Health care worker decompression sickness: incidence, risk and mitigation

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ABSTRACT

Inadvertent exposure to radiation, chemical agents and biological factors are well recognized hazards associated with the health care delivery system. Less well appreciated yet no less harmful is risk of decompression sickness in those who accompany patients as inside attendants (IAs) during provision of hyperbaric oxygen therapy. Unlike the above hazards where avoidance is practiced, IA exposure to decompression sickness risk is unavoidable. While overall incidence is low, when calculated as number of cases over number of exposures or potential for a case during any given exposure, employee cumulative risk, defined here as number of cases over number of IAs, or risk that an IA may suffer a case, is not.

Commonly, this unique occupational environmental injury responds favorably to therapeutic recompression and a period of recuperation. There are, however, permanent and career-ending consequences, and at least two nurses have succumbed to their decompression insults.

The intent of this paper is to heighten awareness of hyperbaric attendant decompression sickness. It will serve as a review of reported cases and reconcile incidence against largely ignored individual worker risk. Mitigation strategies are summarized and an approach to more precisely identify risk factors that might prompt development of consensus screening standards is proposed.

INTRODUCTION

All individuals exposed to compressed-air environments risk decompression sickness (DCS), a condition resulting from the subsequent lowering of pressure upon return to normal atmospheric conditions. If rate of ascent exceeds that required to maintain tissue inert gas in its soluble form, a state of supersaturation will result. When significant enough, normal physiologic circulatory and ventilatory processes are overwhelmed and bubbles form [1]. Where they accumulate and in what volume will largely dictate their clinical significance.

No definitive diagnostic test exists, although Doppler detection of venous gas emboli (VGE) is considered a useful indicator of decompression stress [2]. VGE are frequently detected across the continuum of the compressed-air workforce and in those who dive [3,4,5]. In a majority of instances they result in no obvious harm, at least acutely. Any longer-term consequences are entirely unresearched.

Minor forms of DCS commonly produce musculoskeletal or cutaneous manifestations. More serious are those involving the brain and spinal cord.

Hyperbaric chambers were first introduced in the 1830s. Their use was entirely speculative and unlikely to have resulted in meaningful benefit. By the turn of the 20th century chambers were employed in support of mass transit tunneling and bridge construction projects [6]. Here, they served to facilitate compressed-air worker decompression and treat any resulting DCS. Over the ensuing decades the world's navies incorporated hyperbaric chambers in support of diving operations.

It was not until the late 1950s that hyperbaric chambers found their way into the hospital setting. Previously, they were located entirely at medically remote worksites. Evolution to mainstream medicine was the result of several additional therapeutic effects identified when oxygen is breathed at elevated pressures, and the term "hyperbaric oxygen therapy" introduced [6].

The "multiplace" (multiple occupancy) hyperbaric chamber is compressed with air. Its patients breathe oxygen via individualized systems and are accompanied by support personnel, working as inside attendants (IAs) [7]. With few exceptions IAs breathe the chamber's compressed-air atmosphere throughout the treat-

KEYWORDS: hyperbaric chamber; inside attendant; decompression sickness

ment period, with resulting elevation in tissue nitrogen tension. Consistent with all others who breathe compressed air, IAs must be carefully decompressed in order to minimize DCS risk.

REPORTS OF IA DCS

The earliest reported cases of health care worker DCS are found within the Proceedings of the First International Congress on Clinical Applications of Hyperbaric Oxygen, published in 1964 [8]. Each incident had occurred at Wilhelmina Gasthuis, the academic hospital affiliated with the University of Amsterdam. It was here that the clinical application of hyperbaric oxygen enjoyed its genesis. A hyperbaric operating room had been installed in 1959, primarily to increase cardiac ischemic times in the pre-cardiopulmonary bypass era.

Vermeulen-Cranch wrote:

"Three anesthetists, who were rather obese and remained sitting during decompression, were suspected of the bends (a term used to describe DCS) and each responded to recompression" [8].

He added a fourth case, this time an obese surgeon, the following year [9]. In the same 1964 Proceedings, Meijune noted:

". . . one of our people got the bends when she was running to the shop shortly after decompression" [10].

The first formal attempt to determine the incidence of health care worker DCS was published that same year [11]. Anderson and colleagues at Duke University Medical Center, in North Carolina, surveyed 58 of the 62 staff members involved in the initial 1,615 personnel exposures, supporting 666 diverse clinical and research-related chamber compressions. Four employees had earlier left the program so were not surveyed. Pressures ranged from 1.68 atmospheres absolute (ATA) to 6.0 ATA with a mean of 2.97 ATA. Ten episodes of post-decompression acute change in status were reported by the 52 (90%) respondents. Most symptoms were consistent with largely musculoskeletal and cutaneous DCS, none of which had been previously reported. These 1,516 individual exposures represented, then, an incidence of 0.62% yet a cumulative risk nearing 16%.

Duke University's hyperbaric team subsequently introduced several measures aimed at reducing DCS incidence below that inherent within their prevailing air decompression tables. A more conservative computation of the decompression obligation was employed, as were selected periods of oxygen breathing during decompression. Further, staff members were required to remain under observation within the facility for one-hour post decompression, not to be left alone that same evening, and not permitted to fly for 18-24 hours after exposure [11]. No formal reporting as to the effectiveness of these additional mitigation strategies could be found.

The first IA decompression fatality occurred in 1991. A 52-year-old nurse died within 90 minutes of exiting the Bay Medical Center hyperbaric chamber, in Panama City, Florida [12,13]. Autopsy findings confirmed her cause of death as DCS. Several operational failures and policy shortcomings had contributed to this tragic event. Her compressed-air exposure was significant in that it involved a period of time at a relatively high 6.0 ATA during a 13-hour patient treatment period. Critically, and while at 6.0 ATA, the nurse's breathing mask was supplied with air instead of an intended 50% oxygen/ 50% nitrogen "nitrox" mixture, thereby serving to further increase her tissue nitrogen uptake. Compounding this error, she had served as an IA earlier that same day, so she began her fateful chamber exposure with already-elevated tissue nitrogen levels. She became symptomatic during the latter stages of decompression, her presentation consistent with pulmonary manifestations of DCS. She was allowed to leave the hospital in the absence of any formal evaluation of her complaint.

To determine if a gender-related risk of IA DCS might exist, researchers at Virginia Mason Medical Center, in Seattle, Washington, undertook a case control analysis of 7,910 pressurizations conducted between 1976 and 1990 [14]. This period was associated with 26 cases of DCS, manifestations of which ranged from minor to severe. Exposure ratio for males and females was similar to their DCS risk. While no gender risk was identified, menses represented a significant risk factor among affected female IAs. Chamber pressures ranged from 2.0 ATA to 6.0 ATA, with 2.36 ATA most commonly employed. Rate of DCS was dependent upon maximum chamber pressure ($p<0.001$), with most cases occurring following exposures to 2.8 ATA or higher. While overall DCS incidence was a seemingly modest 0.33%, with an estimated 20-30 IAs employed during the study period [15], cumulative risk exceeded 80%.

This group likewise introduced several measures at

various intervals to mitigate against DCS, with ongoing empirical revisions as new cases occurred. They involved slowing and even briefly pausing decompression, breathing oxygen during the latter stages of decompression, and breathing oxygen during both the latter isobaric phase and decompression. A final revision in 2009, increased the period of isobaric oxygen breathing. Five additional cases were reported subsequent to this last revision. Two followed 2.36-ATA exposures, two others involved U.S. Navy Treatment Table 6 and the fifth after a 3.0-ATA exposure [15].

A 23-year study of IA DCS was reported from the University of Maryland Shock Trauma Center in 1995, involving 25,164 exposures [16]. Chamber pressure range was largely identical to that reported from Virginia Mason Medical Center, as was its most commonly used treatment pressure. Nineteen cases of DCS occurred in 13 IAs, again with no observed gender differences. Overall incidence was, therefore, 0.076% but with 120 IAs accounting for 97.5% of all exposures, cumulative risk approached 15%.

Upon the 1992 introduction of a multiplace hyperbaric chamber at Turku University Central Hospital, in Finland, it quickly became apparent to the clinical team that there were no published standards or consensus recommendations to guide IA decompression decisions [17]. Their initial approach, then, was to adopt Finnish recreational diver air decompression profiles. The first 236 pressurizations were undertaken at 2.8 ATA involving 43 IAs. Three cases of DCS occurred, representing an incidence of 1.3% and a 7% cumulative risk. The third case had occurred despite adding oxygen breathing during the entire decompression. The next 661 pressurizations were conducted at an arbitrarily reduced pressure of 2.5 ATA with two IAs rotated during each treatment in order to minimize individual nitrogen uptake. Even with this "conservative" approach another nurse suffered DCS. The authors suggested that a subsequent European Hyperbaric Consensus Conference should take up the issue of IA DCS. The 6th European Consensus Conference of Prevention of Dysbaric Injuries did just that and generated several recommendations [18]. Seemingly focused on a low reported incidence while failing to appreciate individual employee risk, however, the report concluded that *"working as a multiplace hyperbaric attendant is safe."* Other than agreeing that a linear correlation between increasing pressure and increasing DCS incidence appeared evident, that both oxygen breathing during decompression and that rotating staff members were *"extremely useful,"* the report provided nothing more specific to better guide the hyperbaric practitioner.

In 1995 Geiger and colleagues reported eight cases of IA DCS resulting from 3,350 exposures at 2.36 ATA, with ascents in accordance with U.S. Navy air decompression procedures [19]. Following the first four cases, chamber exposure time was slightly reduced and oxygen breathing introduced during decompression. The next two cases prompted heightened IA pre-exposure screening. After the final two cases, oxygen breathing was added for 10 minutes during the terminal isobaric phase. Four more cases occurred subsequent to this last revision.

A 1999 report from Catalina Island's hyperbaric chamber, a facility associated with the University of Southern California School of Medicine, Los Angeles, focused attention of the problem of unscheduled chamber decompression from high inert gas tissue loading exposures [20]. Two of three IA DCS cases occurred secondary to a chamber "abort." In this situation there are few if any opportunities to engage various protective measures.

A 2001 urgent chamber abort resulted in a particularly damaging and career-ending case of cerebral and spinal cord DCS. A seriously injured diver went into ventricular fibrillation after several hours under pressure that had earlier reached 6.0 ATA. The IA involved, a 44-year-old nurse, breathed oxygen during a very rapid chamber ascent and for the six minutes it took to remove and initiate what would ultimately prove unsuccessful resuscitation, and ready the chamber for reuse in order to address her omitted decompression. She was then recompressed to 1.9 ATA and breathed oxygen and air for three 60/15-minute cycles. Upon eventual exit from the chamber, she felt "very tired" and returned home, where she was found several hours later in acute distress. Serial hyperbaric oxygen therapy and supportive care was incompletely successful, and she remains cognitively impaired today [21].

Following three cases of cutaneous DCS in 395 pressurizations involving 18 nurse IAs (0.76% incidence; 17% cumulative risk), Brattebo and colleagues elected to modify their standard IA decompression protocol [22]. In addition to breathing oxygen during a seven-minute ascent from 2.4 ATA, a five- or ten-minute oxygen

breathing period (dependent upon chamber compression time) was introduced during the terminal isobaric phase. Encouragingly, no further cases occurred during the next 1,139 pressurizations. Confidence in this revised protocol was eventually shaken when, in 2002, one of their IAs suffered *"a serious episode of neurological DCI",* an acronym for decompression sickness [23]. This incident was carefully investigated and no procedural errors or specific individual risk factors could be identified.

As measurement of subsequent DCS incidence against modifications in the decompression process was considered impractical, Risberg and colleagues, from this same Bergen, Norway hospital, chose instead to assess changes in Doppler-detected VGE scores as a consequence of varying DCS mitigation efforts [23]. Walker and colleagues had done something similar in that they compared two different hyperbaric treatment protocols, 2.0 ATA and 2.8 ATA, with regard to degree of detectable "bubble grade" [4]. Walker's group found that Doppler-detected VGE were actually commonplace following these two somewhat routine clinical treatment pressures/durations. Bubbles were observed in 8/18 exposures (44%) on the 2.0-ATA protocol and 13/19 exposures (68%) when studying the 2.8-ATA protocol. DCS occurred in only one of the 37 subjects involved and in whom no bubbles were observed, suggesting that bubbles form and produce harmful effects beyond those detectable within the vascular bed. Somewhat surprisingly, Risberg's group found that VGE scores were not significantly altered by differing levels of oxygen breathing and varying rates of chamber ascent [23]. They concluded, therefore, that following their standard hyperbaric treatment pressure of 2.4 ATA for approximately 115 minutes, IAs remain exposed to significant decompression stress. This data could also be interpreted as evidence that Doppler-detected bubble scores do not correlate with DCS risk in IAs. Risberg et al.'s findings were considered unacceptable by their institution to the extent that all future elective hyperbaric treatments were to be provided in oxygen-filled monoplace (single-patient; no attendant) chambers.

In May 2007 a second health care worker died as a result of her decompression injury, a case not widely known, nor formally reported, due the manner in which death occurred [24,25,26]. This 43-year-old nurse was employed by the hyperbaric medicine service at Los Robles Medical Center, in Thousand Oaks, California. In April 1999 she attended an injured diver. Clinical decision-making related to her patient was inconsistent with commonly accepted practice standards, resulting in a considerably longer chamber exposure/nitrogen uptake period than otherwise medically necessary. Immediately upon exiting the chamber the nurse experienced shortness of breath and collapsed against a patient gurney. She first thought this to be an asthma attack and initiated treatment with her asthma reaction kit. Soon thereafter she developed weakness in both upper and lower extremities. Within a relatively short period she was essentially paralyzed and diagnosed as suffering cardiopulmonary and spinal cord DCS. Despite serial hyperbaric oxygenation and physical therapy, she failed to respond and was rendered a complete quadriplegic. Her subsequent clinical course was complicated by ongoing infections, consistent with her paraplegic state, and eventually succumbed to overwhelming sepsis eight years after her accident. If this was not tragic enough, the nurse operating the chamber at the time of this incident was so overcome with remorse that, despite the fact she had been following a physician's order, she took her own life.

In 2012 a particularly serious and career-ending case of cerebral and spinal cord DCS was described in a 50-year-old experienced male IA [27]. Within 10 minutes of exiting the chamber he became irritable and confused. He subsequently developed progressive weakness and paresthesia in both lower extremities and was soon unable to ambulate. His chamber exposure had been a relatively modest 2.2 ATA for 110 minutes, with return to surface using the appropriate U.S. Navy air decompression table. Within 60 minutes of completing decompression he was returned to the chamber for treatment. Several days of hyperbaric oxygenation resulted in complete resolution. Interestingly, this individual underwent a transthoracic echocardiogram some 18 months later and was diagnosed with a large patent foramen ovale. His cerebral manifestations of DCS were attributed to paradoxical embolization.

REPORTS OF NO IA DCS

The first of three papers to report zero incidence of IA DCS, each representing the experience of a single institution, was published in 2009. Cooper, et al. summarized Royal Hobart Hospital, Tasmania, Australia,

data between 1992 and 2005, involving 155 IAs and 5,821 exposures [28]. Treatment pressures ranged up to 4.0 ATA, with the majority (84%) at 2.36 ATA. Oxygen breathing had been employed during all decompressions. IAs were limited to four hyperbaric exposures per week, with no more than three in consecutive days. In most instances, a minimum 18-hour break was required between exposures, and flying forbidden for 24 hours from the last chamber exposure. Because of regional mountainous terrain, IAs living at altitudes greater than 600 meters were required to remain at sea level (that of the hospital) for a minimum of four hours.

Interestingly, two cases of IA DCS did occur at this institution in the post-study period, each associated with altitude exposure [29]. The first involved a 41-year-old female who walked to her home, at an altitude of 275 meters, approximately one hour after a 2.36-ATA 100-minute exposure. She immediately developed constitutional symptoms consistent with DCS. Symptoms persisted into the following day, at which point she contacted the hyperbaric service. She was instructed to return to the hospital immediately, where she successfully underwent serial hyperbaric oxygen therapy. The second case involved a 48-year-old female who had completed a largely identical chamber exposure. Four hours and 40 minutes after decompressing she drove to an altitude of 1,268 meters, becoming symptomatic while passing 750 meters. Realizing she had contravened hospital policy she promptly returned to sea level, with symptoms improving as she descended. It was not until the following day that she reported her residual condition and likewise recovered fully following a course of hyperbaric oxygenation.

In a companion paper, the Hobart group reported the results of a Doppler analysis of IA decompression stress secondary to their 2.36-ATA treatment protocol, involving 28 subjects [30]. Based upon the Kisman-Masurel scoring system [31], 68% of exposures resulted in a low bubble grade, 22% an intermediate grade and 10% a high bubble grade. This degree of bubbling was considered acceptable by the authors when referenced against the Canadian Defense and Civil Institute for Environmental Medicine "safe" decompression recommendation of Grade II or fewer bubbles in 50% or more of the subjects. Some would argue, however, that what is acceptable for military and civilian professional divers is not necessarily the case within the health care workforce. While no cases of DCS had occurred, the longitudinal impact of repeated development of intravascular gas continues as an unknown. Again, one might conclude that Doppler VGE scores do not necessarily correlate with IA DCS.

Uzun, et al. undertook a retrospective analysis of 4,532 hyperbaric exposures delivered at Gulhane Military Medical Academy Haydarpasa Teaching Hospital, Ankara, Turkey, between 1997-2006 [32]. Forty nurses served as IAs during the study period, and chamber pressures ranged from 2.4 ATA-6.0 ATA. Oxygen was breathed during the final 10 minutes of the isobaric phase and throughout decompression. For longer exposure periods and higher treatment pressures two IAs were rotated during each exposure. A policy of no more than one daily exposure and a maximum of three weekly exposures had been enforced.

In the final no-DCS paper, Witucki, et al. reviewed 28 years of experience at the University of California, San Diego [33]. While this program's treatment pressures ranged up to 6.0 ATA, they chose to address only their 2.36-ATA protocol from a DCS incidence perspective. For exposure periods of less than 80 minutes IAs decompressed entirely in accordance with U.S. Navy air diving procedures. For exposure times between 80-119 minutes, IAs breathed oxygen for 15 minutes during the terminal isobaric phase. Of the 24,160 treatments delivered at 2.36 ATA, 98% occurred within this time frame. For exposures between 120-139 minutes, IAs breathed oxygen for 30 minutes during the terminal isobaric phase. Notably, air was breathed during decompression for all three time periods. Operational policy limited IAs to a single exposure within a 12-hour period. For higher chamber pressures, IAs were not permitted to undergo another exposure for a minimum of 24 hours.

EVOLUTION OF IA DECOMPRESSION PROCEDURES

Safe return of health care workers from the hyperbaric chamber's compressed-air atmosphere has been an important operational consideration since the advent of hyperbaric medicine. Table 1 lists the evolution of decompression procedures over the past six decades, each adaption dictated in large part by a perceived inadequacy in an existing approach.

TABLE 1: EVOLUTION OF DECOMPRESSION PROCEDURES

In accordance with prevailing (usually naval) air decompression tables
Introduction of a 'safety stop' during latter stage of air decompression
Significant slowing of prevailing air decompression rate
Oxygen breathing during decompression
Oxygen breathing during slowed decompression
Oxygen breathing during terminal isobaric phase and decompression
Increasing period of oxygen breathing during terminal isobaric phase and decompression
Rotating IAs during isobaric phase
Limiting IA attendance/exposure to an 'as needed' only basis
Nitrox and air breathing cycles throughout the isobaric phase
Nitrox (50% or 60% oxygen) breathing throughout the isobaric phase
Oxygen breathing during initial and terminal isobaric phase and decompression

Initially, prevailing air decompression tables generated for military and professional diving disciplines were adopted. These tables, however, were formulated with an incidence of DCS considered acceptable to respective underwater tasking so it was not long before their inherent risk became unacceptable for the expectations of the health care workplace. Clinical programs eventually adopted the common recreational diving community practice of a "safety stop" during the latter stage of decompression. This pause had been shown to reduce Doppler-detected VGE. Slowing the rate of decompression became another strategy. The U.S. Navy had earlier introduced an operational reduction, slowing by 50% the ascent rate for surfacing divers. Hyperbaric practitioners slowed the rate even further, with many combining it with the safety stop. The intent of all of this was to limit the degree of decompression-induced inert gas supersaturation.

Subsequently, oxygen replaced air breathing during decompression, via an individualized oral nasal mask. Here, oxygen's value is twofold. Primarily, it removes the 78% nitrogen in air, thereby steepening its elimination gradient from blood to lung. Further, should nitrogen bubbles form within blood, surrounding high oxygen tensions prompt volume reduction through the process of inherent unsaturation [34].

With cases of DCS continuing to occur, oxygen breathing was extended into the latter isobaric stage, which served to limit nitrogen uptake prior to decompression. This was at first controversial as it placed IAs at risk of potentially incapacitating central nervous system oxygen toxicity. They were instructed, therefore, not to secure their oxygen mask so it would fall off in the event of seizure, thereby returning the IA to air breathing, and to limit physical activity. With no subsequent reports of toxicity, this concern was eventually allayed. Isobaric oxygen breathing periods were subsequently extended from 5-10 to as many as 20 or more minutes. Less common has been the practice of breathing oxygen upon arrival at the isobaric phase [17]. Doing so adds a measure of protection in the event of unscheduled decompression, but has to be reconciled against the greater patient care demands common during this early treatment phase.

Where staffing levels are sufficient, some insitutions elect to rotate IAs during a given treatment [17]. This again serves two purposes. Principally, it reduces nitrogen uptake to the point that DCS risk is essentially eliminated for both staff members upon scheduled decompression. Further, any unscheduled decompression, secondary to a medical emergency, critical equipment failure or chamber structural issue, can place the IA at high risk of DCS. Having two IAs "divide" the nitrogen exposure significantly lowers this threat.

Another protective method is for IAs to breathe a "nitrox" mixture (commonly 50% oxygen/50% nitrogen), either continually or intermittently [41,42]. This can, however, be logistically burdensome, particularly when

TABLE 2: IA DCS RISK MITIGATION

Pre-employment "fitness to work in pressurized settings" health screening.
Periodic re-screening
Rescreening following illness or injury
Maintain general wellbeing (cardiovascular fitness and body mass index issues)
Ensure pre-exposure fitness: adequate hydration, well rested, report any acute change in health
Enhance decompression procedures beyond prevailing air decompression tables
Mandatory observation period at the hyperbaric facility immediately post-decompression
Mandatory reporting of any acute change in post-decompression health status
Mandatory 'hold' period post-decompression before altitude exposure/pressure reduction
Limit number of weekly and consecutive daily exposures
Avoid scheduling of 'repetitive' exposures
Employ minimum chamber pressure necessary to produce desired therapeutic effect

meeting multiple patient care needs and involving larger chambers where mask supply and exhaust hoses are of a fixed length.

Finally, that many uneventful decompressions can be followed by an injurious one, all having involved a largely identical chamber exposure, suggests one or more individual risk factors. While not within the scope of this review to address this aspect of IA decompression safety, recommended operational policies and employee expectations to mitigate against DCS are included here as Table 2.

IA DECOMPRESSION PROCEDURE GUIDANCE

The empirical nature of various decompression procedures, absence of controlled comparative data, lack of consensus standards, and with little procedural uniformity, makes it largely impossible to provide high confidence guidance across the range of hyperbaric chamber pressure-exposure profiles. While patient therapeutic dosing and IA decompression decisions rest ultimately with the treating physician, what follows are recommendations that represent the opinions of this author.

1. **Decompression sickness and cerebral arterial gas embolism**

U.S. Navy Treatment Tables 5, 6 and 6A are arguably the most widely adopted dosing protocols for these two indications [35]. Several revisions of the U.S. Navy Diving Manual have incrementally extended IA oxygen breathing times [36]. The current standards appear in Revision 7, 2016, of the Manual [35] and included here as Table 3. The goal of increasing periods of oxygenation is to completely eliminate DCS, and a response to a Freedom of Information Act request suggests that this may have been achieved [37]. There have been no reported cases during U.S. Navy operations between 2006-2016. Not addressed in this FOIA request was a breakdown of number of exposures per treatment table. In the absence of anything to the contrary, then, U.S. Navy IA decompression procedures should be employed.

2. **Clostridial myonecrosis and carbon monoxide poisoning**

Weight of prevailing evidence supports treatment of clostridial myonecrosis at 3.0 ATA in order to maximize its anti-microbial effect [38]. Likewise, laboratory and clinical data support 3.0 ATA (at least for the initial treatment) for carbon monoxide poisoning [39,40]. This relatively high chamber pressure warrants a particularly conservative approach to IA decompression.

Rotating IAs is effective in both limiting respective individual nitrogen uptake to the point that DCS is highly unlikely, and in protecting against DCS in the event of unscheduled decompression. This will, however, require a larger staffing pool that might not always be available. Alternatively, IAs can breathe a nitrox mixture (50% or 60% oxygen, balance nitrogen), involving 15 minutes of nitrox/10 minutes of chamber air atmosphere cycles [41]. Again, this greatly limits nitrogen

TABLE 3: U.S. NAVY TABLE 17-7. TENDER OXYGEN BREATHING REQUIREMENTS (NOTE 1)

Treatment Table (TT)		ALTITUDE: surface to 2,499 feet	ALTITUDE: 2,500 feet to 7,499 feet	ALTITUDE: 7,500 feet to 10,000 feet
TT5 (Note 2)	without extension	:00	:00	:00
	with extension @ 30 fsw	:00	:00	:20
TT6 (Note 2)	up to one extension @ 60 fsw or 30 fsw	:30	:60	:90
	more than one extension	:60	:90	:120
TT6A (Note 2)	up to one extension @ 60 fsw or 30 fsw	:60	:120	:150 (Note 3)
	more than one extension	:90	:150 (Note 3)	:180 (Note 3)

Note 1: All tender O_2 breathing times in table are conducted at 30 fsw. In addition, tenders will breathe O_2 on ascent from 30 fsw to the surface.

Note 2: If the tenders had a previous hyperbaric exposure within 18 hours, use the following guidance for administering O_2:
For TT5, add an addition 20-minute O_2 breathing period to the times in the table.
For TT6 or TT6A, add an additional 60-minute O_2 breathing period to the times in the table.
For other treatment tables contact Naval Experimental Diving Unit for guidance.

Note 3: In some instances, tender's oxygen breathing obligation exceeds the table stay time at 30 fsw. Extend the time at 30 fsw to meet these obligations if patient's condition permits. Otherwise, administer O_2 to the tender to the limit allowed by the treatment table and observe the tender on the surface for 1 hour for symptoms of DCS.

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uptake/risk in the event of unscheduled decompression. An even more unique and somewhat controversial approach is to leave patients largely unattended during treatment. Here, an IA will enter the chamber only to attend a specific patient need, and this is done while breathing nitrox. The IA decompresses immediately thereafter. The amount of nitrogen taken up with this approach would never be sufficient to complicate decompression and has been adopted at Copenhagen University Hospital, Denmark [42]. While many are likely to find this an unacceptable level of attendance, the Copenhagen group is no doubt influenced by a 2012 report from the Danish Agency for Safety and Health at Work that considered working in a hyperbaric chamber to be particularly risky, rating it as one of the 10 most dangerous professions [43].

In the absence of nitrox availability, an unwillingness to leave patients unattended and an inadequate pool of IAs for rotational purposes, one is left with oxygen breathing and slowed decompression. Arguably, oxygen should be breathed for a yet ill-defined period during the terminal isobaric phase (with the IA at rest and avoiding cramped positions) and throughout decompression.

3. **All other common indications**

A majority of multiplace chamber treatments are rendered at chamber pressures ranging from 2.36 ATA to 2.5 ATA. It is here that an opportunity exists to significantly reduce DCS incidence and risk by lowering chamber pressure to the minimum required to achieve the desired therapeutic effect. It was the pioneering work at the U.S. Air Force School of Aerospace Medicine, in San Antonio, Texas, that introduced the now widely

adopted multiplace chamber dosing protocol [44]. This early experience involved patients breathing oxygen via oral nasal masks in chambers compressed with air. As these masks do not achieve 100% delivery [45,46], the 2.0-ATA *oxygen* pressure in common use in Europe and North America at the time, invariably using oxygen-filled monoplace chambers, could not be achieved with a multiplace *chamber* pressure of 2.0 ATA. Pressure was, therefore, adjusted upward to a somewhat arbitrary 2.36 ATA [44]. Some practitioners have since rounded this up to 2.4 ATA, others to 2.5 ATA. With the introduction of a hood that fully encloses the patient's head, 100% oxygen inhalation is assured. It is no longer necessary, therefore, to adjust chamber pressure to compensate for this chamber pressure-oxygen pressure mismatch. Increasingly, multiplace programs have elected to lower chamber pressure to 2.0 ATA as a function of the hood's effective delivery of 100% oxygen and a consequence of IA DCS at higher pressures. This 2.0-ATA chamber pressure recommendation would not apply where oral nasal masks continue to be used.

An oxygen pressure of 2.0 ATA is consistent with the treatment protocol guidelines of the Undersea and Hyperbaric Medical Society [47], coverage determinations of the U.S. Medicare program, practice standards of a great majority of monoplace chamber facilities, and that employed in the first randomized double-blind sham-controlled clinical trial to establish efficacy in deficient wound healing [48]. The second of the two large (from a hyperbaric medicine perspective, at least) randomized, double-blind sham-controlled trials to demonstrate a significant wound healing advantage used a 2.5-ATA chamber pressure and oral nasal mask oxygen delivery [49]. As noted above, this mask type does not affect 100% oxygen inhalation [45,46]. It is quite likely, therefore, that study subjects were treated with an oxygen dose ranging from 2.1 to 2.2 ATA. An early animal study did demonstrate increasing angiogenesis with increasing hyperbaric oxygen pressures [50]. While this research did not determine the level at which its density had become sufficient enough to prompt an adequate therapeutic response, the study's first author acknowledged that 2.0 ATA was *"clinically acceptable"* [51]. Prevailing efficacy data and a great deal of clinical experience indeed support 2.0 ATA oxygen in this regard.

A pressure of 2.0 ATA for a typical IA exposure duration of up to 120 minutes (a time not generally exceeded) is a relatively modest inert gas loading exposure, representing just 51% of the time allowed by the prevailing U.S. Navy air decompression table before staged decompression (a brief pause during ascent) becomes necessary [35]. One anticipates that breathing oxygen during decompression from this exposure and slowing rate of ascent to slower than the U.S. Navy's prescribed 30 feet per minute would be very protective. Adding a period of oxygen breathing (10-20 minutes) during the terminal isobaric phase would appear particularly conservative.

For those who elect to continue to practice within the 2.36-ATA to 2.5-ATA chamber pressure range, IA decompression recommendations would be essentially those noted in Number 2, above. Some argument exists for these higher oxygen pressures to be employed in the management of necrotizing soft tissue infections and when treating/mitigating against ischemia-reperfusion injury.

Denitrogenization

There is another mitigation strategy yet to be considered, one that involves IAs breathing oxygen immediately prior to entering the chamber. This denitrogenization process has been routinely employed in military aviators and astronauts. Castagna et al. [52] studied the effects of 30 minutes of oxygen breathing in 21 recreational divers, ending 10 minutes before entering the water. Post-dive Doppler analysis of VGE found significantly decreased counts in all dives preceded by preoxygenation. Decreases were even greater following a second dive two hours later, one that did not involve additional preoxygenation. This suggests a second mechanism, as nitrogen would be expected to reaccumulate during the air breathing interval. It has been hypothesized that replacement of nitrogen by oxygen occurs within pre-existing micronuclei before they are able to form bubbles [53]. Further, reduction of tissue oxygen upon reverting to air breathing several minutes before pressurization might serve to enhance consumption of oxygen from nuclei, to the point of eliminating them completely.

Some 30 minutes of oxygen breathing beginning 40 minutes before pressurization would not appear too burdensome as others prepare the chamber. Its particular value might be for 3.0-ATA pressures and where the upcoming treatment represents the IA's second exposure within the previous 12 hours.

SUMMARY

The hyperbaric work environment has not received the degree of oversight afforded those with potential for exposure to radiation, blood-borne pathogens and chemical hazards, in terms of establishing exposure limits, employee screening standards and incident reporting. That neurologically damaging and career-ending DCS continues to occur, that such cases are certainly under-reported, and that at least two IAs have succumbed to their decompression insults, argues for more critical attention on this important health care worker risk issue.

A case can, therefore, be made for the development and introduction of a population-based longitudinal study, perhaps in the form of a robust registry. Ideally, it would be international in scope and participation at the very least encouraged by those whose remit extends to safety and well-being within the health care workplace. The results of any such undertaking would be likely to generate a more comprehensive appreciation of scope and degree of IA decompression injury. It would better identify risk factors, thereby prompting consensus screening standards that could serve to optimize decompression procedures. The sum of all of this would be improved employee safety within this unique occupational environmental setting.

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