



Exercise Following Simulated Parachuting from 35,000 Feet: Is Tolerance Reduced and is the Probability of Post Descent Decompression Sickness Increased?

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ABSTRACT

Developments in parachutes and life support systems now allow the insertion of military parachutists from very high altitudes. This study was designed to examine, and to attempt to quantify, the effect of High Altitude High Opening (HAHO) parachute deployment on the risk of DCS and exercise tolerance both during and after descent, in the most realistic manner. Ten experienced military parachutist subjects were exposed to two altitude chamber profiles in balanced order in a hypobaric chamber on separate occasions: a 'control' exposure to 17,500ft and a 'test' exposure to 35,000ft followed by a slow descent to ground level. This was followed immediately by 45 minutes of strenuous exercise with subjects carrying a heavy Bergen. The parachutists were monitored throughout for venous gas emboli (VGE), symptoms of decompression sickness (DCS) and subjective and objective measures of exercise performance. No VGE or symptoms of DCS were observed in any subject at any stage of either exposure to 35,000ft when compared to 17,500ft.

1.0 INTRODUCTION

Developments in parachutes and life support systems now allow the insertion of military parachutists from very high altitudes. High Altitude High Opening (HAHO) parachute deployment offers many operational advantages; however, parachuting from altitudes in excess of 25,000 feet carries an increased risk of decompression sickness (DCS). DCS occurs as a result of dissociation of nitrogen from the tissues, which leads to the formation of gas bubbles. These bubbles can obstruct circulation, disrupt tissues by distension, and alter biochemical and haematological balances, resulting in a range of clinical symptoms progressing from local joint pain to neurological or respiratory disturbance.

The risks of developing DCS are minimised by 'denitrogenation', the removal of dissolved nitrogen from tissues and fluids of the body. This is achieved by pre-breathing 100% oxygen, or preoxygenation, for a period of time prior to exposure to reduced pressure. This normally takes place in the dispatch aircraft, both on the ground and during transit to the drop zone, from where the parachutist descends with their portable life support system, which continues to deliver 100% oxygen. However, with increasing parachute opening altitudes, slow descent rates and cold temperatures the risk of DCS may be increased despite preoxygenation.

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It has been shown that the presence of circulating venous gas emboli (VGE) generally precedes the onset of symptoms of decompression sickness (Balldin & Borgstom, 1976 and Olson *et al.*, 1988). Although VGE are not a sign of DCS their detection, by 2D or Doppler echocardiography, is a positive indicator that the individual concerned is in a state of nitrogen super-saturation, the primary factor in the development of DCS.

It has been demonstrated that moderate exercise at altitude increases venous gas emboli and incidence of DCS symptoms (Ferris & Engel, 1951 and Krutz & Dixon, 1987). Though not fully understood, exercise (especially vigorous exercise) is thought to enhance bubble formation through tribonucleation and vacuum phenomena. It is known, from echocardiography, that venous gas emboli can occasionally be detected after recompression from altitude to ground level, indicating incomplete resorption of gas during re-compression. Whether exercise after recompression could increase the risk of resurgence of bubbles by tribonucleation or the development of symptoms of DCS is not known.

Some military personnel are instructed to refrain from exercise after exposure to high altitude. This recommendation may have its origins in the interpretation of the effects of exercise after decompression from diving (Fulton, 1951). Exercise following diving is equivalent to exercise *during* altitude-decompression, where the body is supersaturated with nitrogen. The recommendation to refrain from exercise following descent may thus prove unnecessary. Investigation of the effects of exercise restrictions impractical, especially when they may be required to rapidly cover much ground on foot, carrying a very heavy load, immediately following landing. Any symptoms of DCS would adversely affect their ability to do this, and small units would be unwilling to leave mission-critical members of the team behind.

Webb *et al.* (2002) have previously undertaken work to investigate the effects of post altitude exposure exercise. Following an altitude exposure of 30,000 feet and 60 minutes of prior preoxygenation, they observed no cases of DCS amongst exercising or resting subjects. No resurgence of VGE was observed in any of their subjects at ground level and any VGE persisting from the altitude exposure were no longer present 50 minutes after reaching ground level. This appeared to indicate that moderate to strenuous exercise after exposure to altitude would not elicit resurgence in venous gas emboli and would not cause recurring or delayed DCS after exposure to altitude, when compared with ground level rest.

It is known, however, that the incidence of DCS increases with altitude. It is also known that exercise at altitude corresponds with increased occurrence of VGE, possibly promoting greater denitrogenation at altitude. Exercise, during preparation and descent during HAHO operations, however, is generally minimal, compared to that employed by Webb *et al.* Finally, Webb *et al.* (2002) primarily looked at the possibility that exercise might increase the incidence of VGE at ground level. However, they used a level of exercise considerably lower than what would be expected of military parachutists following landing. In addition, little consideration was given to the possibility that altitude induced VGE might alter an individual's tolerance to exercise at ground level. It was therefore thought necessary to extend this work using altitudes and exercise levels, which might be associated with hypothetical mission profiles.

The aim of this trial was twofold:

- to assess the incidence of Venous Gas Emboli (VGE) and Decompression Sickness (DCS) during the altitude and post descent phase of a hypothetical simulated HAHO mission at a representative altitude of 35,000 ft.
- to assess whether an individuals subsequent ground level exercise tolerance might be effected by the preceding exposure to a potentially DCS inducing altitude.



2.0 Method

Ten highly experienced and fit parachutists with an average age of 27.9 years were used in the study. Variability in the incidence of VGE and susceptibility to DCI between individuals required that subjects act as their own control and that any subsequent analysis was made on an intra-subject basis. Following satisfactory completion of ground level training subjects were exposed to two 'single blind' altitude chamber profiles in balanced order in a hypobaric chamber: a 'control' exposure to 17,500ft and a 'test' exposure to 35,000ft, separated by 48hrs. The selection of 17,500 feet as the control exposure was because this being below the traditionally accepted threshold for DCS and yet was high enough to allow the use of a profile that would be subjectively difficult to distinguish from the test exposure for the parachutists. Presentations of 'test' and 'control' profiles were randomly assigned to each subject and subjects performed both profiles at the same hour of the day to eliminated any circadian effects.

For both exposures subjects pre-oxygenated at ground level for one hour, and were decompressed to the chosen peak altitude (35,000 ft or 17,500 ft) over seven minutes, where they remained for fifteen minutes, before being recompressed to ground level at a constant descent rate (1,000 ft min⁻¹ or 500 ft min⁻¹) over 35 minutes. Subjects continued to breathe 100% oxygen throughout. During descent subjects were suspended by a military parachute harness from a specially constructed frame so as to reproduce the effect parachute harness straps may have on blood flow in the lower limb during descent and any possibly effect on the development of VGE and DCS.

Descent was followed immediately by 45 minutes of ground level exercise comprising a 4km.hr⁻¹ route march on a treadmill inclined at 0.5[°] carrying a Bergen weighing 40kg (88 lbs). Subjects were monitored for VGE using precordial 2D and Doppler echocardiography (Hewlett Packard SONOS 2500) after ten minutes at peak altitude, twice during descent, again on arrival at ground level and every 15 minutes thereafter. Any VGE present were graded according to the Spencer Scale (Spencer, 1976). Subjects were also instructed to report any symptoms or sensations that developed during exposure to altitude, or subsequently at ground level, that they were not previously experiencing before commencement of the experiment.

During the altitude phase all subjects were monitored with continuous ECG recording, pulse oximetry (Kontron Instruments 7840), and breath by breath respiratory mass spectrometry (Innovision AMIS 2000). Mass spectrometry was used primarily to ensure that 100% oxygen was being breathed at all times and to indicate any reduction in end-tidal CO_2 due to hyperventilation.

During ground level exercise heart rate was monitored and recorded using a telemetric heart rate monitoring system (Polar Vantage NV). During the final 90 seconds of each 15 minute exercise period, expired gas was collected in Douglas bags via a mouthpiece for subsequent volumetric analysis using a water sealed gasometer (Collins 1201) and mass spectrometer (Innovision AMIS 2000). Oxygen uptake rate or VO₂ was subsequently calculated using the methodology and formula described by Turner & Hoppeler (1999). In addition, the parachutists were asked to indicate how hard they felt they were working using the Subjective Rating of Perceived Exertion (RPE) Scale (Borg, 1982). Data were analysed using Analysis of Variance (ANOVA) to determine any differences in tolerance to exercise. Where significant differences were indicated, Newman Keuls post hock tests were performed to detail the nature and extent of the variability.

3.0 Results

During both the altitude and subsequent post descent exercise phases no VGE were observed in subjects at any time during or following exposure to either the 'test' (35,000 ft) or 'control' (17,500 ft) profiles. Furthermore, no subjects displayed signs or reported any symptoms consistent with DCS during either 'test' or 'control' profiles.



Analysis of Variance test (ANOVA) indicated no significant effect of previous altitude or exercise time on RPE scale reports. The subjective (Borg) rating of perceived exertion (RPE) therefore appeared to be unaltered by previous altitude exposure or by exercise duration.

There was a significant effect of time (p<0.05) on heart rate (HR). The Newman-Keuls post hock test indicated that heart rate was significantly higher (p<0.05) during the last fifteen-minute exercise block than the 1st after both 'Control' (17,500 ft) and 'Test' (35,000 ft) altitude exposures. However, there was no significant difference between these rises in heart rate after exposure to control or test altitudes.

There was a significant effect of time (p<0.05) on VO₂. The Newman-Keuls test indicated VO₂ was significantly higher (p<0.05) during the 2^{nd} fifteen-minute exercise period than the 1^{st} , while the 3^{rd} fifteen minute exercise block was significantly higher (p<0.001) than both the 1^{st} and 2^{nd} fifteen-minute exercise blocks, following both 'Control' (17,500 ft) and 'Test' (35,000 ft) altitude exposures. However, there was no significant difference between these rises in VO₂ after exposure to control or test altitudes.

The order in which altitude profiles were presented to individuals was seen to have no effect on any of the measurements taken.

The random effect; subject, was not observed to have any effect on any of the results.

4.0 Discussion

This study aimed to compare the incidence of VGE and DCS symptoms during exposure to two simulated altitude profiles and subsequent post-descent exercise. It also compared individuals post-descent exercise tolerance following exposure to the different altitude profiles.

It is known that the incidence of venous gas emboli and symptoms of decompression sickness increase with altitude. Previous work performed by Lee & Hay (2001) at the Defence Evaluation Research Agency, now QinetiQ, Farnborough, observed VGE in 93% of individuals exposed to simulated altitude of 35,000 ft while breathing 100% O_2 after preoxygenating for a period of 1 hour. Furthermore symptoms consistent with DCS were reported in 53% of the subject population. However, this work employed prolonged exposure to altitude, with the first VGE being observed after 30 minutes and onset of first symptom of DCS occurring after 58 minutes. By contrast, the current study exposed parachutists to a much shorter, though perhaps more representative, duration of time (15 minutes) at the highest altitude followed by a simulated descent of 1,000 feet min⁻¹ which meant that subjects were back to a less hazardous (and less VGE inducing) altitude of 20,000 feet, 30 minutes after the onset of exposure to 35,000 feet. This study's failure to find any evidence of VGE or DCS with the preoxygenation regime and altitude profile used is therefore entirely consistent with the previous work of Lee (2001).

Work performed by Webb *et al.* (2002) appeared to indicate that exercise at ground level would not trigger a resurgence in VGE or symptoms of DCS following a two hour exposure to an altitude of 30,000 ft. Our results are in accordance with their work in that there was no evidence of VGE at any time during or after strenuous exercise subsequent to exposure to an altitude of 35,000 feet. In addition, there was no evidence of any symptoms of DCS at any time.

Similarly, exposure to the 35,000 feet profile had no effect on the parachutist subjects ability to tolerate the strenuous exercise of carrying a 40 kg (88 lbs) Bergen at 4km.hr⁻¹ up an incline of 0.5° for 45 minutes. There were no significant differences in any of the variables measured, both objective and subjective, to determine any difference in exercise tolerance irrespective of whether the subjects had been exposed to the test or control altitude previously. This indicates that limited exposure to 35,000 ft using the profile described above had no greater effect on subsequent ground level exercise tolerance than prior exposure to 17,500 ft. Some differences were found over time between the final 15-minute block of



exercise and previous blocks of exercise following both 17,500 ft and 35,000 ft simulated HAHO parachute deployments. It is assumed that this is a result of the cumulative effect of fatigue during the 45 minute Bergen carry but there were no differences between the results seen after exposure to either 17,500 ft or 35,000 ft.

It should be noted that temperature differences due to altitude could not be simulated and hence were not considered during this assessment. At an altitude of 35,000ft the ambient temperature is generally in the region of -56° Celsius and rises by approximately 2 °C for every 1,000 ft descent. Cold temperatures are thought to increase susceptibility to DCS though the precise mechanism is not clearly understood. However, without adequate insulating clothing, parachutists may be at greater risk during live jumps than in a warm hypobaric chamber.

Various other factors, such as age and increased Body Mass Index (BMI) may also influence an individual's susceptibility to DCS. Analysis of existing hypobaric research reveals a trend towards increased susceptibility to DCS with age, particularly for those older than 42 (Macmillan, 1999). The incidence of DCS is also observed to be greater in obese individuals.

The results of this study would therefore have greatest validity for this particular scenario, which employed young (average age 27.9 years) fit individuals in a simulated hypothetical HAHO descent from 35,000 ft followed by a period of prescriptive ground level exercise.

5.0 Conclusions

Simulated HAHO exposures to 35,000ft in a hypobaric chamber, following one hour pre-oxygenation at ground level, showed no evidence of any VGE or DCS in any subjects at any time, either at altitude or subsequently on the ground. Heavy exercise on the ground after altitude exposure did not result in any evidence of VGE or DCS, either during or after the 45 minutes of carrying 40kg (88lbs) at 4km.hr⁻¹ up an incline of 0.5⁰. Finally, there was no difference in exercise tolerance as indicated by the objective and subjective variables measured, irrespective of whether subjects had been exposed to the 35,000 ft profile or the 17,500 ft profile immediately before. On the assumption that parachutists preoxygenated for 1 hour these data suggest that the risks of both VGE and DCS during a hypothetical HAHO profile to 35,000 feet, or post descent DCS or VGE associated with subsequent heavy exercise, are perhaps overstated. Exercise tolerance is unlikely to be affected, or to result in any increase in the risk of post-descent DCS. Therefore the advice given to some parachutists to restrict physical activity following HAHO jumps from high altitudes may be unnecessary.

6.0 References

[1] BALLDIN U. I. & BOGSTROM P. (1976). Intracardial bubbles during decompression to altitude in relation to decompression sickness in man. *Aviat. Space Environ. Med.* **47(2)**: 113-116

[2] BORG G.A., (1982). Physiological bases of physical exertion. *Medicine and Science in Sports and Exercise*. **14**: 377-381

[3] FERRIS E.B. & ENGEL G.L. (1951). The Clinical nature of high altitude decompression sickness. In: *Decompression Sickness*. pp 4-52. Ed. Fulton J.F. W.B. Saunders Company. Philadelphia.

[4] FULTON J.F. (1951). Ed. *Decompression Sickness*. W.B. Saunders Co., Philadelphia & London.; 437pp.



[5] HAYWARD A.T.J. (1967) Tribonucleation of bubbles. Br. J. Appl. Phys. 18: 641-644.

[6] KRUTZ R.W. & DIXON G.A. (1987). The effects of exercise on bubble formation and bends susceptability at 9,100 m (30,000 ft; 4.3psia). *Aviat. Space Environ. Med.* **58** (9,Suppl): A97-9.

[7] LEE V.M. & HAY A.E. (June 2001) Altitude Decompression Illness – The Operational Risk at Sustained Altitudes up to 35,000 ft *RTO meeting Proceedings MP 062 Operational Medical Issues in Hypo and Hyperbaric conditions*

[8] MACMILLAN A.J.F. (1999). Sub-atmospheric Decompression Sickness. In: Aviation Medicine. 3rd ed. Eds. Ernsting, Nicholson & Rainford. Butterworth Heinmann, London.

[9] OLSON R.M., KRUTZ R.W., DIXON G. A. & SMEAD K.W. (1988). An evaluation of precordial ultrasonic monitoring to avoid bends at altitude. *Aviat. Space Environ. Med.* **59**: 635-9.

[10] SPENCER M.P. (1976). Decompression limits for compressed air determined by ultrasonically detected blood bubbles. J. Appl. Phys. 40: 229-235.

[11] TURNER D.L. & HOPPELER H. (1999) Muscle energetics: aerobic strategies. In: Physiological Determinants of Exercise in Humans. *Studies in Physiology 4*. Eds. Whipp & Sargeant. Portland Press, London.

[12] WEBB J.T., & PILMANIS A.A. (1992). Venous gas emboli detection and endpoints for decompression sickness research. *Safe Journal*. **22**: 22-25.

[13] WEBB J.T., PILMANIS A.A. & FISCHER M.D. (2002). Moderate exercise after altitude exposure fails to induce decompression sickness. *Aviation Space Environ. Med.* **73(9)**: 872-5