SHORT COMMUNICATION

# Variability in venous gas emboli following the same dive at 3,658 meters

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## ABSTRACT

Exposure to a reduction in ambient pressure such as in high-altitude climbing, flying in aircrafts, and decompression from underwater diving results in circulating vascular gas bubbles (i.e., venous gas emboli [VGE]). Incidence and severity of VGE, in part, can objectively quantify decompression stress and risk of decompression sickness (DCS) which is typically mitigated by adherence to decompression schedules. However, dives conducted at altitude challenge recommendations for decompression schedules which are limited to exposures of 10,000 feet in the U.S. Navy Diving Manual (Rev. 7). Therefore, in an ancillary analysis within a larger study, we assessed the evolution of VGE for two hours post-dive using echocardiography following simulated altitude dives at 12,000 feet. Ten divers completed two dives to 66 fsw (equivalent to 110 fsw at sea level by the Cross correction method) for 30 minutes in a hyperbaric chamber. All dives were completed following a 60-minute exposure at 12,000 feet. Following the dive, the chamber was decompressed back to altitude for two hours. Echocardiograph measurements were performed every 20 minutes post-dive. Bubbles were counted and graded using the Germonpré and Eftedal and Brubakk method, respectively. No diver presented with symptoms of DCS following the dive or two hours post-dive at altitude. Despite inter- and intra-diver variability of VGE grade following the dives, the majority (11/20 dives) presented a peak VGE Grade 0, three VGE Grade 1, one VGE Grade 2, four VGE Grade 3, and one VGE Grade 4. Using the Cross correction method for a 66-fsw dive at 12,000 feet of altitude resulted in a relatively low decompression stress and no cases of DCS.

## INTRODUCTION

Diving at altitudes above sea level are conducted for recreation, research, commercial and military operations [1]. Diving at altitude presents additional challenges to decompression algorithms due to augmented pressure difference between that at depth and the surface. Revision 6 of the U.S. Navy Diving Manual was published in 2008 and was the first to include conversion tables for altitude dives up to 10,000 feet, which remains the upper limit of diving at altitude according to the U.S. Navy Diving Manual, Rev. 7 [2]. Recommendations for conducting diving operations and appropriate decompression schedules can still be obtained above 10,000 feet using the U.S Navy Altitude Diving Procedures (i.e., Cross correction for sea level equivalent). However, validation of the adaptation of these methods above 10,000 feet has been limited to few field and laboratory-controlled studies [1].

Dive tables and decompression algorithms are used to mitigate the risk of decompression sickness (DCS), which are based on probabilistic models developed from operationally relevant man-dives. DCS has a complex pathogenesis which results, in part, from inert gas bubbles during or after decompression from hyperbaria (e.g., diving) or acute exposure to hypobaria [3]. Generally, these gas bubbles are present in venous circulation (i.e., venous gas emboli [VGE]), are benign in nature, and are filtered by the lungs. VGE incidence and severity carries a DCS risk of ~10% for diving decompression (i.e., weak positive predictor), while the absence of VGE is a strong, negative predictor of DCS risk [3,4]. Despite evidence of a relation between VGE and DCS [5,6], caution should

KEYWORDS: altitude; decompression illness; decompression stress; intravascular gas bubbles

be taken when extrapolating VGE and DCS risk [7]. However, the use of VGE measurements represents one of the only current objective measures of decompression stress and could be used rather than an incidence of DCS as the study endpoint [4,8] despite its current limitations [7].

Therefore, the purpose of this study was to assess decompression stress following a single dive conducted from a simulated altitude of 12,000 feet by measuring the incidence and severity of VGE using echocardiography. The present study was conducted as an ancillary analysis from a larger study examining the effects of diving at altitude and the efficacy of respiratory muscle training on exercise ventilation during a single, no-stop dive to 66 feet of seawater (fsw)[9].

## METHODS

### Subjects

Ten men participated in the study. Subjects were either certified recreational divers or were experienced with hyperbaric and/or hypobaric exposure. Prior to the experimental dives subjects completed a health history questionnaire and a diver physical examination with a study physician. Subjects were excluded for any neurologic, metabolic, pulmonary or cardiovascular disease. Subjects self-reported to be non-smokers and not currently taking any medications that are known to alter pulmonary, cardiovascular, or neurologic function. Prior to beginning the experimental trials subject anthropometrics (i.e., body height, mass, and three-site skin folds) were measured. Subjects also completed a maximal exercise test ($\dot{V}O_{2peak}$) on a cycle ergometer to assess cardiorespiratory fitness and establish workload during the experimental dives. Subject characteristics are described in Table 1. Additionally, subjects completed a brief familiarization dive in the hyperbaric chamber to ensure that they could comfortably clear their ears with changing depth. Ethical approval was granted by the Institutional Review Board at the University at Buffalo and the study performed in accordance with the Declaration of Helsinki.

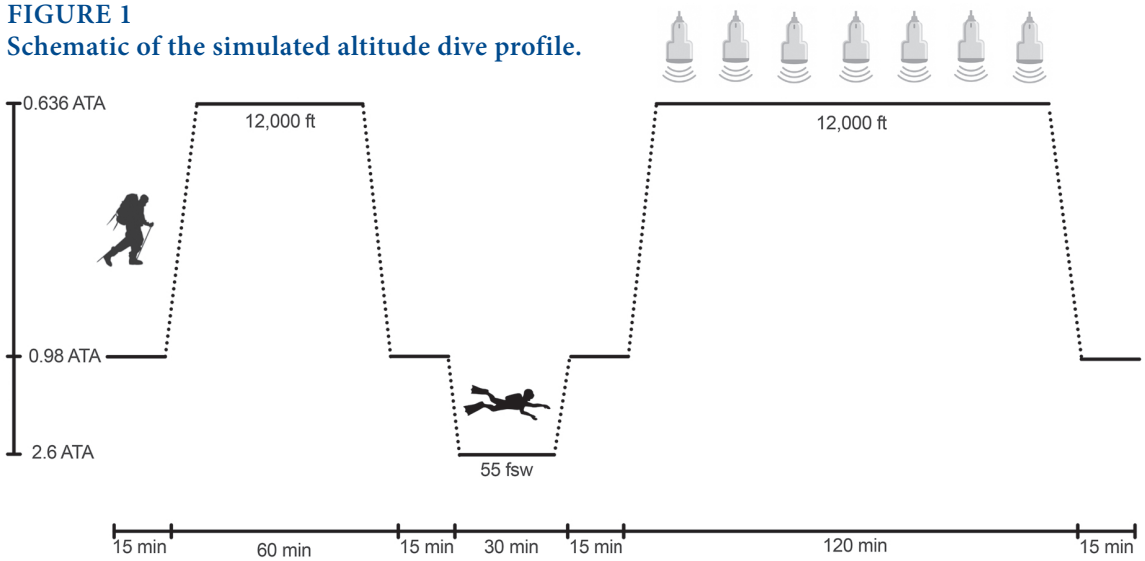
**Table 1. Bubble grades according to the Eftedal and Brubakk Scale**

| Grade | Description |
|---|---|
| 0 | No bubbles |
| 1 | Occasional bubbles |
| 2 | At least one bubble per four cardiac cycles |
| 3 | At least one bubble per cardiac cycle |
| 4 | Continuous bubbling |
| 5 | 'White out'- individual bubbles are indistinguishable |

### Dive protocol

Subjects completed three dives: a single dive to 55 fsw from ground level (i.e., control dive) and two simulated altitude dives from 473 mmHg (0.636 atmospheres

**FIGURE 1**
**Schematic of the simulated altitude dive profile.**




Divers were decompressed from ground level (0.98 ATA) to altitude (0.636 ATA) for 60 minutes prior to recompression to ground level (15-min. stop) and compression to depth (2.7 ATA) for 30 minutes. Following the dive, divers were decompressed to ground level (15-min. stop) followed by decompression back to altitude for 120 minutes where Doppler ultrasound measurements were completed every 20 minutes.

absolute (ATA) or 12,000 feet above sea level; Figure 1). The first two dives (i.e., the control dive and first simulated altitude dive) were separated by at least seven days. The final simulated altitude dive was completed following four weeks of resistance respiratory muscle training (~5 weeks following the first simulated altitude dive). No subjects were exposed to hypo- or hyperbaria outside of the experimental trials during the duration of the study protocol. To simulate the altitude exposure the hypobaric chamber was decompressed at a rate of ~1,000 feet per minute. Subjects rested supine for a 60-minute altitude exposure before compressing back to ground level for a brief (~15-minute) surface interval to allow for the chamber to be prepared to dive. All dives were initiated from 744 mmHg (~ 600 feet above sea level) in the hyperbaric chamber. The dry hyperbaric chamber was pressurized to 1,292 mmHg gauge pressure relative to ground level, to simulate a depth of 2.6 ATA (55 fsw) for 30 minutes.

It is important to note that the simulated altitude dives consisted of two independent descents. First, subjects were recompressed from 0.636 ATA to 0.98 ATA (~11 fsw equivalent) to reconfigure the chamber from hypo- to hyperbaric use. Following a 15-minute stop at 0.98 ATA. The chamber was pressurized to descend at a rate of 75 feet per minute to 55 fsw (i.e., second descent). Therefore, the actual operating depth (relative to altitude) was 66 fsw. Furthermore, the equivalent depth at altitude was calculated in accordance to the U.S. Navy Diving Manual [2] and based on the Cross correction equation for sea level equivalent [10] from 0.98 ATA (i.e., ground level) to 0.636 ATA (12,000 feet) where:

$$\textit{Equivalent Depth at Altitude}\ (103.7\ \textit{fsw}) = \textit{Depth at altitude}\ (66\ \textit{fsw}) \times (\textit{Barometric Pressure at Sea Level}\ [1\ \textit{ATA}] \div \textit{Barometric Pressure at Altitude}\ [0.636\ \textit{ATA}])$$

The descent rate was slowed or paused if the subject or tenders had difficulty equalizing pressure in their ears. Upon reaching 2.6 ATA, bottom time started and subjects were transitioned to the wet side of the chamber. Subjects were seated in the wet pod (25°C) such that the water was at the level of the clavicle; subjects breathed from a surface-supplied demand regulator (i.e., 21% $O_2$, 78% $N_2$, 0.04% $CO_2$). During the immersion period, subjects cycled on an underwater ergometer at a moderate intensity (i.e., 55% $\dot{V}O_{2max}$) for 20 minutes before being transitioned back to the dry pod for ascent. Periodically, the chamber was vented while chamber pressure was maintained as needed to prevent the subjects from being exposed to a hypercapnic environment. The chamber was decompressed to ascend at a rate of 30 feet per minute. Immediately after exiting the chamber, the subjects were given a neurologic assessment to screen for signs and symptoms of DCS. Following a brief surface interval at ground level (<20 minutes) to reconfigure the chamber for hypobaria, subjects re-entered the chamber to be decompressed back to altitude for two hours to assess VGE.

### Measurements

For the single no-stop dive from ground level, echocardiograph measurements were made pre- and immediately post-dive. For the simulated altitude dives, echocardiograph image acquisition were made pre-dive (during the surface interval between the altitude exposure and the dive) and post-dive at altitude every 20 minutes for two hours. Images were acquired initially at rest, but then completed following single upper and lower limb movements. During movement periods, the subject flexed and extended the selected limb five times to mobilize intravascular gas bubbles presumably lodged in the venous pathway. Sonography for the detection of intravascular bubbles was performed by the same operator in all visits via adult cardiac Doppler ultrasound (GE Vivid-Q, Chicago, Illinois, U.S.) using a left lateral decubitus position. An apical four-chamber view echocardiography recording (2D live acquisition mode, phased-array transducer 1.7-3.4 MHz, 70.3 frames per second) was acquired for each time point to evaluate circulating intravascular gas bubbles. Echocardiography videos comprised 10 cardiac cycles and were saved for post-processing. The number of bubbles were counted on 10 consecutive cardiac cycles and averaged to account for beat-to-beat bubble variation according to the methods proposed by Germonpré et al. [11]. Furthermore, bubbles were graded based on the methods of Eftedal and Brubakk (Table 1) [12].

### Data and statistical analysis

For each time point, VGE were assessed independent from the sonographer by the study physician (ESJ) per the methods of Germonpré et al. [11] and Eftedal and Brubakk [12]. Intravascular gas bubbles were counted and graded for measurements completed before and after the control dive that was conducted prior to the respiratory muscle training intervention. Because the two simulated altitude dives were conducted pre- and post-respiratory muscle training, intravascular gas bubbles

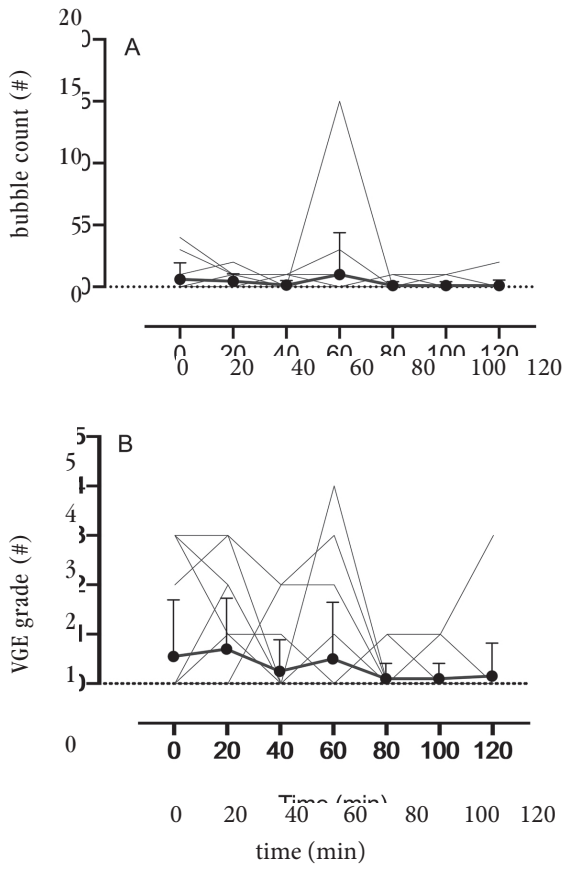



**FIGURE 2**

Venous gas emboli (VGE) analysis during a two-hour post-dive simulated altitude exposure at 3,658 m. Bubble count (A) and VGE grade (B) were quantified using the Eftedal and Brubakk scale every 20 minutes post-dive. Individual values (gray lines; n=20) are plotted with mean and SD (solid black line). Data were analyzed using a repeated measures one-way ANOVA.

were counted and graded within-subject (training effect) and time point (time effect). A two-way, repeated measures analysis of variance (ANOVA) was completed to determine if there was an effect of respiratory muscle training on VGE count and grade. Data were analyzed on Prism (Version 8.4, GraphPad Software Inc., La Jolla, California, U.S.). Significance was set a priori at an alpha level of 0.05.

## RESULTS

A total of 10 control dives (i.e., from ground level) were performed, with all 10 resulting in no intravascular bubbles counted and subsequently, all with VGE Grade 0 (Table 2). There were a total of 20 dives at altitude (pre-RMT n =10; post-RMT n=10). Mean (SD) and individual bubble counts and scores are presented in Figures 2A and 2B, respectively. There was no main effect of time at altitude (p=0.36), effect of respiratory muscle training (p=0.39), or interaction (p=0.39) on intravascular bubble count. Additionally, there was no main effect of time at altitude (p=0.14), effect of respiratory muscle training (p=0.56), or interaction (p=0.87) for VGE grade.

Given that there was no effect of respiratory muscle training on intravascular bubble count or VGE score, all altitude dives were grouped to assess incidence and severity of VGE grade and the time associated with the onset and peak VGE grade (Table 2). There were no cases of DCS symptoms in any of the control or altitude dives. Similar to that of Cialoni et al. [13], after divers were graded they were divided into three groups (Table 3): 1) non-bubblers; 2) variable bubblers; and 3) bubblers. Non-bubblers were those divers who never developed VGE and presented a peak VGE Grade 0 during both simulated altitude dives. Variable bubblers were divers who presented a peak VGE Grade >1 in one of two simulated altitude dives. Finally, bubblers were divers who presented a peak VGE Grade >1 in both simulated altitude dives.

## DISCUSSION

The present study was an ancillary analysis of VGE for two hours following dives conducted from a simulated altitude. Each subject completed a single dive to 55 fsw for 30 minutes from ground level (i.e., control dive) and two dives to 66 fsw from a simulated altitude of 12,000 feet. The primary finding was that the adaptation (from the upper limit of 10,000 feet) of the U.S Navy Altitude Diving Procedures (i.e., Cross correction for sea-level equivalent) for a simulated altitude dive at 12,000 feet resulted in relatively low decompression stress. This is highlighted by the majority of dives (55%) presenting with a peak VGE Grade 0 (range: 0-4) and no dives resulting in symptoms of DCS. A secondary finding was that five of 10 subjects presented with a peak VGE Grade 0 on one of the simulated altitude dives, while presenting with a peak VGE Grade >1 from the same altitude dive exposure (i.e., variable bubblers).

**TABLE 2**

| dive profile (# exposures) | VGE grades (E&B* scale) | peak post-dive (# dives [%]) | peak during ALT (# dives [%]) | onset time any VGE grade (min [# dives]) | onset time peak VGE grade (min [# dives]) | VGE resolution from first onset (min) |
|---|---|---|---|---|---|---|
| **17m/30min dive only (n=10)** | 0 | 10 [100%] | - | - | - | - |
| | 1 | | | | | |
| | 2 | | | | | |
| | 3 | | | | | |
| | 4 | | | | | |
| | 5 | | | | | |
| **60 min ALT + 17m/30min dive + 120min ALT (n=20)** | 0 | 16 [80%] | 11 [55%] | 13(14) min | 31(37) min [9 dives] | 66 (36) min [9 dives] |
| | 1 | | 3 [15%] | | | |
| | 2 | 1 [5%] | 1 [5%] | | | |
| | 3 | 3 [15%] | 4 [20%] | | | |
| | 4 | | 1 [5%] | | | |
| | 5 | | 0 [0%] | | | |

Venous gas emboli (VGE) analysis before and after a single no-stop dive to 17 meters from ground level and during a two-hour post-dive simulated altitude exposure (ALT) at 3,658 meters following a single no-stop dive at altitude.

* Eftedal & Brubakk

**TABLE 3. VGE grade and bubble susceptibility classification**

| subject | classification | time point | 0 | 20 | 40 | 60 | 80 | 100 | 120 |
|---|---|---|---|---|---|---|---|---|---|
| **1** | Bubbler | Dive 1 | 3 | 3 | 2 | 3 | 0 | 0 | 0 |
| | | Dive 2 | 3 | 1 | 0 | 4 | 0 | 0 | 0 |
| **3** | Non-Bubbler | Dive 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| ***4*** | Variable | Dive 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 1 | 0 | 1 | 0 | 0 | 0 |
| **6** | Variable | Dive 1 | 2 | 3 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| **7** | Non-Bubbler | Dive 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| **8** | Non-Bubbler | Dive 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| **9** | Variable | Dive 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 1 | 0 | 0 | 0 | 0 | 0 |
| **10** | Variable | Dive 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |
| | | Dive 2 | 0 | 1 | 1 | 0 | 0 | 0 | 0 |
| **11** | Bubbler | Dive 1 | 0 | 2 | 0 | 0 | 1 | 0 | 0 |
| | | Dive 2 | 3 | 2 | 0 | 0 | 1 | 1 | 0 |
| **12** | Variable | Dive 1 | 0 | 0 | 2 | 2 | 0 | 1 | 3 |
| | | Dive 2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 |

**Venous gas emboli (VGE) analysis during a two-hour post-dive simulated altitude exposure at 3,658 m.**

VGE grades were quantified using the Eftedal and Brubakk scale every 20 minutes post-dive.

Divers were divided in three groups: 1) Non-bubbler (n=3); 2) Variable bubbler (n=5); and 3) Bubbler (n=2).

Similar to Papadopoulou, et al. [14], we observed both inter- and intrasubject variability in the incidence and severity of VGE, as well as the time course of the detection of and peak VGE (Table 2). It is interesting to highlight that there was intrasubject variability of the detection of and peak VGE. Despite having undergone the same altitude dive exposure, subjects presented with a range of decompression stress. We observed that some subjects (2/10) presented with detectable bubbles on each of the simulated altitude dives, while others (3/10) never presented with any detectable bubbles. The underlying mechanism that may augment the susceptibility to develop VGE remains unknown [6]. Furthermore, it is important to note that the subjects in the present study were all young, healthy, fit men, which are characteristics that have been shown to result in fewer bubbles compared to older, heavier or less fit divers [15]. Therefore, there was no clear phenotype within the present study that would inherently make one diver more prone to develop VGE.

VGE have long been the target of correlation for the development and risk of DCS. However, the presence of VGE alone is a poor surrogate for the incidence of DCS when testing probabilistic models. Typically, decompression tables are tested by conducting many man-dives with an endpoint of an acceptable probability of developing DCS (e.g., <3%). Moreover, experiments resulting in potentially severe outcomes should be limited in humans. While VGE do not diagnose DCS per se, measurement of VGE could be a standard to assess decompression stress [3,6, 7,14]. Such measurements could be used for validating decompression schedules like in the present study due to the strong negative relation between VGE and DCS [3].

## Considerations

Several experimental considerations are applicable to our study. Caution must be expressed when extrapolating the findings from our simulated altitude dive decompression stress. While chamber-based dives are generally experimentally controlled (relative to pool or open-water experiments), there are complications in determining the dive profile when simulating altitude dives in a hypo- and hyperbaric chamber. The Navy Diving Manual (Rev. 7) provides two methods for accounting for the augmented susceptibility to DCS after an altitude dive [2]. First, the increase in the fractional change in inert gas partial pressure is augmented when diving at altitude. This is addressed by using the Cross correction for sea-level equivalent. This method sets the operating depth to use for the dive tables and decompression schedules. Second is the equilibration of tissue inert gas to the new altitude. This is generally accepted to be complete 12 hours after ascent. For dives initiated within the 12 hours a repetitive group designator is assigned to a diver depending on the altitude. Our subjects would be assigned a repetitive group designator 'I' for 10,000 feet (not specified for higher altitudes). Therefore, the first dive at altitude should be considered a repetitive dive. The 'I' repetitive group carries a residual nitrogen time of 23 minutes that would need to be added to the bottom time.

Our simulated altitude dive was not a typical square dive profile (Figure 1), which gives rise to two considerations. First, if bottom time is calculated from the time the subjects were recompressed from altitude, the actual bottom time would be ~46 minutes (i.e., one-minute descent from altitude, 15-minute stop at ground level, and 30 minutes at depth). Additionally, if the subjects were designated as 'I' repetitive divers, an additional 23 minutes of residual nitrogen time would need to be added to the actual bottom time of 46 minutes (i.e., 69 minutes total bottom time). Second, we omitted the initial descent (from altitude to ground level) in calculating the actual and sea level equivalent depths, so our working depth was actually 66 fsw (110 fsw sea-level equivalent). Importantly, however, the stop at 0.98 ATA during compression likely reduced nitrogen uptake compared to a square dive, which may have attenuated the decompression stress following the dive. Utilizing the decompression schedules for a 110-fsw and 70-minute bottom time in the U.S. Navy Diving Manual (Rev. 7), divers would have had two decompression stops: 1) 26 minutes at 30 fsw; and 2), 155 minutes at 20 fsw. However, our divers completed a 15-minute decompression stop at 11 fsw, relative to altitude while the chamber was reconfigured for hypobaria.

Third, the dive protocol was conducted in both dry (descent and ascent) and wet (bottom time) conditions. Although unlikely, we cannot rule out that the transition from wet to dry conditions altered decompression. Furthermore, the dive was conducted in thermoneutral conditions, which is unlikely to occur while diving at altitude, as inland diving is primarily cold, fresh water.

Moreover, when conducting VGE measurements at altitude, subjects were instructed to conduct coordinated, single limb movements. However, the Eftedal and Brubakk (E&B) method was not designed to assess VGE following movement [5,16]. If the movements

resulted in showers of VGE compared to measurements conducted at rest, we would have observed a much higher incidence and/or severity of VGE grades. Therefore, it is unlikely that this methodology limits our findings. Fourth, the VGE measurements were completed every 20 minutes at altitude. It is possible that detectable VGE were missed in those subjects that presented with a peak VGE Grade 0. While we cannot rule this out, it is unlikely that we would have missed all detectable VGE within a two-hour post-dive observation. Previous work has observed the onset DCS after two hours (and up to 24 hours) [17,18]. Importantly, we show one subject with VGE at two hours, indicating that two hours may not be long enough to observe resolution of VGE in all cases. Finally, the VGE grading was completed post hoc, which may be a limitation in translatability to practical application of identifying and quantifying decompression stress. However, manual grading of VGE in real time is cumbersome and impractical. Future machine learning applications for VGE counting and grading may offer a solution to a number of these considerations.

## CONCLUSION

The evaluation of VGE with echocardiography following decompression from a dive conducted at a simulated altitude of 12,000 feet assessed decompression stress by quantifying VGE in venous and arterial circulations. By extrapolating the U.S. Navy Diving Manual, Rev. 7 Cross correction for equivalent sea level depth, we conducted a wet dive in a hyperbaric chamber from a simulated altitude, which resulted in no cases of DCS, no detection of paradoxical arteriovenous shunting, and a relatively low decompression stress highlighted by 11/20 of the man-dives resulting in no detectable VGE. Further research and validation is necessary for repetitive dives or multiday dives utilizing the Cross correction method at altitudes greater than 10,000 feet. ■

***Note:*** *This paper – Hess HW, Wheelock CE, St. James E, et al. Variability in venous gas emboli following the same dive at 3,658 meters. Undersea Hyperb Med. 2021 Fourth Quarter 48(4) 469-476 – is a reprint of the original, which originally appeared in the Second Quarter 2021 issue of the Journal, pages 119-126. Figure 1 in the original article has been replaced with the correct figure in this issue.*

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***Conflict of interest statement***

*The review process and decision to accept was handled by an Associate Editor.*

## REFERENCES

1. Egi SM, Pieri M, Marroni A. Diving at altitude: from definition to practice. Undersea Hyperb Med. 2014;41(6):495-503. Epub 2015/01/08. PubMed PMID: 25562941.

2. NAVSEA. Diving at altitude. US Navy diving manual (revision 7). Washington, DC: United States Department of the Navy; 2016. p. 9.46-56.

3. Mahon RT, Regis DP. Decompression and decompression sickness. Compr Physiol. 2014;4(3):1157-1175. Epub 2014/06/20. doi: 10.1002/cphy.c130039. PubMed PMID: 24944033.

4. Mollerlokken A, Gaustad SE, Havnes MB, Gutvik CR, Hjelde A, Wisloff U, et al. Venous gas embolism as a predictive tool for improving CNS decompression safety. Eur J Appl Physiol. 2012;112(2):401-409. Epub 2011/05/20. doi: 10.1007/s00421-011-1998-9. PubMed PMID: 21594696; PubMed Central PMCID: PMCPMC3258401.

5. Eftedal OS, Lydersen S, Brubakk AO. The relationship between venous gas bubbles and adverse effects of decompression after air dives. Undersea Hyperb Med. 2007;34(2):99-105. Epub 2007/05/25. PubMed PMID: 17520861.

6. Gawthrope IC, Summers M, Macey DJ, Playford DA. An observation of venous gas emboli in divers and susceptibility to decompression sickness. Diving Hyperb Med. 2015;45(1):25-29. Epub 2015/05/13. PubMed PMID: 25964035.

7. Doolette DJ. Venous gas emboli detected by two-dimensional echocardiography are an imperfect surrogate endpoint for decompression sickness. Diving Hyperb Med. 2016;46(1):4-10. Epub 2016/04/06. PubMed PMID: 27044455.

8. Hugon J, Metelkina A, Barbaud A, Nishi R, Bouak F, Blatteau JE, et al. Reliability of venous gas embolism detection in the subclavian area for decompression stress assessment following scuba diving. Diving Hyperb Med. 2018;48(3):132-140. Epub 2018/09/11. doi: 10.28920/dhm48.3.132-140. PubMed PMID: 30199887; PubMed Central PMCID: PMCPMC6205931.

9. Wheelock CE, Hess HW, Stooks J, et al. Respiratory muscle training and exercise ventilation while diving at altitude. Undersea Hyperb Med. 2021; 48(2): 107-117.

10. Bell RL, Borgwardt RE. The theory of high-altitide corrections to the U.S. Navy standard decompression tables. The Cross corrections. Undersea Biomed Res. 1976;3(1):1-23. Epub 1976/03/01. PubMed PMID: 1273981.

11. Germonpré P, Papadopoulou V, Hemelryck W, Obeid G, Lafère P, Eckersley RJ, et al. The use of portable 2D echocardiography and 'frame-based' bubble counting as a tool to evaluate diving decompression stress. Diving Hyperb Med. 2014;44(1):5-13. Epub 2014/04/02. PubMed PMID: 24687479.

12. Eftedal O, Brubakk AO. Agreement between trained and untrained observers in grading intravascular bubble signals in ultrasonic images. Undersea Hyperb Med. 1997;24(4):293-299. Epub 1997/01/01. PubMed PMID: 9444060.

13. Cialoni D, Pieri M, Balestra C, Marroni A. Flying after diving: should recommendations be reviewed? In-flight echocardiographic study in bubble-prone and bubble-resistant divers. Diving Hyperb Med. 2015;45(1):10-15. Epub 2015/05/13. PubMed PMID: 25964033.

14. Papadopoulou V, Germonpre P, Cosgrove D, Eckersley RJ, Dayton PA, Obeid G, et al. Variability in circulating gas emboli after a same scuba diving exposure. Eur J Appl Physiol. 2018; 118(6):1255-1264. Epub 2018/04/05. doi: 10.1007/s00421-018-3854-7. PubMed PMID: 29616324.

15. Carturan D, Boussuges A, Vanuxem P, Bar-Hen A, Burnet H, Gardette B. Ascent rate, age, maximal oxygen uptake, adiposity, and circulating venous bubbles after diving. J Appl Physiol (1985). 2002;93(4):1349-56. Epub 2002/09/18. doi: 10.1152/japplphysiol.00723.1999. PubMed PMID: 12235035.

16. Eftedal O, Brubakk AO. Agreement between trained and untrained observers in grading intravascular bubble signals in ultrasonic images. Undersea Hyperb Med. 1997;24(4):293-299. PubMed PMID: WOS:000071330800005.

17. Francis TJ, Pearson RR, Robertson AG, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: latency of 1070 human cases. Undersea Biomed Res. 1988; 15(6):403-417. Epub 1988/11/01. PubMed PMID: 3067433.

18. Weathersby PK, Survanshi SS, Homer LD, Parker E, Thalmann ED. Predicting the time of occurrence of decompression sickness. J Appl Physiol (1985). 1992;72(4):1541-1548. Epub 1992/04/01. doi: 10.1152/jappl.1992.72.4.1541. PubMed PMID: 1592748.