# Oxygen Consumption at Altitude as a Risk Factor for Altitude Decompression Sickness

James T. Webb, Larry P. Krock, and Michael L. Gernhardt

WEBB JT, KROCK LP, GERNHARDT ML. Oxygen consumption at altitude as a risk factor for altitude decompression sickness. Aviat Space Environ Med 2010; 81:987–92.

Introduction: The existence of a general influence of exercise on the incidence of decompression sickness (DCS) has been known for more than a half-century. However, quantification of the effect has not been done for several reasons, including isolation of exercise as the only variable. The DCS database at Brooks City-Base, TX, contains detailed physiologic information on over 3000 altitude exposures. The purpose of this study was to measure Vo<sub>2</sub> during the activities performed during those exposures to retrospectively determine if Vo2, a quantifiable index of exercise intensity, was related to the level of reported DCS. Methods: Ground-level activity was designed to duplicate the standardized activity during the altitude exposures. Breath-by-breath VO2 was determined for each activity using a COSMED® metabolic measurement system. Comparison of the  $\dot{V}O_2$  during four levels of activity performed under otherwise comparable conditions allowed a determination of correlation between Vo<sub>2</sub> and DCS risk observed during the altitude exposures. Results and Discussion: Four previous altitude exposure profiles at 8992 m to 9144 m (29,500 to 30,000 ft; 231 to 226 mmHg) for 4 h following a 1-h prebreathe resulted in 38-86% DCS. This study provided the Vo2 of activities during those studies. The correlation between DCS incidence and the highest 1-min Vo<sub>2</sub> of activity was 0.89. Conclusion: The highest 1-min VO2 showed a high correlation with level of DCS risk. Future exposures involving lower levels of activity could provide data that would allow improvement in modeling of DCS risk. Keywords: oxygen consumption, exercise, DCS 39.169.20.187 On: Tue

DECOMPRESSION sickness (DCS) symptoms may develop during exposure to reduced atmospheric pressure. It takes many hours for the partial pressure of nitrogen dissolved in the body tissues and fluids at ground level to equilibrate with the lower partial pressure of nitrogen at lower atmospheric pressure. Hence, supersaturation of tissue with nitrogen occurs during depressurization and can lead to formation of a gas phase, that is, nitrogen bubbles, in tissues and fluids. The bubbles may put pressure on nerves or block blood flow and possibly lead to the development of a broad range of pain, skin, respiratory, and/or nervous system symptoms.

Prebreathe is denitrogenation by breathing 100% oxygen before decompression to reduce the risk of DCS. Breathing 100% oxygen reduces the nitrogen partial pressure entering the lung alveoli to zero, creating a large diffusion gradient with the nitrogen partial pressure in the lung capillaries. Longer prebreathe results in a lower incidence of DCS (22).

Aerospace environments that can cause DCS include inadequately pressurized cabins during high-altitude flight, extravehicular activity (EVA) from a spacecraft, and depressurization in an altitude chamber. The chance of developing DCS depends mostly on the partial pressure of nitrogen in the tissues (including blood) before decompression and the four primary DCS risk factors: altitude, time at altitude, prebreathe time, and level of activity while decompressed (12,13,21,24). These primary DCS risk factors were used to determine DCS risk during development of the Altitude DCS Risk Assessment Computer (ADRAC) model at Brooks City-Base, TX (13). That model allows input of altitude to the nearest foot, time at altitude, and prebreathe time to the nearest minute, but the levels of activity are limited to 1) rest, 2) mild, and 3) heavy, with no quantification of the  $VO_2$  to differentiate those levels (8,13). Lack of a clear definition for these levels of activity forces subjective interpretation of exercise intensity, which limits the impact of that risk factor on the model's results. Quantitative activity intensity, as it applies to DCS risk, is not known because this factor was not measured when the studies were conducted.

Previous altitude research that included exercise while subjects were decompressed (5,7,12) has consistently shown that physical activity does affect DCS risk. The investigations cited above demonstrated that DCS risk was higher when physical activity was performed while decompressed than when no physical activity was performed. They included various activities that emulated what the aircrew population did while decompressed, but they did not quantify or ascribe importance to the level of activity performed. Therefore, little is known about the role of physical activity intensity in DCS risk. Pilmanis et al. (12) evaluated the mode of activity while decompressed, either isometric or dynamic exercise (3), to determine if it affected DCS risk. They concluded there was no significant difference in DCS incidence between isometric arm, isometric leg, dynamic arm, and

From the NASA Johnson Space Center, Houston, TX.

This manuscript was received for review in March 2010. It was accepted for publication in July 2010.

Address correspondence and reprint requests to: James T. Webb, Ph.D., 13818 Chittim Oak, San Antonio, TX 78232; jtwebb@swbell.net.

Reprint & Copyright © by the Aerospace Medical Association, Alexandria, VA.

DOI: 10.3357/ASEM.2787.2010

dynamic leg exercises that elicited equivalent levels of oxygen consumption ( $\dot{V}O_2$ ).

Some efforts have been made to quantify oxygen consumption during EVA and standardized activity simulations while subjects are decompressed to EVA pressure suit environments. Inderbitzen and DeCarlis (6) reported that six male subjects used 31 L  $\cdot$  h<sup>-1</sup> (148 kcal  $\cdot$ h<sup>-1</sup>) for the three activities described as EVA exercises. Waligora and Kumar (15) reported an average oxygen consumption of 40 L  $\cdot$  h<sup>-1</sup> (194 kcal  $\cdot$  h<sup>-1</sup>) during 59 Shuttle EVAs. No other reports were found which described oxygen consumption during any of the other activities performed while decompressed during research chamber exposures at Brooks Air Force Base/City-Base, TX.

Littell and Joy (10) reported that the very light work of piloting three U.S. Army helicopters (light, utility, and medium) and one utility fixed-wing aircraft required 107 kcal  $\cdot$  h<sup>-1</sup>. Kaufman et al. (9) described the metabolic rates of experienced and inexperienced KC-135 pilots during simulated emergency conditions as 126 and 182 kcal  $\cdot$  h<sup>-1</sup>, respectively. Unfortunately, none of these papers reported peak work rate (for example, highest 1 min) during the activities tested.

The USAF School of Aerospace Medicine (USAFSAM) Altitude DCS Research Database (DCS Database; 16) contains extensive information on over 3000 altitude exposures conducted under standardized procedures and clear endpoint criteria. Once sufficient data had been collected late in this effort, a trend was observed. A considerable range of DCS incidence (38–86%) was observed during altitude exposures to 8992–9144 m (29,500 to 30,000 ft; 231–226 mmHg, 4.5–4.4 psi) for 4 h. The difference observed between these otherwise equivalent exposures was the type of activity. This indicated that physical activity while decompressed may have a considerable influence on DCS risk. A general observation led us to hypothesize higher levels of activity resulted in much more DCS than the lower levels of activity.

The purpose of this study was to measure VO<sub>2</sub> during each activity performed while decompressed during the earlier studies to better evaluate activity intensity, and then determine if a relationship exists between DCS incidence and  $\dot{V}O_2$  during exposure. Subjects performed the same exercise protocols as done during previous altitude exposure studies. Application of a defined relationship between  $\dot{V}O_2$  during exposure and DCS incidence could provide a better basis for the ADRAC model or other altitude DCS prediction models.

## **METHODS**

The protocols which provided data for this effort were reviewed by the Brooks AFB/City-Base Institutional Review Board and approved by the USAF Surgeon General's Research Compliance Office and/ or the Wright-Site Institutional Review Board at Wright-Patterson AFB, OH, and the NASA JSC Committee for the Protection of Human Subjects in accordance with existing regulations. All subjects passed an appropriate subject physical examination and signed a voluntary, fully informed consent document describing the experimental conditions and risks of their participation.

The use of data from subject exposures to reduced pressure in Chamber C at Brooks AFB, TX, in the 1980s and 1990s with data from ground-level tests of oxygen consumption by different subjects in 2008 and 2009 is problematic and represents a potential limiting factor to application of the findings. However, the two sets of subjects were of comparable age, height, and weight with the research subjects who performed the previous altitude decompression exposures (**Table I**) and they performed the same activity in the same chamber with analogous or identical equipment.

## Subjects

*Previous altitude exposures:* Male subjects (107) participated in the previous 165 altitude DCS research subject-exposures described by Pilmanis et al. (12) and Webb et al. (19,25,23). Data for level of DCS risk used in this study were from those 165 male subject exposures to altitude. These profiles were accomplished at a time and for reasons which precluded use of female subjects.

*Current study:* A subject pool of 22 U.S. Air Force active duty male enlisted and officer subjects were employed on days of leave to participate in ground-level activity emulating the activity of the previous altitude exposure subjects while decompressed. The current subjects were representative of subjects in those earlier protocols (Table I). Data on oxygen consumption are from the current study of these 22 male subjects.

TABLE I. ANTHROPOMETRIC AND PHYSIOLOGIC VARIABLES OF 165 MALE SUBJECTS IN PREVIOUS ALTITUDE DCS EXPOSURES AND 22 MALE SUBJECTS IN THE CURRENT STUDY (MEAN  $\pm$  SD).

	Age, yr	Weight, kg	Height, m	BMI	BF, %*	$\dot{V}_{O_{2peak'}} \operatorname{ml} \cdot \operatorname{kg}^{-1} \cdot \operatorname{min}^{-1} ^{+}$
Altitude Exposures	$29.5 \pm 6.2$	$82.9 \pm 11.0$	$\begin{array}{c} 1.79 \pm 0.07 \\ 1.78 \pm 0.08 \end{array}$	$25.9 \pm 2.8$	$21.3 \pm 5.3$	$39.9 \pm 7.8$
Current Study	$31.1 \pm 7.9$	$79.0 \pm 11.3$		$25.0 \pm 3.3$	$15.6 \pm 6.8$	$44.0 \pm 9.5$

\* Body composition (body fat percentage) was estimated using the 3-site caliper method (2).

<sup>+</sup> vo<sub>2peak</sub> was estimated from performance of the USAF submaximal Cycle Ergometry Fitness Test (1) or determined by performance of a maximal treadmill test (Bruce Protocol) or estimated by performance of a maximum cycle ergometry test (26).

Subjects in the altitude exposures had a higher percentage of body fat (BF, %) than subjects in the current study (P = 0.001). There were no other significant differences. Body fat and  $vo_{2peak}$  were not available for all 165 subjects who participated in the previous altitude protocols. Of these subjects, 107 had recorded body fat and 70 had recorded  $vo_{2peak}$  values. BMI = body mass index, kg · m<sup>-2</sup>.

### Facilities and Endpoint Criteria

Previous altitude exposures: Chamber facilities, equipment, and procedures during the previous altitude DCS research were described by Webb (16,17). Subjects were monitored for, and data collected on, DCS symptoms. Endpoints of the earlier altitude exposures were: completion of the scheduled altitude exposure (4 to 8 h, with DCS data truncated to 4 h); or development of DCS signs or symptoms. A more complete description of the exposure endpoints may be found in Webb (17).

Previous altitude exposure profiles and activities while de*compressed:* A comparison of the effects of VO<sub>2</sub> while decompressed on DCS incidence could only be done if it was the only variable. The altitude exposure profiles were chosen to have the same altitude, time at altitude, and prebreathe time, leaving level of activity,  $\dot{V}O_2$ , as the only variable. The four altitude profiles were research chamber exposures to 8992-9144 m (29,500 to 30,000 ft) following a 1-h prebreathe. All altitude exposures lasted 4 h; results from longer exposures were truncated to 4 h as has been shown to be valid in previous studies (13,23). The large range of DCS incidence, 38-86%, observed during these earlier altitude exposures (Table II) employed disparate types and intensities of activity while decompressed. Detailed instructions prescribed the minute-by-minute activities of the subjects during all of these profiles (12,19,20,16). The data used in this effort was from exposures completed or terminated without deviation from the prescribed activities or procedures. All of these activities included echocardiographic (echo-) imaging with a Hewlett-Packard® SONOS 1000 Echo Imaging System<sup>™</sup> (Andover, MA) (or analogous equipment requiring the same movements) while reclined on a gurney. During the imaging, subjects would fully extend and slightly twist each limb twice to facilitate movement of venous gas emboli to the heart 20.187 On: Tuesequences 18 13:46:1

The four activities consisted of:

- 1. Ambulatory rest (seated rest for 15 min with one ambulation to a gurney for echo-imaging taking less than 4 min); 2. Rope pull (pulley with 7.7 kg, one pull from eye level to waist
- each 5 s for 5 min, one ambulation to a gurney for echo-imaging taking less than 4 min during each 15-min exposure segment, with the remaining time spent at seated rest; 25);
- 3. Knee bends & overhead presses [five chair-height deep knee bends and five 2.3-kg (5-lb) dumbbell overhead presses with

each arm, from shoulder height while seated, in less than 2 min, and one ambulation to a gurney for echo-imaging taking less than 4 min]. Each chair-height deep knee bend was accomplished by the subject standing up from a seated position and sitting back down, repeated five times. This sequence of exercises was performed one time during each 15-min exposure segment and the remaining time was spent at seated rest [Webb and Pilmanis (23)]: and

- 4. Ambulatory EVA activity (one ambulation to a gurney for echoimaging taking less than 4 min and three ambulatory upper-body activities performed for 4 min each during the 16-min exposure segment; 4):
  - cycle ergometer (24 rpm, 4-Newton resistance; light);
  - torque wrench (25 ft-lb held 5 s in each position); and
  - rope pull (pulley with 7.7 kg, once from eye level to waist each 5 s).

Subjects breathed aviators oxygen (99.7% oxygen) during the altitude exposures. They were slightly hyperoxic, thus hypoxia was not a factor.

Current study—facilities, equipment, and ground-level activities: The subjects performed all activity at ground level inside the same chamber and used the same exercise equipment as used during the altitude DCS protocols described above. VO<sub>2</sub> during this study's activities was determined with a COSMED© K4b<sup>2</sup> Cardio Pulmonary Exercise Testing<sup>™</sup> system (COSMED, Sri, Italy) worn by the subjects and validated by McLaughlin et al. (11). After donning the lightweight COSMED  $K4b^2$ , subjects remained at seated rest for at least 10 min before beginning each timed sequence of activities to allow recovery from the previous mild or moderate activity. Simulated echo imaging was accomplished to emulate the movements made during echo imaging of the decompressed subject's heart. Each activity sequence was accomplished twice. The breath-by-breath data were reduced to 1, 2, and 3-min averages of the 15- and 16-min sequences. The highest  $VO_2$  is based on the maximum VO<sub>2</sub> from either of the two identical activity

Copyright: Aerospace Me Determination of the Pearson's correlation coefficient between VO<sub>2</sub> during the activity sequences and DCS incidence during the altitude exposures was accomplished. The correlations are shown in Table II and a linear regression trend line and display of the equation for R<sup>2</sup> are shown in Fig. 1. A one-way ANOVA for correlated groups on the means of the highest  $VO_2$  during any 1 min of these activities was also accomplished.

TABLE II. OXYGEN CONSUMPTION (Vo2) OF PROFILE SEQUENCES AND VO2 DURING HIGHEST 1 MIN OF ACTIVITY WITHIN PROFILE SEQUENCES.

Activity	DCS Incidence	Mean $\dot{V}o_2^{\dagger}$ , ml $\cdot$ kg <sup>-1</sup> $\cdot$ min <sup>-1</sup>	Mean $\dot{V}o_2^+$ , kcal $\cdot h^{-1}$	Highest 1-min $\dot{V}_{O_2}^{\dagger}$ , ml $\cdot$ kg <sup>-1</sup> $\cdot$ min <sup>-1</sup>
Seated Rest	Unknown	3.5	78.6	5.4
Ambulatory Rest*	38%	3.7	82.9	7.6
Rope Pull*	61%	4.9	110.4	8.8
Knee Bends & Overhead Presses*	86%	4.8	97.4	10.4
Ambulatory EVA*	75%	7.6	170.9	11.2
Regression (R <sup>2</sup> )		0.309	0.793	0.793
Correlation Coefficient (r)		0.556	0.788	0.890

\* These activities by 22 male subjects were the only variables in Fig. 1 where all altitude research subjects were exposed to otherwise equivalent conditions. All activities involved simulated echo-imaging joint articulations during each activity sequence. <sup>+</sup> Mean  $V_{O_2}$  refers to the mean  $V_{O_2}$  during all sequences of a specific activity, whereas the highest 1-min  $V_{O_2}$  refers to the highest 1 min of  $V_{O_2}$  during any specific activity.



**Fig. 1.** % DCS as a function of highest  $\dot{V}O_2$  in any 1 min of activity.

### RESULTS

Current subjects' mean values for anthropometric and physiologic variables reasonably matched those of the subjects from the altitude DCS profiles who provided the DCS risk data (Table I). Table II shows the mean VO<sub>2</sub> and oxygen consumption of the male subjects who participated in the ground-level activity sequences. The data show that mean  $\dot{V}O_2$  during the activity sequences has a Pearson's correlation coefficient with DCS incidence of 0.556 (Table II). Additional determinations of the highest 1-, 2-, and 3-min VO<sub>2</sub> were accomplished for each type of activity. The correlation coefficient was most increased when the highest VO<sub>2</sub> in any 1 min of activity was plotted vs. DCS incidence (Fig. 1; r = 0.890). A one-way ANOVA for correlated groups revealed that the means of the highest  $VO_2$ during any 1 min of these activities were significantly different [F(3, 21) = 37.8, *P* < 0.0001].

During the 16-min sequence of the ambulatory EVA activities in this study, the mean oxygen consumption of 36 L  $O_2 \cdot h^{-1}$  (171 kcal  $\cdot h^{-1}$ ) was comparable to the 31 L  $O_2 \cdot h^{-1}$  (148 kcal  $\cdot h^{-1}$ ) reported by Inderbitzen and DeCarlis (6) and the average oxygen consumption of 40 L  $O_2 \cdot h^{-1}$  (194 kcal  $\cdot h^{-1}$ ) during 59 Shuttle EVAs reported by Waligora and Kumar (15). The oxygen consumption, in kcal  $\cdot h^{-1}$ , during all four activities evaluated in this report (Table II) was 24 L  $O_2 \cdot h^{-1}$  (115 kcal  $\cdot h^{-1}$ ; 9.5 ml  $\cdot kg^{-1} \cdot min^{-1}$ ), similar to the 22 L  $O_2 \cdot h^{-1}$  (107 kcal  $\cdot h^{-1}$ ) to 26 L  $O_2 \cdot h^{-1}$  (126 kcal  $\cdot h^{-1}$ ) used by experienced pilots during flight as described earlier (10,9).

Deviations of the points from the regression line in Fig. 1 probably reflects some of the limitations of the study, which depended on the validity of ground-level activity in this study accurately representing the previous studies' activity while decompressed. The meticulous minute-by-minute test plans directing specific activities during analogous activities of the previous and current subjects' activities should have reduced the effects of this limitation.

#### DISCUSSION

This report indicates that a relationship may exist between  $\dot{V}O_2$ , the highest 1 min of activity in a repeated 15- to 16-min sequence while subjects are decompressed, and DCS risk. This relationship of increased activity producing higher incidence of DCS may result from a cumulative effect of the increased activity in the highest 1 min of multiple activity sequences. This effect is reflected in the onset curves of DCS in **Fig. 2**. The implication may be that the growth of accumulated extravascular bubbles is related to repeated activity peaks. Multiple activity peaks may also enhance coalescence of adjacent bubbles, creating larger bubbles that apply greater pressure to nerves and have a greater chance of blocking blood flow.

Each of the three categories of activity used by the ADRAC model spans a considerable range of  $\dot{V}O_2$ . The rest category in ADRAC was based on data during exposures involving activity defined here as ambulatory rest. The additional level of activity required by frequently walking versus remaining at seated rest while decompressed could result in a large error in predicting DCS risk at "rest." The apparent linear relationship between the highest  $\dot{V}O_2$  during any 1 min of activity and DCS incidence may allow a simple factor to be applied to the existing prediction, potentially providing improvement to the ADRAC model.

Application of these data could begin by measuring the VO<sub>2</sub> during various altitude-exposure scenarios where a risk of DCS is recognized. Many of these activities may be sufficiently consistent to allow a reasonable determination of their highest 1-min oxygen consumption. The equipment used during this experiment was mobile and offered little restriction to maneuvering as in an aircraft simulator (cockpit). With several subjects or crewmembers accomplishing these activities in a simulator as prescribed by checklist or logical and standardized sequence in a realistic time scale, reproducible results could be obtained. This method of determining VO<sub>2</sub> in an aircraft is applicable to aircraft simulators such as the simulator for the U-2. The U-2 aircraft can present a challenging DCS risk during high-altitude flight.



Fig. 2. Cumulative DCS incidence in altitude DCS research exposures.

# OXYGEN CONSUMPTION & ALTITUDE DCS-WEBB ET AL.

Although DCS risk is very low in most aircraft, documentation of pilot VO<sub>2</sub> during simulated flight of some aircraft may be used for comparison with the current data. The comparison may provide additional refinement of estimated DCS risk. The highest VO<sub>2</sub> during any 1 min of seated rest is only about 70% of that during ambulatory rest. A corresponding level of DCS would be about 20% based on the linear relationship shown in Fig. 1. Additional altitude-chamber exposures to 30,000 ft (9144 m) while performing seated rest with echoimaging simulation could show whether that extrapolation is valid. The relatively low level of VO<sub>2</sub> during seated rest is similar to that of pilots who remain seated during the entire exposure to reduced pressure. The DCS database indicates that 19% and 50% DCS occurred during 4-h exposures at 22,500 ft (6858 m) (20) without prebreathe. The DCS incidence corresponds with the performance of ambulatory rest or ambulatory EVA activity, respectively, and indicates how a relatively small increase in 1-min peak VO<sub>2</sub> may increase DCS risk even at the low end of the DCS risk altitudes. With addition of analogous exposures while subjects' activity was only seated rest, an additional regression line and equation could be created. This would add considerable information regarding the relationship of  $VO_2$  (highest 1 min) with DCS risk.

The lower incidence of DCS with lower rates of VO<sub>2</sub> described in Fig. 1 also seems to affect the shape of the cumulative incidence curve of DCS during 4-h altitude exposures (Fig. 2; 12,19,25,24). The profile with the lowest oxygen consumption, "Ambulatory Rest," displays the slowest onset of DCS and had several periods where little or no DCS occurred. The onset of DCS during Ambulatory Rest included a nearly flat line during the last hour, increasing by only 6.6%. This same slow increase in incidence may be a factor during long-duration, high-altitude flights during which the pilots have an even lower oxygen consumption. This was shown in the report by Webb and Pilmanis (23), which showed that DCS symptoms occurred after 5 h while subjects performed moderate-intensity activity. Even after 6 h of altitude exposure, DCS cases could continue to occur (14).

The area of the body which is active does not appear to influence the DCS risk, only the location distribution of ensuing symptoms. A previous study showed that joint pain symptoms were more prevalent in the lower body when lower-body activity was present and more prevalent in the upper body when lower-body activity was very low (18). However, in the same study there was no difference in the total level of joint-pain DCS present when similar levels of activity intensity were accomplished. The location of the majority of the activity and occurrence of joint-pain DCS symptoms agrees with previously reported findings (24).

This retrospective study of  $\dot{V}O_2$  identified a strong positive correlation, 0.890, between DCS incidence and the highest 1-min  $\dot{V}O_2$  while subjects were decompressed. Nearly 80% of the variation in DCS incidence was explained by the single variable of activity intensity.

These results expand the findings in many previous reports where data on measured  $\dot{V}O_2$  was not reported, but exercise was shown to produce more DCS than a resting altitude exposure. The relationship presented here could improve efforts to predict the risk of DCS during planned altitude exposures using the ADRAC model or other altitude DCS models. This improved accuracy could be expanded further with additional research at other altitudes and prebreathe times. Improved accuracy in quantification of activity intensity could allow consideration of modifying the peak level of activity while decompressed as a means of greatly reducing DCS risk. Expansion of the understanding of the role of VO<sub>2</sub> in DCS risk could explain differences between reports of DCS incidence during aerospace operations and its incidence during research studies of altitude DCS. In particular, the low incidence of DCS reported by U-2 pilots who have relatively low activity while decompressed, such as long-haul pilots, versus the high level of DCS observed during experimental exposures involving much higher levels of activity.

#### ACKNOWLEDGMENTS

This research was sponsored by NASA Prime Contract 9-02078. We appreciate the efforts of the volunteer research subjects and medical monitors who made this collection of data and analyses possible. Jason Norcross and Lesley Lee of Wyle in Houston provided professional technical support, and Frances Laue of San Antonio edited for clarity, flow, spelling, etc. Opinions, interpretations, conclusions, and recommendations are those of the authors and are not necessarily endorsed by NASA or the United States Air Force.

Authors and affiliations: James T. Webb, Ph.D., M.S., and Larry P. Krock, Ph.D., M.A., USAF School of Aerospace Medicine, San Antonio, TX; and Michael L. Gernhardt, Ph.D., M.S., NASA Johnson Space Center, Houston, TX.

#### REFERENCES

- 1. U.S. Air Force. Air Force Instruction 10-248. Fitness program, ATCH. Washington, DC: U.S. Air Force; 2006; 9:58–61.
- ue, 2.<sup>2</sup>American College of Sports Medicine. ACSM's guidelines for Medicaexercise testing and prescription, 7th ed., chapter 4. Indianapolis, by Inger IN: American College of Sports Medicine; 2006:62–3.
  - Brooks GA, Fahey TD, Baldwin KM. Exercise physiology: human bioenergetics and its applications, 4th ed. New York: McGraw-Hill; 2004.
  - Dixon GA, Adams JD, Harvey WT. Decompression sickness and intravenous bubble formation using a 7.8 PSIA simulated pressuresuit environment. Aviat Space Environ Med 1986; 57:223–8.
  - Fryer DI. Subatmospheric decompression sickness in man. Neuilly-sur-Seine, France: NATO AGARD; 1969. AGARDograph #125.
  - Inderbitzen RS, DeCarlis JJ Jr. Energy expenditure during simulated EVA workloads. Warrendale, PA: SAE; 1986. SAE Technical Paper #860921, 16th ICES, San Diego, CA; 1986.
  - Jauchem JR. Effects of exercise on the incidence of decompression sickness: a review of pertinent literature and current concepts. Int Arch Occup Environ Health 1988; 60:313–9.
  - Kannan N, Raychaudhuri DA, Pilmanis AA. A loglogistic model for altitude decompression sickness. Aviat Space Environ Med 1998; 69:965–70.
  - 9. Kaufman WC, Callin GD, Harris CE. Energy expenditure of pilots flying cargo aircraft. Aerosp Med 1970; 41:591–6.
  - 10. Littell DE, Joy RJT. Energy cost of piloting fixed- and rotary-wing aircraft. J Appl Physiol 1969; 26:282–5.
  - McLaughlin JE, King GA, Howley ET, Bassett DR Jr, Ainsworth BE. Validation of the COSMED K4b<sup>2</sup> Portable Metabolic System. Int J Sports Med 2001; 22:280–4.
  - Pilmanis AA, Olson RM, Fischer MD, Wiegman JF, Webb JT. Exercise-induced altitude decompression sickness. Aviat Space Environ Med 1999; 70:22–9.

- Pilmanis AA, Petropoulos L, Kannan N, Webb JT. Decompression sickness risk model: development and validation by 150 prospective hypobaric exposures. Aviat Space Environ Med 2004; 75:749–59.
- Waligora JM, Horrigan DJ Jr, Conkin J. The effect of extended 02 prebreathing on altitude decompression sickness and venous gas bubbles. Aviat Space Environ Med 1987; 58:A110–2.
- Waligora JM, Kumar KV. Energy utilization rates during Shuttle extravehicular activities. Acta Astronaut 1995; 36:595–9.
- Webb JT. Documentation for the USAF School of Aerospace Medicine Altitude Decompression Sickness Research Database. Brooks City-Base, TX: USAFSAM; 2010. AFRL-SA-BR-SR-2009-0007.
- Webb JT. Guide to altitude decompression sickness research. Brooks City-Base, TX: USAFSAM; 2010. AFRL-SA-BR-SR-2009-0008.
- Webb JT, Beckstrand DP, Pilmanis AA, Balldin UI. Decompression sickness during simulated extravehicular activity: ambulation vs. non-ambulation. Aviat Space Environ Med 2005; 76: 778–81.
- 19. Webb JT, Fischer MD, Heaps CL, Pilmanis AA. Exercise-enhanced preoxygenation increases protection from decompression sickness. Aviat Space Environ Med 1996; 67:618–24.

- Webb JT, Kannan N, Pilmanis AA. Gender not a factor for altitude decompression sickness risk. Aviat Space Environ Med 2003; 74:2–10.
- Webb JT, Pilmanis AA. Altitude decompression sickness risk prediction. SAFE J 1995; 25:136–41.
- 22. Webb JT, Pilmanis AA. Preoxygenation time versus decompression sickness incidence. SAFE J 1999; 29:75–8.
- 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.
- Webb JT, Pilmanis AA, Balldin UI, Fischer JR. Altitude decompression sickness susceptibility: Influence of anthropometric and physiologic variables. Aviat Space Environ Med 2005; 76:547–51.
- Webb JT, Pilmanis AA, Kannan N, Olson RM. The effect of staged decompression while breathing 100% oxygen on altitude decompression sickness. Aviat Space Environ Med 2000; 71: 692–8.
- Wiegman JF, Ohlhausen JH, Webb JT, Pilmanis AA. Validation of dual-cycle ergometer for exercise during 100% oxygen prebreathing. 29th Annual SAFE Symposium Proceedings; November 2-4, 1991; Las Vegas, NV. Creswell, OR: SAFE; 1992:231–5.



IP: 139.169.20.187 On: Tue, 24 Apr 2018 13:46:11 Copyright: Aerospace Medical Association Delivered by Ingenta