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14. ABSTRACT
Introduction: The objective of this study was to determine the effect of exercise after altitude exposure (post-exposure exercise) on subsequent altitude decompression sickness (DCS) incidence. Existing USAF prohibition of exercise following altitude chamber training exposures and interest from operational personnel prompted our evaluation of post-exposure exercise as a DCS-inducing stressor. Methods: After a 1-h resting preoxygenation, 67 subjects were exposed to 30,000 ft for 2-h while performing mild, upper body exercise. The subjects were monitored for venous gas emboli (VGE) with an echo-imaging system and observed for signs and symptoms of DCS. Subjects without DCS (N=31) or with DCS which resolved during recompression (N=29) were randomly assigned to post-exposure rest (control, N=29) or moderate exercise (50% of peak oxygen uptake, dual-cycle ergometry; N=31) and both groups were monitored for delayed or recurring DCS. Results: The altitude exposure resulted in 48.3% DCS in the 60 volunteers serving as test or control subjects. Of 31 subjects assigned to the post-exposure exercise group, 15 had developed DCS which resolved during descent. No cases of DCS were observed or reported during or following post-exposure exercise. Conclusion: The results show that moderate exercise after exposure did not result in either delayed-onset or recurring DCS.

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

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Moderate Exercise After Altitude Exposure Fails to Induce Decompression Sickness

JAMES T. WEBB, ANDREW A. PILMANIS, AND MICHELE D. FISCHER


Introduction: The objective of this study was to determine the effect of exercise after altitude exposure (post-exposure exercise) on subsequent altitude decompression sickness (DCS) incidence. Existing USAF prohibition of exercise following altitude chamber training exposures and interest from operational personnel prompted our evaluation of post-exposure exercise as a DCS-inducing stressor. Methods: After a 1-h rest exercise (50% of peak oxygen uptake, dual-cycle ergometry; n = 31) and both groups were monitored for delayed or recurring DCS. Results: The altitude exposure resulted in 48.3% DCS in the 60 volunteers serving as test or control subjects. Of 31 subjects assigned to the post-exposure exercise group, 15 had developed DCS which resolved during descent. No cases of DCS were observed or reported during or following post-exposure exercise. Conclusion: The results show that moderate exercise after exposure did not result in either delayed-onset or recurring DCS.

Keywords: altitude, DCS, VGE, emboli, decompression sickness, exercise, denitrogenation, preoxygenation, prebreathe.

ANECDTAL REPORTS OF DCS presumably arising from post-exposure exercise have prompted our research on the possible relationship. U.S. Air Force personnel are taught that exercise performed very soon after an altitude exposure may cause DCS to occur or to recur and could become a serious factor in mission success or failure.

Research at Brooks AFB (Air Force Research Laboratory, AFRL) over the past 20 yr has allowed accumulation of a large database on altitude DCS (AFRL DCS Research Database) including extensive data on DCS symptoms and venous gas emboli (VGE). The vast majority of symptoms occur while at altitude. The AFRL Hypobaric DCS Research Database shows 33 of 2810 exposures with delayed-onset or recurring DCS symptoms. None of these delayed-onset or recurring DCS symptoms were provoked by exercise after exposure. Some reports from the literature also indicate occasional delayed-onset or recurring altitude DCS symptoms (8,3).

It is generally accepted that gas bubbles are the cause of DCS symptoms (4). It is also commonly accepted that the large majority of altitude DCS symptoms are caused by extravascular gas bubbles (8), not circulating VGE (8,2) which we routinely observe during altitude exposures using echocardiography. In previous studies, we have also observed VGE following recompression to ground level, although they were not associated with recurring or delayed DCS symptoms. The presence of VGE does not predict DCS because DCS without bubbles and bubbles without DCS are common occurrences (9). However, VGE resident at altitude or at ground level after exposure provide an objective measure of overall decompression stress.

During any operational altitude exposure, exercise may be involved in any of the three phases: before exposure, during exposure, and post-exposure. Exercise during preoxygenation provides protection from DCS (11,12). The literature is replete with reports of exercise increasing DCS incidence during exposure to altitude (4,7,9,13); however, we could find no citations in the research literature about the effects of post-exposure exercise on DCS based on actual experimental trials. Some interpretations of post-exposure exercise effects on DCS may have been based on the effects of exercise after decompression from diving (6), which is analogous to exercise during altitude decompression. Both decompressions involve supersaturation of body tissues with nitrogen, whereas recompression from altitude to ground level represents cessation of nitrogen supersaturation. Despite the lack of any experimental evidence, the U.S. Air Force restricts personnel from physical exercise for 12 h following hypobaric chamber training exposures (1).

This study tested the effect of post-exposure exercise on incidence and severity of post-exposure VGE and DCS symptoms in comparison to a resting post-exposure

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EXERCISE AFTER ALTITUDE EXPOSURE—WEBB ET AL.

TABLE I. SUBJECT ANTHROPOMETRIC (MEAN ± SD).

<table>
<thead>
<tr>
<th>Subject, N</th>
<th>Age, yr</th>
<th>Height, cm</th>
<th>Weight, kg</th>
<th>BMI (wt/ht. m²)</th>
<th>VO₂peak, ml kg⁻¹ min⁻¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>60 Resting</td>
<td>31.6 ± 6.2</td>
<td>176.7 ± 7.6</td>
<td>82.9 ± 11.7</td>
<td>26.5 ± 2.7</td>
<td>42.5 ± 6.9</td>
</tr>
<tr>
<td>29 Resting</td>
<td>31.6 ± 5.5</td>
<td>176.3 ± 7.3</td>
<td>82.4 ± 9.7</td>
<td>26.5 ± 2.6</td>
<td>42.8 ± 6.5</td>
</tr>
<tr>
<td>30 Exercising</td>
<td>31.6 ± 7.0</td>
<td>177.2 ± 8.0</td>
<td>83.3 ± 13.4</td>
<td>26.4 ± 2.9</td>
<td>42.3 ± 7.3</td>
</tr>
</tbody>
</table>

*All subjects who entered resting vs. exercising decision tree (did not have symptoms at ground level) (see Fig. 1).

control. The hypothesis was that post-exposure exercise would result in delayed or recurring cases of DCS following an altitude exposure resulting in approximately 50% DCS. With 50% incidence available for either increased or decreased DCS incidence due to post-exposure exercise, a statistical difference was more feasible to obtain.

METHODS

The voluntary, fully informed consent of the 67 male subjects (Table I) used in this research was obtained in accordance with Air Force Instruction 40-402 (1) and a protocol approved by the Brooks IRB. All subjects passed an appropriate physical examination and were representative of the USAF rated aircrew and NASA EVA crew population. Subjects were not allowed to participate in scuba diving, hyperbaric exposures, or flying for at least 72 h before each scheduled altitude exposure. Seven of the subjects who developed DCS were removed from the study prior to beginning post-exposure test or control activity due to referral for possible hyperbaric oxygen therapy to alleviate symptoms (Table II; Fig. 1). Anthropometric and other subject information is shown in Table I for the 60 volunteers who became either test or control subjects.

Prior to each 2-h, 30,000-ft research chamber exposure, a physician conducted a short physical examination of subjects to identify any signs of illness or other problem that would endanger the subject or bias the experimental results. Breathing gas during the 1-h preoxygenation, while decompressed, and during desent was 100% oxygen (aviator’s breathing oxygen; normal analysis 99.7–99.8% oxygen). A neck-seal respirator made by Intertechnique® (Plaisir Cedex, France) was used to deliver oxygen. This mask provided a slight (2 cm of water) positive pressure, which reduced the opportunity for inboard leaks of nitrogen from the atmosphere and was more comfortable than the standard aviator’s mask.

At 16-min intervals during the exposure, the subjects were monitored for VGE using a Hewlett Packard Sonos 1000 Doppler/Echo-Imaging System. This system permits both audio and visual monitoring and recording of gas emboli in all four chambers of the heart and allows for easier and more accurate determination of emboli presence than Doppler alone. Detection of any left ventricular gas emboli (LVGE) was made possible due to these echo-imaging sessions and was used for immediate recompression to avoid potential serious symptoms resulting from arterial gas emboli. VGE were graded using a modified Spencer Scale (10) where Grade I is observation of any VGE, the criteria used for determination of the time of VGE onset and percent incidence reported in Table III and Fig. 2.

During exposure, subjects performed three, mild, upper-body exercises (rope pull, torque wrench, and arm crank) as described in detail in Webb et al. (12) and represented approximately 15–20% of maximal oxygen uptake (VO₂peak). The subjects walked less than 10 steps between exercise stations and the echo-imaging station at 4-min intervals.

Endpoints (test termination criteria) of the exposures

TABLE II. SYMPTOMS EXPERIENCED BY 36 OF THE 67 SUBJECTS.

<table>
<thead>
<tr>
<th>Profile</th>
<th>Symptom Description</th>
<th>Subjects Affected, N</th>
<th>Onset Time‡</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Joint Pain</td>
<td>12</td>
<td>56 (26–95)</td>
</tr>
<tr>
<td>A</td>
<td>Pain, other</td>
<td>1</td>
<td>85</td>
</tr>
<tr>
<td>A</td>
<td>Tingling and joint pain***</td>
<td>1</td>
<td>61</td>
</tr>
<tr>
<td>B</td>
<td>Joint Pain</td>
<td>13</td>
<td>70 (36–114)</td>
</tr>
<tr>
<td>B</td>
<td>Tingling</td>
<td>1</td>
<td>47</td>
</tr>
<tr>
<td>B</td>
<td>Dyspnea and tingling</td>
<td>1</td>
<td>24</td>
</tr>
<tr>
<td>C</td>
<td>Skin Motting,*** Dizziness, Fatigue,*** Light Headedness,*** Joint Pain,*** Nausea,*** Headache***</td>
<td>1</td>
<td>65</td>
</tr>
<tr>
<td>C</td>
<td>Substernal Distress++</td>
<td>1</td>
<td>27</td>
</tr>
<tr>
<td>C</td>
<td>Pins &amp; Needles Hot/cold sensation++</td>
<td>1</td>
<td>45</td>
</tr>
<tr>
<td>C</td>
<td>Joint Pain</td>
<td>1</td>
<td>42</td>
</tr>
<tr>
<td>C</td>
<td>Hot/cold sensation++, Pruritus++, Joint Pain, Skin Motting++</td>
<td>1</td>
<td>38</td>
</tr>
<tr>
<td>C</td>
<td>Joint Pain, Muscle Pain++</td>
<td>1</td>
<td>88</td>
</tr>
</tbody>
</table>

*†, ‡A, post-exposure rest; B, post-exposure exercise; C, removed from study due to HBO therapy, post-exposure breathing of 100% oxygen, or presence of LVGE.

*Subject presented with left ventricular gas emboli (LVGE).

†Received hyperbaric oxygen therapy (HBO).

‡Symptom present after recompression.

+++Symptom recurred after recompression.

§Onset time = Median of first symptom (range) in min.

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were: 1) completion of the 2-h scheduled exposure; 2) development of any DCS signs or symptoms including respiratory symptoms, neurologic symptoms, paresthesia, and constant pain; 3) detection of LVGE. On recompression to ground-level pressure, subjects with no DCS symptoms or with resolution of DCS symptoms doffed their oxygen masks, began breathing room air, and were examined by a physician to determine if referral for examination by a hyperbaric treatment physician was necessary. After the physician ensured that any or all symptoms were alleviated, the 60 subjects who either did not develop DCS symptoms (n = 31) or whose symptoms resolved on descent (n = 29) were randomly divided into two groups; exercising (n = 31) or resting (n = 29) (Fig. 1). The resting group served as the control for the exercising test group. Chi square tests were used to compare results from the control and test groups (level of confidence, p < 0.05).

The post-exposure exercise consisted of three, 15-min periods of 60 rpm dual-cycle ergometry at 50% of \( \dot{V}_{O_2} \text{peak} \), beginning 5 min after recompression. The dual-cycle ergometer consisted of a Monarch® 818 leg ergometer (Varberg, Sweden) and a Monarch® 881 Rehab Trainer mounted so the subject could sit upright pedaling the 818 leg ergometer while reaching forward to operate the 881 arm ergometer. To determine the appropriate resistance settings and target heart rate during the post-exposure moderate exercise periods, subjects were tested to \( \dot{V}_{O_2} \text{peak} \) (mean R value > 1.1) with dual-cycle ergometry. The post-exposure exercise was accomplished while breathing room air and neither exercising nor resting subjects were restricted from normal activity, including walking and eating. The exercise periods were separated by 15 min of rest that included VGE monitoring as necessary to determine when VGE resolved at ground level. Resting subjects were also monitored for VGE to determine time of resolution. Due to equipment and personnel limitations, the resting subjects were monitored for post-exposure VGE 10 min earlier than the exercising subjects.

RESULTS

No DCS symptoms resulted from post-exposure exercise after this provocative altitude exposure. Table II shows the symptoms developed during the exposures and their disposition. The subjects removed from the test and control groups clearly had developed symptoms which required at least precautionary post-breath with 100% oxygen. Table III and Fig. 1 show the DCS and VGE results of the exposure for the 60 subjects who entered the decision tree to determine post-exposure activity (Fig. 1).

Post-exposure VGE monitoring indicated 13 resting subjects and 8 exercising subjects with VGE during the first monitoring period, which occurred 10 min into the resting subjects' post-exposure activity and 20 min into the exercising subjects' post-exposure activity. No significance is attached to the difference due to timing of

![Fig. 2. VGE (upper, grey line) and DCS (lower, black line) of 60 subjects (exercising and resting pos-exposure groups).](image-url)
monitoring. None of the exercising subjects had VGE remaining by the second monitoring period, which began 50 min after recompression. One resting subject had VGE still present during the third monitoring period, which began 68 min after recompression. These post-exposure findings are consistent with data from other studies resulting in similar levels of DCS (AFRL DCS Research Database).

DISCUSSION

The failure of moderate exercise after recompression to elicit DCS was unexpected and counter to the hypothesis. Recompression apparently reduced the size and number of gas bubbles remaining at the end of the exposures to non-symptomatic levels under the conditions of this experiment. The provocative nature of this experiment, yielding 76.7% VGE and 48.3% DCS, should have made more subjects susceptible to any DCS-causing stress after exposure than would a more operationally relevant exposure with much less DCS risk. The rapid resolution of VGE following two, 15-min periods of moderate exercise at ground level tends to indicate that moderate exercise at ground level, after decompression, does not increase DCS or VGE incidence.

CONCLUSIONS

No cases of recurring or delayed DCS occurred with post-exposure exercise, suggesting that exercise after decompression from altitude exposure does not increase DCS risk.

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Opinions, interpretations, conclusions, and recommendations are those of the author and are not necessarily endorsed by the United States Air Force.

REFERENCES