modern rebreather diving. A summary of these data is provided to help assess the relevance to the topic at hand.

Open-circuit gas supplies allow for precise control of the subject's breathing medium and allow for better control of a subject's oxygen exposure in a research setting. One hundred and twenty-six open-circuit exposures have been conducted by NEDU around the 1.3 PO_2 limit. Seventy-two six-hour (360-minute) and 54 eight-hour (480-minute) resting exposures were performed with PO_2s predominately at 1.36 atm but occasionally rising as high as 1.46 atm for brief periods due to changes in diver depth.[22] During these dives a 5-minute air break was permitted either every hour or every two hours of dive time.[23–25]

Exposures using closed-circuit rebreathers provide more comparable resistive effort and gas composition to civilian rebreather diving. The US Navy MK 16 MOD 1 rebreather controls the PO_2 in the breathing loop around a setpoint of 1.3 ± 0.15 atm from the time the diver descends deeper than 10 metres of seawater (msw) (33 feet of seawater [fsw]) until the diver ascends shallower than 3.7 msw (12 fsw).[26] Seventeen exposures lasting for eight hours with the diver at rest were conducted using the MK16 MOD 1 rebreather.[22] An additional 88 exposures were conducted using the MK 16 MOD 1 and MK 25 MOD 2 rebreathers in combination, with a duration of six–seven hours. The MK25 MOD 2 is a mechanical oxygen rebreather. However, due to the purging scheme employed during these dives and the off-gassing into the breathing loop by the divers the oxygen pressure in the breathing loop was typically 1.29 atm PO_2. These dives incorporated 43–60 minutes of exercise followed by decompression. Throughout the decompression 45 minutes of chamber air breathing occurred.[27]

More recently, 116 dives at a PO_2 setpoint of 1.3 atm were conducted using the MK 16 MOD 1 rebreather lasting between 5.4 and 8 hours.[28] These dives included moderate exercise during the first 90–150 minutes of bottom time. The decompression was completed warm and dry, there was a 20-minute air break upon reaching 30 msw (100 fsw) to facilitate transferring the divers out of the Ocean Simulation Facility (OSF) wet pot and into the OSF dry hyperbaric

chambers and then a five minute air break for every 60 minutes of decompression.

No oxygen toxicity seizures were recorded in any of the above dives, and none were terminated due to oxygen toxicity symptoms. While these outcomes are reassuring and provide an objective justification for change, a measured approach is still required since the data are limited, many of the exposures were conducted at rest, and some included five-minute air breaks every hour and portions of decompression in a dry state. All of these features may differ from typical diving practice. The relatively precise control of inspired PO_2 in research trials may represent another possible contrast with real world diving where variability in PO_2 due to differences in rebreather oxygen injection patterns or other influences may be greater. The US Navy limits the allowable setpoint variability to ± 0.15 atm, and it is unclear what variability would be seen with different rebreathers and controller configurations. Similar questions surround differences in physical activity between controlled and typical exposures, although it is generally true that in 'real-world' diving, PO_2 exposures most often approach limits during long decompressions when the divers are at rest, at shallower depths, and are breathing low-density gas. Under these circumstances CO_2 retention and its associated exacerbation of CNS oxygen toxicity risk is unlikely.[29]

It is acknowledged that this reported experience with 1.3 atm PO_2 exposures does not inform the risk assessment for either lower or higher setpoints. It can be expected that any duration limit deemed acceptable for a 1.3 atm PO_2 setpoint would result in a lower CNS toxicity risk at lower inspired PO_2 values, but there are insufficient data to generate limits that take the form of the progressively longer existing NOAA table limits (Table 1). Cases of CNS toxicity have been reported at ≥ 1.4 atm, but the true risk in the 1.4–1.6 atm exposure range remains very poorly characterised and once again, generating evidence-based limits that take the form of the existing NOAA table is not possible. In either direction, longer PO_2 exposures, particularly at the higher points in the range, will increase the likelihood of pulmonary toxicity becoming a controlling factor. While pulmonary oxygen toxicity managed in a timely manner is reversible with no known long-term consequences for divers, it may become a limiting factor that requires additional work to characterise appropriate operational restrictions.

Workshop discussion

The workshop was hosted by NOAA in Seattle in conjunction with the 2025 American Academy of Underwater Sciences Annual Meeting and it was attended by sixty participants. The participants were a mix of recreational technical divers and scientific divers. Many of the attendees were in leadership roles, and many used 'technical diving' methods in the course of their work. The consensus discussion was preceded by presentations by authors SJM (topics approximately confluent with the introduction, technical and scientific diving, and CNS oxygen toxicity sections of this paper), MM (content of Table 2), and FGM (experimental diving evidence supporting change). A series of draft consensus statements was then presented and discussed. A verbatim transcript of the discussion is not reported, but a summary of discussion themes is as follows.

Workshop participants convened for a discussion about the proposed changes to the current NOAA time limit at an inspired PO_2 of 1.3 atm. Although 1.4 atm inspired PO_2 is used in some rebreather systems as a manufacturer's setpoint, the data remain insufficient for formal recommendations related to an inspired PO_2 of 1.4 atm (or above). For this discussion, there was also an expressed need to avoid conflating pulmonary oxygen toxicity with CNS oxygen toxicity, to avoid the same ambiguity currently present in the NOAA limits.

Additional operational perspectives were provided from divers and clinicians participating in the workshop. Clinically, experts noted that hyperbaric oxygen treatments routinely involve higher inspired PO_2 exposure (e.g., over 2.0 atm) but these exposures are dry, resting, and do not involve significantly increased work of breathing or the risk of drowning from a CNS oxygen toxicity seizure. For recreational and scientific diving, an inspired PO_2 setpoint of 1.3 atm provides a balance between safety and decompression efficacy.

Participants asked about the physiological implications of multi-day and high-intensity diving when considering revised limits. While US Navy data pertaining to four consecutive days of diving reaching recommended limits at 1.46 atm inspired PO_2 suggested low CNS oxygen toxicity risk when incorporating mitigations such as rest during decompression and reduced gas density, pulmonary symptoms and hyperoxic myopia were reported following these exposures. Based on these data, the US Navy currently limits multi-day diving to four consecutive days with four hours at 1.3 atm inspired PO_2.

The US Navy data also underscore the need to distinguish between working and resting phases during dives, with resting decompression being more favorable for safely extending exposure durations, and 'resting' being formally defined as a respiratory minute volume (RMV) < 22.5 $L \cdot min^{-1}$. Practically, 'resting' is defined as not performing any activity beyond what a diver needs to do to decompress safely under optimal or near-optimal conditions. To expand operational data, dive profile data-sharing was suggested for capturing dive logs and outcomes. However, concerns were raised about the limitations of such data due to survivor bias and incomplete contextual information.

The consensus statement discussion concluded that traditional CNS limits are derived primarily from high-

PO_2 experimental conditions that are not reflective of PO_2 setpoints used in typical field diving, and available evidence supports the acceptability of extending exposure time for 1.3 atm inspired PO_2. Mitigation strategies, such as periodic breaks with lower PO_2 and reduced gas density, reduced activity during decompression, buddy systems, full-face masks, and decompression tethers, were encouraged for longer exposures. Importantly, this consensus was focused on adjusting time limits at 1.3 atm inspired PO_2, rather than treating 1.3 atm PO_2 as a rigid setpoint cap. For a potential limit extension beyond 240 minutes for a working dive at 1.3 atm inspired PO_2, there was concern about the limited robustness of both dry and wet dive data. However, 240 minutes at 1.3 atm inspired PO_2 in various operational and testing scenarios has resulted in safe CNS profiles with reasonably robust supporting data. Indeed, it was suggested that even a 240-minute exposure limit for 1.3 atm inspired PO_2 remains conservative.

Closing remarks addressed the practical application of these recommendations, especially within occupational diving programs, for increased diver productivity while still prioritising safety. The group favoured formalising a revised standard, allowing for up to four hours of working diving followed by up to four hours of resting decompression at an inspired PO_2 of 1.3 atm as being both conservative and evidence-based. Outreach to the wider diving community was highlighted as a necessary next step to explain the rationale behind the changes and ensure understanding. The vast majority of the workshop participants supported the consensus direction, with further contributions encouraged to refine these evolving standards.

The audience gave the author group permission to edit the draft statements to incorporate revisions or additions arising from the consensus discussion. Those revised statements are presented below. Of note, NOAA plans to update subsequent editions of the NOAA manual consistent with the information contained herein.

Workshop consensus statements for promulgation to the community

1. HISTORIC PERSPECTIVE

The pressure-time exposure limits promulgated by NOAA in 1991 allow 180 minutes (single exposure) and 210 minutes (cumulative exposure in 24 hours) for an inspired PO_2 of 1.3 atm, however, they are based almost exclusively on experimental exposure data for higher inspired PO_2s (≥ 1.6 atm) with an emphasis on open-circuit diving. Sustained moderate exposures (e.g., 1.3 atm) typically experienced in modern closed-circuit diving were not a consideration at the time. Further, although the limits are primarily employed to minimise the risk of central nervous system oxygen (CNS) toxicity, they were set in consideration of both CNS and pulmonary or 'whole-body' risk. While consideration of both these consequences of excessive oxygen exposures is important in dive planning, the severity of consequence is far greater for CNS. Therefore, the risk acceptance decision in setting limits to avoid CNS toxicity should be distinct from pulmonary toxicity.

2. ACCEPTABLE CENTRAL NERVOUS SYSTEM RISK OF 1.3 ATM INSPIRED PO_2

Available data suggest that a four-hour exposure to an inspired PO_2 of 1.3 atm* during the working phase of a dive is associated with a very low risk of CNS oxygen toxicity. Recent additional evidence suggests that a further four-hour exposure to an inspired PO_2 of 1.3 atm* under resting conditions[#] (for example, during decompression with minimal physical demands) is also associated with an acceptably low risk of CNS oxygen toxicity. There is anecdotal evidence describing even longer decompressions performed without incident by technical divers, but at present, extending dive time beyond eight-hours is poorly informed by data.

** Most relevant data come from studies where the inspired PO_2 was precisely controlled. There may be variability in inspired PO_2 around a selected setpoint when using a rebreather, or arising from changes in depth when using open-circuit gas. Divers should be cognisant of this issue, and attempt to minimise upward drift in the inspired PO_2 when applying this guideline.*

[#] Resting involves no greater physical activity than that required to maintain depth position and physical comfort under controlled conditions. When decompressing, use of a decompression stage or surface marker buoy to minimise required effort would aid in reducing physical activity.

3. PULMONARY OXYGEN TOXICITY RISK

Longer exposures to elevated inspired PO_2s will result in an inevitable tendency to greater development of pulmonary oxygen toxicity symptoms which may become a limiting factor in dive duration. Pulmonary symptoms do not carry the same absolute life threat as CNS manifestations, and seem invariably reversible if allowed to fully recover after the dive in which they first appear. It is strongly recommended that if pulmonary oxygen toxicity symptoms develop, no further diving is undertaken until they are fully resolved.

4. DAILY EXPOSURE GUIDANCE

When using this consensus to guide cumulative exposure to an inspired PO_2 of 1.3 atm for more than one dive in a day it is recommended that the cumulative total does not exceed the combination of four hours of working dive and four hours of underwater rest (e.g., during decompression

with minimal physical demands) per 24-hour period (starting from the beginning of the first dive).

5. RISK MITIGATION

There are several strategies divers may consider to mitigate the risk of CNS oxygen toxicity or its consequences should it occur. Risk of occurrence can be reduced by periodic five-minute 'breaks' breathing a lower inspired PO_2 (for example, an inspired PO_2 equivalent to breathing air at the same depth). This would typically be undertaken during decompression. Risk of occurrence can also be reduced by lowering the likelihood of CO_2 retention through strategies such as minimising underwater work where practicable, avoiding gas densities greater than ~6 $g{\cdot}L^{-1}$, appropriate management of CO_2 scrubbers in rebreathers, and use of low work-of-breathing underwater breathing apparatus. The risk of adverse outcomes associated with an underwater oxygen seizure can be reduced by the use of a buddy system and a mouthpiece retaining device or full-face mask, by tethering to decompression stages and use of habitats during decompression.

6. LIMITATIONS ON SCOPE OF GUIDANCE

In the absence of relevant data, no calculated increase in exposure duration guidelines for inspired $PO_2 < 1.3$ atm is proposed. It can be confidently assumed the revised 1.3 atm guidelines promulgated here could be applied at an inspired PO_2 of < 1.3 atm with less risk of CNS oxygen toxicity. No changes to the exposure duration guidelines for $PO_2 > 1.3$ atm are proposed given the lack of high-quality safety data. Although this revised guideline is based on superior evidence to the one it replaces, it should nevertheless be understood that adherence to these limits does not guarantee that CNS oxygen toxicity will not occur.

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