Decompression Sickness Treatment Using a Pressure Suit After Loss of Spacecraft Atmosphere

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BACKGROUND: Loss of spacecraft atmosphere (LOA) during Earth-Moon transit may require up to 144 h of pressure suit operations. This work investigates the feasibility of DCS treatment in this paradigm and discusses the operational and engineering implications.

METHODS: Three scenarios of LOA-induced DCS were considered: a permanent LOA secondary to a 0.25-in (0.64 cm) hole (unrecoverable cabin leak), a transient LOA, and a permanent LOA with early suit over-pressurization (beyond suit specification). Each was simulated in the context of the current Orion spacecraft operational concept with regards to atmosphere and anticipated cabin depress profile. Probability of DCS symptom resolution (*P*(SR)) was estimated using the previously derived Hypobaric DCS Treatment Model, with ΔP calculated from a Three Region Well-Stirred Tissue (3RWT) bubble dynamics model. Analysis was conducted and analogies drawn from experiences with the development and testing of the Orion Crew Survival System (OCSS).

RESULTS: Maintaining 8 psia at 100% F_iO_2 following LOA resulted in an eventual halt and regression of bubble growth with a *P*(SR) of 87% (at 8 h, time to symptom onset (T_s) = 105 min, with ambulation). If cabin atmosphere was not restored and psia dropped to 4.3, bubble growth returned, but again eventually slowed and regressed over time (*P*(SR) = 75% at 21 h). If the leak is repaired within the 8-h period, 8 psid (psia = 22.7) resulted in *P*(SR) of greater than 95%. Similarly, if the suit was over-pressurized (12 psid/psia) within 3 h after LOA, *P*(SR) exceeded 95%.

DISCUSSION: A launch/entry pressure suit represents a contingency option for DCS management in the event of LOA.

KEYWORDS: hypobaric decompression sickness, pressure suit, cabin depressurization, loss of spacecraft atmosphere.

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Hypobaric decompression sickness (DCS) remains a risk in nearly all strata of human space exploration. It is precipitated by a decompressive exposure following inert gas breathing resulting in relative tissue tension supersaturation. Decompression may be intentional, such as during extravehicular activity (EVA) or deliberate cabin depressurization, or unintentional, such as a cabin depressurization caused by a structural failure, inadvertent valve-opening (resulting from a system failure or operator error), or a seal leak.

Regardless of hazard source, the prevailing approach to DCS in human spaceflight emphasizes the optimization of preventative risk management strategies well before treatment capability considerations.[5] For EVA, DCS risk is estimated and mitigated to within a low but deemed acceptable risk level and is therefore considered an occupational hazard of nominal human spaceflight operations.[5] Risk mitigation is achieved through conservative prebreathing protocols that are iteratively refined through empiric and analytical investigations, and have been profoundly successful. Nevertheless, treatment capabilities and protocols have also been developed which, for ISS operations for example, employ the EMU spacesuit for pressure and oxygen delivery (with or without application of the Bends Treatment Apparatus to enable overpressurization beyond rated specification).[5] Unplanned cabin depressurization and ensuing loss of spacecraft atmosphere (LOA) are mitigated by structural design,

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vehicle contingency operations (such as "feed-the-leak" capabilities), and pressure suits. These suits are routinely employed in modern spaceflight operations as a protective mechanism against the negative physiological impacts of LOA, which, in addition to DCS include hypoxia, barotrauma, arterial gas embolism, hypothermia and visual obstruction from fog condensate (both secondary to adiabatic cooling), noise, vibration, and/or injuries from flying objects.

During dynamic periods of increased risk of structural failure, such as launch, entry, and docking, the crew assumes a protective configuration with pressure suits donned, gloves on, and visors down. In between those times–during the nondynamic phases of flight–these suits are not worn as the risk is considered sufficiently small and any residual benefit is outweighed by decreased mobility (implications on performance, dexterity, fatigue and/or potentially overuse/friction injuries), thermal concerns, and toileting. Pressure suits are required during these periods, however, if LOA were to occur, to provide safe haven throughout the transit back to Earth. These periods will be increasingly long for exploration-class missions.

The Orion spacecraft is a next generation exploration-class vehicle designed to transport a nominal crew of four to destinations beyond low Earth orbit. Orion's Earth-Moon transit trajectory results in Earth-return contingency options that vary up to a maximum of 144 h. In the event of LOA, this mission timeline imposes a requirement for continuous pressure suit operations throughout the Earth-return period. This contingency requirement introduces design and operational constraints that differ considerably from previous launch/entry pressure suits or EVA spacesuits, and is the fundamental driver of the S1041 Orion Crew Survival System (OCSS) design.[9] The OCSS suits operate in a parallel suit loop within the Orion's closed loop continuous flow Environmental Control and Life Support System (ECLSS), with variable pressure capability from 0.5 to 8 psid (nominal minimum operating pressure 3.5 psi).[9]

In the event of cabin depressurization, Orion (nominal atmosphere of 14.7 psi 80/20 N_2/O_2 mix) is capable of feeding a 0.25-in (0.064 cm) hole for approximately 1 h while maintaining an 8.0 psi cabin pressure. This hour provides the crew time to don the OCSS ensemble (including long-term waste management system). The pressure suit will then provide the differential pressure necessary to maintain 8.0 psia with enriched ($\sim$95%) O_2 gas mixture, for approximately 8–9 h.[9] The suit pressure will then drop to 4.3 psia/psid at 100% O_2 until return to Earth, which may take up to 6 d.[9]

Despite a lack of opportunity for preoxygenation, the risk of developing DCS symptoms during a decompressive exposure from 14.7 to 8 psi (approx. MSL to 16,000 ft equivalent altitude) is generally still considered quite low.[18,19] However, it is not fully eliminated and a number of DCS cases have been reported at this altitude.[10,14] In 2013, Butler and Webb presented a review of 111 cases of DCS below 18,000 ft (20 of which were Type II).[1] A hypobaric DCS probability model derived by NASA estimated a 5% DCS altitude threshold (A_{05}) between 13,000 and 16,500 ft for a 4-h exposure (95% confidence interval), although noted uncertainty of this estimate resulting from a particular paucity of data for lower altitude exposures in their source database.[6] Another model, fitted to previously derived experimental data of DCS at lower altitudes, estimated an A_{05} for a 6-h exposure with mild exercise at slightly higher but comparable altitudes (17,000–18,000 ft) and an absolute DCS threshold of 11,000 ft,[10] which is relatively much lower than NASA's no prebreathe no-DCS ascent altitude of 14,500 ft.[7] The ADRAC model predicted a 17% cumulative DCS risk at 18,000 ft (compared to 13 $\pm$ 12% from the actual data), but this was with heavy exercise.[13] Venous Gas Embolism (VGE; including severe, Grade III and IV – Spencer scale) can certainly be expected at this altitude, particularly with no prebreathe, increased exercise induced by self-donning of the pressure suit, and – if onboard O_2 breathing masks are not employed during depress – the high initial N_2 concentration of the initial cabin mixture.[12,19] The zero-preoxygenation VGE threshold altitude is considered to be around 9.5 psi (11,500 ft).[11] It is important to note that the expected 8- to 9-h exposure without denitrogenation and subsequent 4.3 psia exposure for a potentially protracted duration (up to 144 h) exceeds a significant majority of the exposure durations found in the data of the studies cited above. Furthermore, DCS symptom acquisition is inherently probabilistic, demonstrates multivariable dependency, and individual variability. It was therefore considered sufficient for the purposes of this study to proceed under the assumption that DCS risk remains present even if the unrecoverable cabin leak scenario falls within the mission design specification (for a 0.25-in hole). Consideration of contingency management strategies are therefore important, particularly if LOA were to occur during Earth-Moon transit where definitive management options may be up to 6 d away.

The contingency pressure suits represent a possible option, in a manner similar to the current EMU treatment strategy for ISS. This work endeavored to provide preliminary insight into the potential efficacy of using the OCSS pressure suit to treat DCS in this paradigm. Given the pressure suit capability to provide the three therapeutic pillars of DCS management, pressure, oxygen, and time, it was anticipated that treatment will be plausible in certain cases and that additional capability may expand this treatment envelope, but with potentially significant engineering and operational implications.

METHODS

Analysis of DCS treatment feasibility was conducted through computational modeling of several scenarios where symptoms arise following LOA, with various cabin depress and suit function profiles. Case selection was based on relevance to modern operations (e.g., Orion and OCSS), realism of treatment (i.e., nonsevere/rapid decompressions), yet where DCS risk remains nonzero as discussed. Although a necessary part of a holistic approach to DCS risk mitigation, probabilistic DCS risk estimation was considered outside of the scope of this work.

The following case scenarios of LOA-induced DCS were considered:

1. A permanent LOA secondary to a 0.25-in leak (unrecoverable cabin leak);
2. A transient LOA secondary to a 0.25-in hole (with and without subsequent suit psid). Following hole repair, suit maintains the maximum 8 psid within re-established 14.7 psi cabin (22.7 psia or 1.54 ATA); and
3. A permanent LOA with early ($t = 2$ h) suit overpressurization (12 psid/psia).

This first case considers baseline Orion contingency operations in the event of a 0.25-in hole, as described previously. This case provides insight into the therapeutic contribution of oxygen and time, and serves as an appropriate reference point from which to compare subsequent cases. Case 2 investigates the influence of relative hyperbaric therapy achievable following leak repair (while maintaining psid within the OCSS design specifications). Finally, Case 3 simulates a permanent LOA, with suit overpressurization (exceeding the current OCSS specification) 2 h following LOA.

Probability of DCS symptom resolution (P(SR)) was estimated using the previously derived Hypobaric DCS Treatment Model.[2,3] This model (shown in Eq. 1) was derived empirically from NASA prebreathe test data acquired between 1983 and 2014. A log-logistical regression was applied to 154 symptoms (17 of which were Type II) with their resolving pressures and several additional potential explanatory variables, among which ambulation during hypobaric exposure (AMB) and time from the beginning of the exposure to the onset of symptoms (T_s) were found to be significant predictors of treatment success.

$$P(SR) = \frac{1}{1 + e^{\left(\frac{-\ln\Delta P - 1.510 + 0.795 \cdot AMB - 0.00308 \cdot T_s}{0.478}\right)}} \qquad \text{Eq. 1}$$

Where AMB is a binary variable indicating if ambulation occurred or not during decompression ($1 = \text{yes} \mid 0 = \text{no}$), and T_s is time to symptom onset. The effective treatment pressure, ΔP is calculated by Boyle's law ($P_1 * \frac{V_1}{V_2} - P_1$), where P_1 and V_1 are the pressure and dry-gas bubble volume, respectively, at the decompression dose (determined by the peak bubble size, described below) and V_2 is the dry-gas bubble volume at a time following treatment, when P(SR) is calculated. Bubble volumes were determined by a Three Region Well-Stirred Tissue (3RWT) bubble dynamics model (described below), which considers bubble volume recession (and hence treatment) mechanisms of both, direct pressure influences (i.e., bubble compression via Boyle's law), and increased O_2 partial pressure, over time (by virtue of the oxygen window). The validity of the aforementioned prediction method is predicated on the assumption that symptom resolution is the direct result of bubble dissolution.

The biophysical model used here (Eqs. 2 and 3) considers three regions: the gas-containing bubble, the diffusion layer immediately surrounding the bubble, and the outer tissue region.[15] The growth of a spherical bubble is determined by gas-tissue diffusion (Fick's law), ambient hydrostatic pressure (Boyle's law), constant metabolic gases and water vapor tensions, bubble surface tension (Laplace's law), and local tissue perfusion. It is a well-stirred model, referring to the characterization of the outer tissue layer, as it does not retain a gas concentration gradient. Therefore, gas exchange between bubble and tissue occurs only by diffusion across the region immediately surrounding the bubble. This model further assumes ideal gases with a diffusion layer of constant thickness and a stationary, spherical bubble (ignoring convection due to bubble movement and interaction between multiple bubbles). Finally, arterial blood is assumed to be in equilibrium with alveolar gas.

$$\frac{dr_i}{dt} = \frac{\alpha_t D_b \left(\frac{1}{h} + \frac{1}{r_i}\right)(P_t - P_i) - \frac{r_i}{3}\frac{dP_{amb}}{dt}}{P_{amb} + \frac{4\sigma}{3r_i}} \qquad \text{Eq. 2}$$

$$P_i = P_{amb} - P_{idg} + \frac{2\sigma}{r_i} \qquad \text{Eq. 3}$$

Here α_t is the gas solubility in tissue (the Ostwald nitrogen constant was used: 0.0125 cm^3 gas/cm^3 tissue), D_b is the boundary layer diffusion coefficient (2.2×10^{-8} cm^2/s), h is the boundary layer thickness (3.0×10^{-4} cm), r is the bubble radius (cm), P_{amb} is the ambient pressure (dyne/cm^2), P_{idg} is the metabolic gas tension (47 mmHg H_2O + 46 mmHg O_2 + 53 mmHg CO_2 = 19,470 dyne/cm^2), σ is the surface tension (30 dyne/cm^2), and t is time (s). P_t is the total inert gas tissue tension (dyne/cm^2), assumed here to be exclusively N_2 tissue tension ($P_t = P_{tis}N_2$). Derivation of the expression for $P_{tis}N_2$ follows from the mass balance between gas flux and tissue content with gas transported by perfusion; it therefore varies with local tissue perfusion, N_2 solubility, breathing gas mix (N_2 partial pressure) and arterial N_2 tension. Each scenario was divided into a series of sequential intervals, defined by changes in any of the aforementioned variables. Eq. 4 (Conkin et al.[2,4]) was used to calculate Pt ($P_{tis}N_2$) within each sequential interval, and at each integrated time step of the numerical 3RWT bubble model solver.

$$P_{tis}N_2(i) = P_{a(i-1)} + \left(P_{ai} - P_{a(i-1)}\right)\left(1 - e^{-k_i \Delta t_i}\right) + s_i \Delta t - \frac{s_i}{k_i}\left(1 - e^{-k_i \Delta t_i}\right) \qquad \text{Eq. 4}$$

Where P_a is ambient (or breathing gas) partial pressure of nitrogen and s_i is the average rate of change of P_a. As mentioned, nitrogen tissue washout is proportional to perfusion, captured by the rate constant k.

$$k_i = \frac{e^{\lambda \dot{V}_{O_2 i}}}{519.37} \qquad \text{Eq. 5}$$

Where $\dot{V}_{O_2 i}$ is the oxygen consumption (ml $O_{2(STPD)}$/kg/min), a surrogate marker of cardiac output and thus systemic perfusion, which is assumed proportional to tissue perfusion.

The value of the denominator is specific to the 360 min half-time compartment, where k = ln(2)/360 = 1/519.37 while at rest ($\dot{V}_{O_2 i} = 0$). The λ constant was kept at 0.03, the value derived during prior prebreathe testing analysis.[4] Note that the 360 min half-time tissue compartment represents one of numerous possible statistical surrogate constructs used for this purpose. The 360 min compartment was used as, in addition to being shown previously to optimize decompression dose to DCS and VGE,[17] it was found to provide the highest prediction of Type II DCS secondary to LOA, representing the most conservative estimate in that analysis.[8]

The preceding system of equations was programmed into a numerical analysis environment (MatlabR2018b). The first order nonlinear differential equation (Eq. 3) has no closed-form solution, it was therefore solved numerically using a 4th and 5th order Runge-Kutta solver (Dormand-Prince method). Outputs from the bubble model used here (derived by Srinivasan et al.[15]), were compared with publicly available simulation examples from other (empirically validated) models, which demonstrated close correlation. Although no formal validation was conducted, this model was considered sufficient for the purposes of this feasibility study (further addressed in the discussion section).

RESULTS

The ratio of final bubble radius to initial micronuclei radius (3 microns), referred to here as the Bubble Radius Ratio (BRR), was used to track bubble growth and determine decompression dose (indicated by the peak BRR during a particular decompressive exposure).

Case 1

Initial conditions: 14.7 psia, 80/20 (N_2/O_2), not suited.

Event sequence: A 0.25-in hole vents cabin atmosphere, simulated ascent to 8 psia over 6 min, where it is held for 1 h by the vehicle as the crew dons suit and long-duration assembly. The suit maintains an appropriate pressure differential to sustain 8 psia for a period up to 8 h. At T_s = 105 mins, one crewmember complains of DCS-like symptoms.

Maintaining 8 psia at 100% F_iO_2 following LOA resulted in an eventual halt and regression of bubble growth (**Fig. 1**). At 8 h, BRR_{2a} = 16.72 was reduced from BRR_{1a} = 20.61 (decompression dose). V_1 = 998,098.82 μm^3, P1 = V_2 = 528,794.11 μm^3, ΔP = 7.1. T_s = 105 mins, with ambulation (AMB = 1), *P*(SR) = 87%. If cabin atmosphere is not restored and psia dropped to 4.3 psia, bubble growth returned, but again eventually slowed and regressed over time. BRR_{2b}=24.87 was reduced from BRR_{1b} = 32.1 (decompression dose). V_1 = 3,707,043.84 μm^3, P_1 = 4.3 psi, V_2 = 1,740,223.96 μm^3, ΔP = 4.83 psi. Ts = 105 min, with ambulation (AMB = 1), *P*(SR) = 75% at 21 h. If ambulation did not occur, a ΔP of 4.83 psi results in a *P*(SR) of only 37% for 8 h.

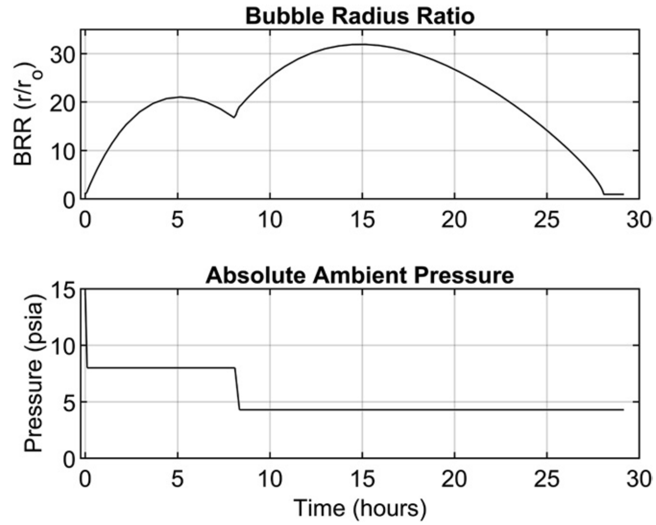

Fig. 1. BRR and absolute pressure profile during the first 30 h period following Orion cabin depressurization from a 0.25-in hole with nominal pressure suit contingency operations.

Case 2

Following the same initial conditions as Case 1, if the leak is repaired and cabin atmosphere restored within the 8-h period, staying in the suit and continuing to maintain maximum suit pressure differential (of 8 psid) results in the delivery of hyperbaric treatment (psia = 22.7) without violating the current OCSS specification (note that the gas mixture in the suit is air (80/20), as 100% O_2 at hyperbaric pressures introduces flammability concerns). With this strategy, the bubble model predicts complete bubble dissolution (and therefore *P*(SR) approaching 100%) within just under 2 h (t = 9 h 48 min) following cabin leak repair (**Fig. 2**), as opposed to within 3 h (10 h 36 min) when the suit is not used (difference of approx. 48 min) following restoration of cabin atmosphere (**Fig. 3**).

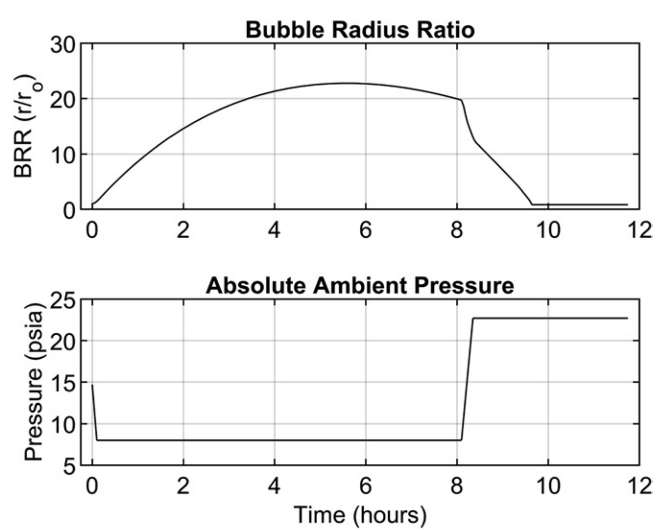

Fig. 2. Cabin leak repair at 8 h with hyperbaric therapy delivery (air) using the OCSS pressure suit within a restored cabin atmosphere.

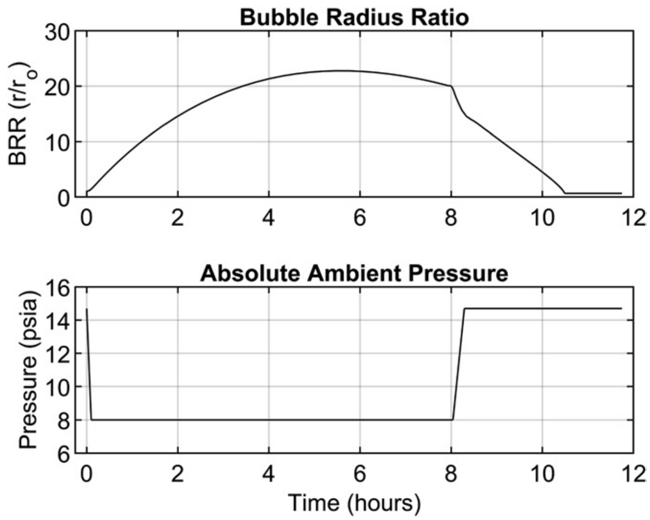

Fig. 3. Cabin leak repair at 8 h with cabin atmosphere restored and no suit psid applied.

Case 3

Although beyond the current Orion suit specifications, this case investigates the potential efficacy of early suit over-pressurization. Again, equivalent initial conditions as the prior cases, with suit overpressurization (12 psid/psia) initiated at approximately 2 h following LOA. Pressure was held there indefinitely for the purposes of this simulation to ascertain the time at which complete bubble dissolution occurs. As shown in **Fig. 4**, this occurred prior to t = 6 h, with *P*(SR) exceeding 95%.

DISCUSSION

The primary objective of this work was to conduct a preliminary investigation into the potential utility of a launch/entry pressure suit for treating DCS, with specific considerations to the Orion vehicle, operational concept, and OCSS suit. Although only a small number of scenarios were presented here, they illustrate several important concepts and provide considerations for further analysis of this potential contingency option. It is important to note that all tissue gas bubbles, and hence all acute Type I DCS symptomatology, will generally eventually resolve (by virtue of the oxygen window).[16] The nitrogen washout demonstrated in Case 1 (Fig. 1) shows BRR remaining elevated for a prolonged period, with eventual dissolution (> 24 h) while breathing 100% O_2 despite remaining at 4.3 psi. Having DCS symptoms during this prolonged period presents risks with potential mission impact, including discomfort, human performance implications, and concerns of long-term sequelae. Cases 2 and 3 illustrate the benefit of increased barometric pressure on bubble dissolution and DCS resolution.

Case 2 considers a plausible scenario and highlights the utility of a launch/entry pressure suit as an off-nominal contingency option in DCS management, particularly if the defect is transient. Upon repair of the hole, the suit differential pressure can be harnessed within a re-established atmosphere to provide hyperbaric therapy. This treatment would be available well before definitive care would otherwise be accessible. This treatment modality would also be possible in the Earth-return scenario, if a crewmember continued to suffer DCS symptoms postlanding and it may be several hours or more before retrieval.

As expected, early suit over-pressurization in Case 3 resulted in early bubble regression and increased *P*(SR). It is certainly not advisable to pressurize the suit beyond the specification in the current paradigm; however, this case provides a preliminary quantification of the potential benefit of expanding the suit capabilities and identifies a potential area warranting further consideration. Higher than nominal pressures could also be achieved within suit spec by several other means. One, early suit donning (within the 1-h period while Orion maintains pressure), with application of 8 psid (16 psia), exploiting residual cabin pressure. One issue with that option in this particular analysis, however, is that symptoms rarely occur within this time frame and therefore would represent "prophylactic" hyperbaric treatment, which was not included in the DCS treatment model.[2] This option is better suited to a risk assessment model. The second means of achieving hyperbaric therapy would be through expanding the Orion vehicle's 1 h "feed-the-leak" capability. Future work will analyze these scenarios.

Several engineering design considerations should be noted with regards to the application of the latter two cases. It is important to consider the current parallel arrangement of the suit loop. The result is that pressurization (or over-pressurization) of one suit requires pressurization of all the suits – it is not possible to isolate one suit while multiple are in use (note: if cabin atmosphere is restored, an individual suit may be used, the self-sealing valves at the suit loop to the loop close with removal of the suits). Depending on length of treatment, this can severely limit the function and comfort of the other crewmembers. Furthermore, mobility will be somewhat limited while pressurized, rendering leak repair efforts more difficult. Pressurization of the suit over a breathable cabin atmosphere would likely be reserved until crewmembers have repaired the leak and the

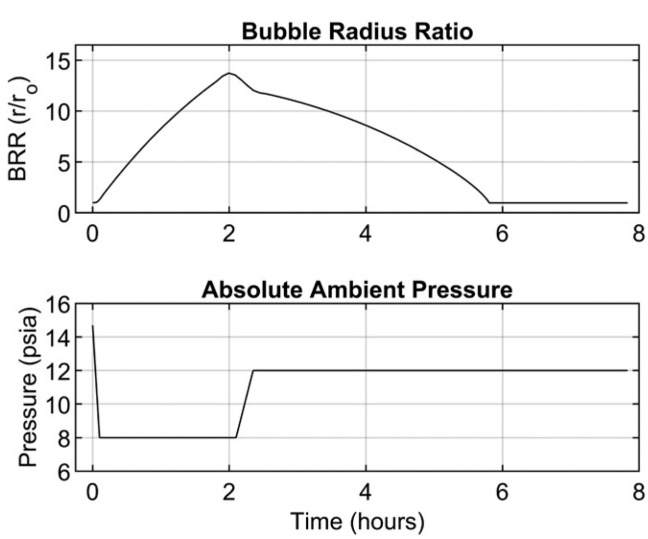

Fig. 4. Suit over-pressurization (12 psid/psia) at 2 h following LOA.

vehicle was returned to a nondynamic state. Finally, structural limitations must be considered. The OCSS suit has been designed to operate nominally at 8 psid, and as such its structural design can handle much higher pressures.[9] It is possible that this structural capability of the suit could be used for DCS treatment, however this would require further analysis and formal validation and acceptance testing. Finally, flammability concerns are readily apparent with elevated oxygen environments. While pressurizing the suit above the cabin atmosphere would potentially lower DCS risk, it would increase flammability risk, particularly at higher oxygen concentrations. The closed-loop nature of the OCSS system would ensure that the cabin oxygen concentration remains within prescribed limits.

The 3RWT bubble dynamics model used here makes several assumptions that do not follow a true mechanistic model. First, a diffusion layer was assumed a constant (and relatively arbitrary) thickness; second, an abrupt change in gas flux from diffusion layer to area just beyond is modeled; third, the resulting model requires violation of Henry's law under the assumption of equal gas fluxes despite ambient pressure changes (as noted by the original author[15]); and finally, the computed bubble volume (and corresponding version of Boyle's law used here) are referenced to ideal, dry-gas conditions. This includes the contribution of water vapor, as opposed to considering it as a true liquid dispersion or wet-gas, such as defined under body temperature and pressure saturated (BTPS) conditions. Furthermore, the interaction between bubbles is not modeled and this model was a generalization that neglected tissue viscoelasticity. Parameterization was performed through literature review and no formal sensitivity analysis, empiric validation, numeric verification, or uncertainty quantification was conducted. Another limitation to this study is the application of the hypobaric DCS treatment model to the particular cases used here. Specifically, this model was derived based on a set of symptom and treatment data that differ from the suite of treatment options proposed here. New data is required to validate the model under these specific protocols (which will not realistically be available for many years).

General improvements to overall simulator fidelity are also planned for future work. The computer modeling conducted for this work provides a platform upon which to develop a design trade-off analysis tool. Future work will focus primarily on improving the validity and fidelity of the modeling tool and greatly expand the case scenarios investigated. There are a number of immediate next steps, which include: refinement and validation of the tissue-bubble model (incorporating wet-gas volume referenced to BTPS conditions and possibly including tissue viscoelasticity), development of a cabin depressurization model using Bernoulli equation with Sonic Orifice assumption (depress to vacuum), inclusion of more representative transient values (rate of change of gas-mixtures, time to depress/repress that incorporate specific OCSS suit-loop purge efficiency, etc.), and consideration of consumables. Eventually, the holistic approach for DCS risk mitigation will include combining probability of DCS risk estimates with treatment efficacy predictions with the aim of informing design trade-off. Further considerations may also include other or adjuvant treatment options (e.g., fluids, lidocaine, NSAIDs, etc.). Lastly, the authors would like to generalize this tool to non-Orion vehicles and suits.

While emphasis must continue to be placed on rigorous preventative measures enabled through engineering and administrative controls, treatment options are an important element of the holistic approach to DCS risk mitigation. The analysis presented here supports the hypothesis that pressure suits may offer utility in treating DCS resulting from spacecraft cabin decompression in certain scenarios.

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