Altitude Decompression Sickness Between 6858 and 9144 m Following a 1-h Prebreathe

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WEBB JT, PILMANIS AA. *Altitude decompression sickness between 6858 and 9144 m following a 1-h prebreathe.* **Aviat Space Environ Med 2005; 76:34–8.**

Introduction: The zero prebreathe altitude threshold for developing 5% decompression sickness (DCS) symptoms in men has been reported to be 6248 m (20,500 ft). However, such an altitude threshold when 1 h of oxygen prebreathe is used has not been well documented and was the primary purpose of this study. ***Methods:*** The 51 male human subjects were exposed to 9144 m (30,000 ft), 8382 m (27,500 ft), 7620 m (25,000 ft), and/or 6858 m (22,500 ft) for 8 h. They were monitored for symptoms of DCS and venous gas emboli (VGE). ***Results:*** DCS symptom incidence after 4 h of exposure decreased with exposure altitude from 87% at 9144 m to 26% at 6858 m. VGE were lower during the 4-h 6858-m exposures (32%) than at the higher altitudes (76–85%). The symptom incidences during the first 4 h of exposure were lower at 6858 m and 7620 m following a 1-h prebreathe as compared with analogous zero-prebreathe exposures. There were no differences between incidences of VGE or DCS at any of the four altitudes after 8 vs. 4 h of exposure. ***Conclusion:*** The altitude threshold for 5% DCS symptoms is below 6858 m after 1 h of prebreathe. However, during 6858-m and 7620-m exposures, a 1-h prebreathe is highly beneficial in reducing DCS incidence and delaying the onset of DCS, keeping the incidence to less than 6% during the first 90 min of exposure. Use of 4-h vs. 8-h exposures does not appear to underestimate DCS risk at or above 7620 m.

Keywords: DCS, hypobaric, exercise, prebreathe, preoxygenation, venous gas emboli.

THE ALTITUDE threshold for development of decompression sickness (DCS) symptoms has been described in many textbooks and publications over the past 60 yr (8,9,14,17,20). Although the zero-prebreathe threshold of DCS was concluded to be as high as 7620 m (25,000 ft) to 9144 m (30,000 ft) during World War II, their endpoint criteria for defining a symptom as DCS were more severe than used currently (18). In a contemporary report, the zero-prebreathe threshold for 5% DCS in male subjects was concluded to be 6248 m (20,500 ft) (20) using less severe endpoint criteria during 4-h exposures with mild exercise. Kumar et al. (7) described a model based on previously published research, which estimated an altitude threshold for 5% DCS symptom incidence between 5182 m and 5486 m (17,000–18,000 ft) with analogous mild exercises during a 6-h exposure. Both papers described a 50% DCS incidence in the range of 7010 m to 7315 m (23,000–24,000 ft) under the stated conditions.

With a zero-prebreathe altitude threshold for DCS well below 7620 m, United States Air Force (USAF) General Flight Rules (1) allow continued flight at a cabin altitude of 7620 m for unlimited time following a loss of cabin pressure. This altitude would very likely result in DCS if continued for more than 30 min (5,20). Such exposures should be preceded by some denitrogenation, possibly similar to USAF High Altitude Airdrop Procedures (2), which currently call for at least 30 min of prebreathe and a maximum exposure time of 1 h. Therefore, defining the altitude threshold following a 1-h prebreathe could be operationally important if exposures last for more than 1 h.

The primary goal of this study was to define the threshold altitude at which the incidence of DCS would be 5% following a 1-h prebreathe, typical of many operational scenarios. Exposure altitudes were selected to encompass the altitude threshold of 5% DCS following a 1-h resting prebreathe based on reports dating to the World War II era. A secondary goal was to better define the benefit of a 1-h prebreathe in comparison to no prebreathe at 7620 m and 6858 m.

METHODS

The 51 male, military subjects between 19 and 48 yr of age participated in 109 8-h altitude chamber exposures to 9144 m (30,000 ft), 8382 m (27,500 ft), 7620 m (25,000 ft), and/or 6858 m (22,500 ft). Each subject was exposed to at least one of the four altitudes studied. Although some subjects were exposed more than one time to an altitude, we used only data from the first exposure to ensure equal weighting of data. A 4-h incidence was obtained by counting only those DCS symptoms which occurred at or before 4 h of exposure. This allowed comparison of these results with the majority of Air Force Research Laboratory studies that terminated after 4 h of exposure.

The voluntary, fully informed written consent of the subjects used in this research was obtained and the protocol was approved by the Brooks Institutional Review board. All 51 subjects passed an appropriate physical examination, and were representative of the USAF

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This manuscript was received for review in May 2004. It was accepted for publication in October 2004.

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rated aircrew population in terms of age (30.0 ± 6.9 yr), height (1.78 ± 0.6 m), and weight (75.5 ± 10.8 kg; 24.8 ± 2.9 kg · m^{-1}.) Body fat content was available on 35 of the subjects (20.9 ± 6.9%).

Subjects were trained on the use of oxygen equipment and safety procedures before any research exposures. Subjects were not queried as to their health or well-being during the altitude exposure, but received a briefing on the morning of the exposure which emphasized their responsibility to inform the chamber personnel of any changes in well-being. The altitude exposures were conducted between May 1983 and August 1987 in a hypobaric research chamber at Brooks AFB, TX, using the same procedures, endpoints, and venous gas emboli (VGE) grading criteria as used in all studies conducted to date. An aerospace physiologist was in the chamber vicinity for all subject exposures. Trained personnel assisted with and maintained all oxygen and communications equipment, monitored the chamber pressure and oxygen concentration, and watched for adverse subject reactions. All of the chamber personnel, including the research technicians, investigators, and medical observers were trained to recognize DCS signs and symptoms in subjects. The subjects were also trained to recognize DCS symptoms and how to report their occurrence and progress, and were encouraged to do so expeditiously. If chamber personnel felt that the subject was experiencing unrecognized or serious DCS symptoms, they could initiate recompression and additional interventions in coordination with the physiologist or local dive medicine experts, as necessary. Hyperbaric medicine personnel and facilities were immediately available on site to treat DCS that persisted at ground level.

Before the experiment started, subjects accomplished a communication and ear and sinus pressure equalization check while the altitude chamber was decompressed to provide a simulated altitude of 1524 m (5000 ft) and recompressed to ground level at a rate of 1524 m · min^{-1}. Time spent at the simulated 1524-m altitude was less than 5 s. The subjects donned an Intertechnique™ neck seal respirator and breathed 100% oxygen during the 60-min prebreathe with 100% oxygen, ascent, exposure to the altitudes shown in Table I, descent, and postbreathing. The rate of pressure change was 1524 m · min^{-1}. The respirator provided a slight, 2 cm of water positive pressure which reduced the opportunity for inboard leaks of nitrogen from ambient air. An aviator-type mask was used during some postbreathing.

Every 15 min during each 8-h altitude exposure, the subjects performed five chair-height deep knee bends and raised a 5-lb weight to arm's reach above their head five times with each arm. This exercise was considered mild at about 10–15% of $\dot{V}O_{2max}$. The subjects walked to and from the chair where they rested when not accomplishing other tasks and the echo-imaging station where they reclined and were monitored for VGE every 15 min while performing mild joint articulation for about 4 min. A preliminary report (6) contained results from a limited number of subjects prior to completion of the study and addressed the effect of this exercise.

Medical observers ensured subject health and safety, and made the diagnosis of DCS. Subjects were accompanied by an inside observer. The subjects were instructed to report any changes to the medical observer and the determination to terminate the exposure was made from these reports. The subject was examined after recompression to ground level. The medical observers were trained in the diagnosis of DCS and had the ability to consult with the physicians in Hyperbaric Medicine. Endpoints of the exposures were: 1) completion of the scheduled exposure time; or 2) diagnosis of DCS signs and/or symptoms. Additional detail on endpoint criteria used may be found in Pilmanis et al. (13). Subjects with symptoms requiring potential additional care were referred to the on-site Hyperbaric Medicine staff where they were evaluated and treated with hyperbaric oxygen therapy if necessary. After the exposures, the subjects were given a written list of possible signs and symptoms of DCS. They were told to contact the Hyperbaric Medicine staff in the event of recurring or delayed problems resulting from their hypobaric exposure.

Recording of VGE was nominally accomplished four times per hour using one of two methods. A Hewlett-Packard™ SONOS 500 Echo Imaging System (Andover, MA) used an ultrasound probe via an entry port in the chamber wall and was positioned by the inside observer at the subject's third intercostal space on the left side for a parasternal, short-axis view of the heart. This view allowed clear observation of all four chambers of the heart while the probe was aimed at the apex of the right ventricle (10). In addition to the visualization of the circulating bubbles, the echo image provided visual feedback for probe orientation to allow reception of the best image and ultrasound signals. A Precordial Doppler Ultrasound (Bidirectional Doppler Model 1053, Institute of Applied Physiology and Medicine™, Sound Products Division, Seattle, WA) used in conjunction with a 2D Cardiac Ultrasonic Echo-Imaging, IREX System 3 echo-imaging system (IREX Medical Systems™, Ramsey, NJ) was also employed for many of the VGE observations.

Sequential articulation of each limb during the observation period facilitated movement of VGE to the vena cava and right atrium. Quantification of VGE was estimated using a modified 4-grade Spencer Scale where Grade 1 is infrequent VGE and Grade 4 VGE are of sufficient magnitude to overwhelm the heart sounds (15). Each VGE monitoring session was videotaped and onset times for each level of VGE were recorded to provide information on exposure severity independent of DCS incidence.

Chi-squared tests were used to determine if differences existed between the overall incidence levels resulting from the four altitude scenarios. Kaplan-Meier Survival Analyses (log rank tests) were accomplished to compare the incidence curves. A Microsoft Excel 2003 linear regression trend line was used to determine the correlation between final DCS incidence of each group of subjects and the altitude to which they were exposed. McNemar's test was used to determine if responses after 4 h vs. 8 h of exposure were different.

TABLE I. RESULTS OF 4- AND 8-H EXPOSURES TO VARIOUS ALTITUDES WITH MILD EXERCISE FOLLOWING A 1-H PREBREATHE.

Exposure Altitude N	9144 m (30,000 ft) 30	8382 m (27,500 ft) 33	7620 m (25,000 ft) 27	6858 m (22,500 ft) 19
		VGE		
Incidence (4-h)*	76.7%‡	75.8%‡	85.2%‡	31.6%
Incidence (8-h)	80.0%‡	81.8%‡	88.9%‡	47.4%
Mean latency†, min	67.5	100.2	91.9	161.3
		Grade 4 VGE		
Incidence (4-h)*	60.0%‡	63.6%‡	55.6%‡	21.1%
Incidence (8-h)	63.3%‡	63.6%‡	63.0%‡	26.3%
Mean latency†, min	86.5	97.8	112.7	136.4
		DCS		
Incidence (4-h)*	86.7%‡**	69.7%‡	63.0%‡	26.3%
Incidence (8-h)	86.7%‡	81.8%‡	77.8%§	52.6%
Mean latency†, min	85.9	142.5	150.8	210.8

*Incidence after 4 h of 8-h exposure.
†During 8-h exposure.
‡Higher incidence than at 6858 m ($p < 0.05$; Chi-squared and Kaplan-Meier log rank tests).
§Higher incidence than at 6858 m ($p < 0.05$; Kaplan-Meier log rank test).
**Higher incidence than at 7620 m ($p < 0.05$; Chi-squared and Kaplan-Meier log rank tests).

RESULTS

After 4 h of exposure, the incidence of DCS was lower at 6858 m (26%) than at any of the higher altitudes ($p < 0.02$) (**Table I, Fig. 1**). The relatively large drop in DCS from 7620 m to 6858 m is mirrored by a large reduction in VGE (**Fig. 2**). The DCS incidence at the end of 4 h of exposure (**Fig. 3**) showed a high correlation with altitude ($R^2 = 0.90$; $R = 0.95$). The Chi-squared and Kaplan-Meier survival analyses were in agreement on the significant differences indicated in Table I with a single exception. The Kaplan-Meier log rank test showed a significant difference between the shapes of the 8-h DCS curves representing 6858 m and 7260 m, whereas the Chi-squared test was not significant at 3.2 ($p = 0.07$).

VGE and Grade 4 VGE levels were much lower at 6858 m vs. all higher altitudes tested after 4 or 8 h of exposure (Table I; $p < 0.02$). During the 8-h, 8382-m exposure, 82% (27/33) developed DCS and 82% developed VGE. Although this coincidence may imply a direct relationship, if incidence of VGE and DCS were completely independent results, then 67% (0.82×0.82) would have developed both VGE and DCS. Since 22 of 33 subject-exposures developed both VGE and DCS (67%) during these exposures, the result does not appear to be indicative of any capability to predict DCS from these VGE data for any individual (3,11). However, the level of VGE is, in itself, a measure of exposure severity for a population, just not indicative of susceptibility to DCS on an individual basis (20).

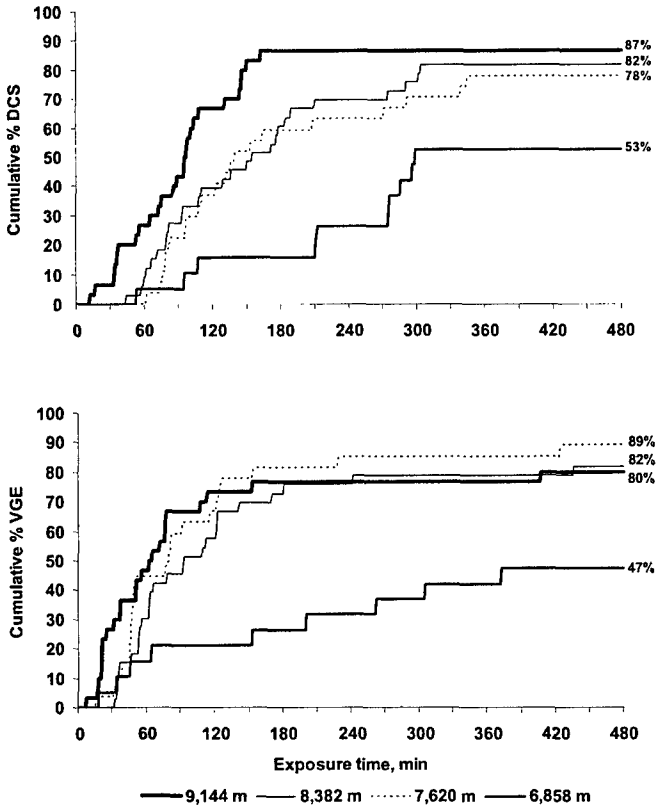

Fig. 1. Cumulative % DCS and VGE during 8-h exposures following a 1-h prebreathe.

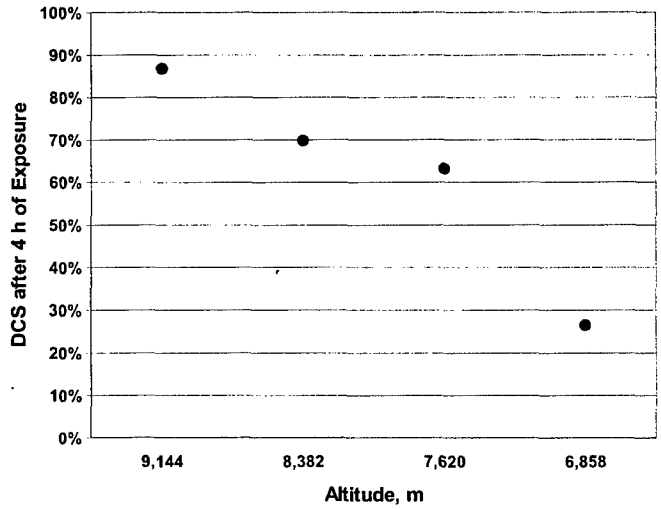

Fig. 2. Reduction in DCS incidence with altitude after 4 h of exposure with mild exercise following a 1-h prebreathe.

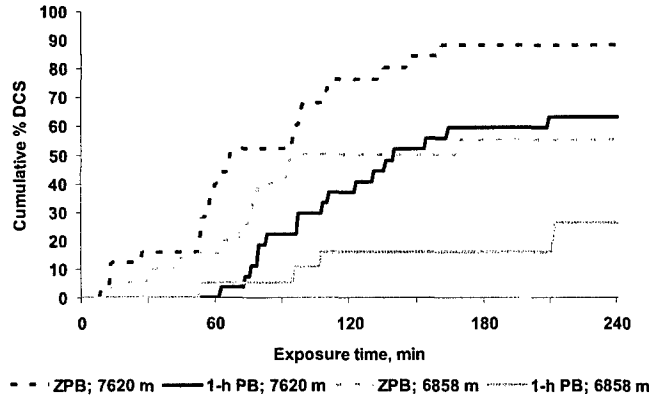

Fig. 3. Comparison of zero-prebreathe (ZPB) exposures with exposures following a 1-h prebreathe to 6858 m and 7620 m. Note: ZPB data from Webb et al. (20).

DISCUSSION

An altitude threshold for 5% DCS after 1 h of prebreathe and 4 h of exposure could not be established within the parameters of this study. The 26% DCS at 6858 m, the lowest altitude tested, indicates that the threshold is lower. That threshold may be comparable to the 6462-m (21,200 ft) altitude threshold for 5% DCS without benefit of prebreathe (20) due to the dynamics of denitrogenation. Prebreathe of 1 h accomplishes nearly complete denitrogenation of the lungs and blood (fast tissues) and considerable denitrogenation of medium tissues such as muscles and nerve tissue. However, a 60-min prebreathe has a relatively minor effect on the level of nitrogen in bones and tendons (slowly denitrogenating tissues; slow tissues). Elimination of the nitrogen in the fast tissues and partial denitrogenation of the medium tissues could explain the virtual elimination of serious DCS symptoms compared with DCS during zero-prebreathe exposures. Partial denitrogenation of the slow tissues may explain the remaining effect of a 1-h prebreathe. This lack of a large change in altitude threshold for 5% DCS symptoms may be explained by the lack of denitrogenation of the slow tissues, implying a different nature of the symptoms resulting from bubble formation in the different types of tissue.

The sigmoidal curve representing cumulative DCS incidence vs. exposure duration following a 1-h prebreathe is moved significantly to the right as compared with the analogous curve representing zero prebreathe (20) (Fig. 3; $p < 0.05$ at 6858 m and 7620 m; Kaplan-Meier tests). This result was expected due to the benefit of prebreathe as described in reference texts. The 1-h prebreathe has the effect of increasing latency for symptom development, reducing DCS incidence at any time during the 4-h exposures. After 4 h of exposure, the incidence following a 1-h prebreathe was 63% vs. a zero-prebreathe incidence of 87% (20). The delay of DCS symptom onset with 1 h of prebreathe prior to 6858-m exposures kept the incidence to less than 6% during the first 90 min and 16% during the first 2 h of exposure. This compares to 40% and 50% DCS without prebreathe. This comparison revealed similar results at 7620 m where the incidence after 2 h of zero-prebreathe exposure was 37% greater than with a 1-h prebreathe (Fig. 3). Thus, if the exposure is relatively short, up to 2 h, the benefit of a 1-h prebreathe is more noticeable. Although the slopes of the curves appear to be very similar, Fig. 3 shows the considerable value of a 1-h prebreathe at 7620 m and 6858 m in delaying symptom onset and reducing DCS incidence after 4 h of exposure.

The relatively high level of DCS during zero-prebreathe exposures to 7620 m and 6858 m (Fig. 3) emphasizes the need for DCS protection at those altitudes (4,15,18). The protection offered by a 1-h prebreathe prior to long-duration exposures to 7620 m provides an alternative. During any reevaluation of USAF directives regarding zero-prebreathe exposures in the range of 7620 m to 6858 m, the curves shown in Fig. 1 and 3 and information in Haske and Pilmanis (5) could be of use. To help ensure development of only minor and infrequent symptoms of DCS, the limit of exposure time at 7620 m should be no more than 30 min, 45 min if the possibility of some more serious symptoms is deemed acceptable (5). Increasing exposure times at lower altitudes allowing up to 60 min at 6858 m would help ensure no more than a low incidence of DCS and very few serious cases.

The reduction in DCS incidence from 9144 m to 7620 m is of particular interest due to the large number of studies which looked at DCS incidence at one of those altitudes. There is a need to relate results at 9144 m to results at 7620 m. During a 4-h exposure, the 63.0% DCS at 7620 m is 24% less than the 86.7% DCS at 9144 m (Table I). Stated another way, DCS incidence at 7620 m was 73% of that observed at 7620 m (0.630/0.867) after 4 h of exposure. This serves as an indication of the altitude effect on DCS incidence following identical prebreathe procedures.

Incidence of additional cases of DCS developing after 4 h of exposure following a 1-h prebreathe is of interest in evaluating the level of DCS risk during long operational scenarios which use prebreathe to reduce risk. After 5 h of oxygen breathing (1-h prebreathe plus 4 h of exposure while breathing 100% oxygen), continuing denitrogenation, it is reasonable to assume that further bubble formation is curtailed during the type of exposures discussed here. A comparison of symptom incidence after 8 h vs. 4 h would be of value in documenting the validity of using the shorter, 4-h exposures to evaluate DCS risk. The ability to use shorter exposures to obtain relevant data would reduce the workload and overall cost of DCS research studies which address risk associated with long exposures. The difference between cumulative incidence of VGE, Grade 4 VGE, and DCS at 4 h and 8 h was not significant at any of the altitudes (Table I; McNemar's Test; $p > 0.05$). At altitudes above 6858 m, the onset curves in Fig. 1 show nearly complete leveling beyond 4 h of exposure. These findings provide support for using 4-h exposures in lieu of longer exposures to determine level of risk at altitudes above 6858 m. In addition to saving time, the decision to use shorter exposures would allow a considerable reduction in the personnel required to monitor the subject-exposures.

The data from this study also provided possible insight regarding the effects of exercise at 9144 m on DCS incidence. The 87% DCS at 9144 m during this study was higher than during other exposures to 9144 m (or 8992 m) for the same duration, also with mild exercise and 1 h of prebreathe ($p < 0.05$) (12,16,19). This higher DCS incidence could be related to the difference in mild exercises performed. The exercises used in this experiment involved more stress on the lower body (chair-height deep knee bends) than walking between stations to do the mild, upper-body exercises used in most of our experiments (4,16,19).

CONCLUSIONS

The altitude threshold of DCS (5% symptoms) was shown to be below 6858 m after 1 h of prebreathe. However, at 6858 m and 7620 m, a 1-h prebreathe is highly beneficial in reducing DCS incidence, compared with zero prebreathe, and delaying the onset of DCS,

keeping the incidence to less than 6% during the first 90 min of exposure. Following a 1-h prebreathe, a 4-h altitude exposure with mild exercise at 7620 m resulted in approximately 24% less DCS than at 9144 m. Use of 4-h vs. 8-h exposures does not appear to result in underestimation of DCS risk in the altitude range from 7620 m to 9144 m. Aerobically similar exercises during altitude exposure appear to result in different DCS outcomes and should be the subject of further research.

ACKNOWLEDGMENTS

This research was sponsored, in part, by NASA under Contract T-82170. The authors gratefully acknowledge the conceptual and investigative efforts of Kenneth W. Smead, M.D., William T. Harvey, M.D., Gene A. Dixon, B.S., and Robert W. Krutz, Ph.D.; the medical monitoring support from Robert M. Olson, M.D., Ph.D.; the statistical support by Joseph R. Fischer, M.S.; and the technical efforts of the research and chamber personnel involved throughout the study. Opinions, interpretations, conclusions, and recommendations are those of the authors and are not necessarily endorsed by the United States Air Force.

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REPORT DOCUMENTATION PAGE

Form Approved
OMB No. 0704-01-0188

1. REPORT DATE (*DD-MM-YYYY*)	2. REPORT TYPE	3. DATES COVERED (*From - To*)
1-Jan-2005	Interim	Aug-1983 - Dec 2005

4. TITLE AND SUBTITLE
Altitude decompression sickness between 6858 and 9144 m following a 1-h prebreathe.

5a. CONTRACT NUMBER
F41624-97-D-6004

5b. GRANT NUMBER

5c. PROGRAM ELEMENT NUMBER
62202F

6. AUTHORS
James T. Webb, Ph.D. and Andrew A. Pilmanis, Ph.D.

5d. PROJECT NUMBER
7184

5e. TASK NUMBER
58

5f. WORK UNIT NUMBER
01

7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)
Wyle Laboratories, Life Sciences, Systems and Services Inc.
2485 Gillingham Drive
San Antonio, TX 78235-5105

8. PERFORMING ORGANIZATION REPORT NUMBER

9. SPONSORING/MONITORING AGENCY NAME(S) AND ADDRESS(ES)
High Altitude Protection Research
Aircrew Performance and Protection Branch
2485 Gillingham Drive
San Antonio, TX 78235-5105

10. SPONSORING/MONITOR'S ACRONYM(S)
AFRL/HEPG

11. SPONSOR/MONITOR'S REPORT NUMBER(S)
AFRL-HE-BR-JA-2004-0010

12. DISTRIBUTION/AVAILABILITY STATEMENT
Approved for Public Release

13. SUPPLEMENTARY NOTES
This report was published as a peer-reviewed article in Aviation, Space and Environmental Medicine 2005;76:34-8.

14. ABSTRACT

Introduction: The zero prebreathe altitude threshold for developing 5% decompression sickness (DCS) symptoms in men has been reported to be 6248 m (20,500 ft). However, such an altitude threshold when 1 h of oxygen prebreathe is used has not been well documented and was the primary purpose of this study. ***Methods:*** The 51 male human subjects were exposed to 9144 m (30,000 ft), 8382 m (27,500 ft), 7620 m (25,000 ft), and/or 6858 m (22,500 ft) for 8 h. They were monitored for symptoms of DCS and venous gas emboli (VGE). ***Results:*** DCS symptom incidence after 4 h of exposure decreased with exposure altitude from 87% at 9144 m to 26% at 6858 m. VGE were lower during the 4-h 6858-m exposures (32%) than at the higher altitudes (76–85%). The symptom incidences during the first4hof exposure were lower at 6858 m and 7620 m following a 1-h prebreathe as compared with analogous zero-prebreathe exposures. There were no differences between incidences of VGE or DCS at any of the four altitudes after 8 vs.4hof exposure. ***Conclusion:*** The altitude threshold for 5% DCS symptoms is below 6858 m after 1 h of prebreathe. However, during 6858-m and 7620-m exposures, a 1-h prebreathe is highly beneficial in reducing DCS incidence and delaying the onset of DCS, keeping the incidence to less than 6% during the first 90 min of exposure. Use of 4-h vs. 8-h exposures does not appear to underestimate DCS risk at or above 7620 m.

15. SUBJECT TERMS
decompression sickness, venous gas emboli, exercise, prebreathe, preoxygenation

16. SECURITY CLASSIFICATION OF:			17. LIMITATION OF ABSTRACT	18. NUMBER OF PAGES	19A. NAME OF RESPONSIBLE PERSON
a. REPORT	b. ABSTRACT	c. THIS PAGE			James T. Webb
U	U	U	UU	5	19b. TELEPHONE NUMBER (*Include area code*) 210-536-3439