Postural Control and Venous Gas Bubble Formation During Hypobaric Exposure

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Background: Earlier studies have shown that acute hypoxia at simulated altitudes up to 18,000 ft affects postural control. The main objective of this study was to investigate whether this is caused by hypoxia or by other effects of reduced barometric pressure. Doppler monitoring was included to rule out venous gas emboli (VGE) as a possible cause of disturbed postural control. A secondary objective was to evaluate two conventional altitude chamber training profiles regarding release of VGE. ***Hypothesis:*** Chamber flights up to 18,000 ft affect postural control due to acute hypoxia or other effects of reduced barometric pressure such as bubble formation. VGE probably will not be formed at the altitude chamber flight profiles and procedures selected for this study. ***Methods:*** Repeated registrations of postural control and Doppler monitoring for detection of possible VGE were performed on 12 subjects before, during, and after exposure to two different altitude chamber flight profiles. In chamber flight profile 1 the subjects were first preoxygenated for 45 min and then exposed to a normoxic environment at altitudes of 25,000, 18,000, 14,000, and 8000 ft. Chamber flight profile 2 consisted of an 80 min exposure to 14,000 ft without preoxygenation or supplemental oxygen for the first 60 min. ***Results:*** In chamber flight profile 1, where normoxic conditions were achieved during all balance testing, no significant changes in postural control were found. No VGE were observed and no subjective dizziness was reported during this exposure. In chamber flight profile 2, a significant influence on postural control was reported for the eyes-open condition, when breathing air at 14,000 ft. These changes normalized when reaching ground level. VGE were observed in one of the 12 subjects after 75 min at 14,000 ft. Another subject complained of severe dizziness during the initial part of the decompression to 14,000 ft, and was excluded from further experiments. ***Conclusions:*** Changes in postural control at altitudes up to 18,000 ft is probably due to acute hypoxia. VGE may form during acute altitude exposure to 14,000 ft.

Keywords: stabilometry, hypoxia, balance, altitude exposure, human, Doppler, VGE.

THE POSTURAL SYSTEM is highly complex and includes feedback loops from several sensory modalities (e.g., the vestibular system; vision; proprioception from joints, tendons, and muscles; and superficial and deep tactile sense interacting with the central nervous system). While the effects of accelerative forces on the vestibular system have been thoroughly investigated (5), the effects of hypobaric conditions on this system have attracted less attention.

Computerized stabilometry based on input from a static balance platform offers an objective, simple, sensitive, and quantitative method for measuring postural stability. It has been successfully used in normobaric (14), hypobaric (6,9,16,25), and hyperbaric (1–3,15) environments.

In a previous balance platform study we reported that acute hypoxia at simulated altitude exposures up to 18,000 ft impaired postural control. That experiment was performed on 16 military aircrew undergoing a refresher course in aviation medicine. Balance platform testing was done at 18,000, 14,000, and 8000 ft at the tail end of a standard aircrew altitude chamber training profile, with baseline registrations at ground level before the chamber flight. The chamber profile consisted of a 45-min preoxygenation at ground level, a 10-s decompression from 8 to 22,500 ft, and individual hypoxia testing for 2–5 min at 25,000 ft. No subjective dizziness and no clinical unsteadiness were noted. However significant changes in body sway were found on the balance platform during hypobaric exposure to 18,000, 14,000, and 8000 ft compared with the baseline registrations. The relative increase in sway movements was greater in the eyes-open condition compared with the eyes-closed condition, and significant for movements in the anteroposterior plane but not in the lateral plane. Most sway parameters returned to pre-exposure values on return to ground level (16). This was in agreement with other studies showing that vision is the first of the special senses to be altered by lack of oxygen.

The main objective of the present study was to investigate whether the observed postural instability at altitude, as shown in our previous study, was caused by hypoxia or by other effects of reduced barometric pressure such as bubble formation. We, therefore, repeated

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the same altitude chamber training profile to 25,000 ft as in the previous study, but this time normalizing oxygen saturation during the balance testing periods by means of a continuous flow oxygen system. Doppler monitoring was included to detect possible venous gas emboli (VGE), which could influence postural control.

Although 10,000 ft is generally accepted as the maximum cabin altitude without supplemental oxygen in military operations, many nations allow exposure to 14,000 ft for a limited period of time. In order to determine whether such an exposure causes impaired postural control or bubble formation, we also included a second chamber flight profile to 14,000 ft for 80 min without preoxygenation.

A secondary objective was to find out if the two selected altitude chamber training profiles would release VGE.

METHODS

The study was performed in cooperation with the University Hospital of Bergen, the Royal Norwegian Air Force (RNoAF) Institute of Aviation Medicine, Norwegian Underwater Intervention, and the Royal Norwegian Navy.

All 12 subjects (9 males and 3 females) were RNoAF aircrew or employees without any previous ear, balance, or hearing problems. Mean age was 35 yr (range 20–62). All participants passed a routine medical examination, including a health questionnaire and ECG. Informed consent to participate in the experiment was obtained from all subjects, and they were informed of their right to withdraw at any time. The study was approved by the National Committee for Research Ethics.

The experiments were performed in the hypobaric chamber complex of the RNoAF Institute of Aviation Medicine. The subjects were exposed to two different chamber flight profiles (profile 1 and 2) with more than a week between each exposure. None of the subjects had been exposed to diving or other hyperbaric environments for at least 2 wk prior to the chamber flights. The altitude references are according to the U.S. Standard Atmospheric Pressure Table. Two subjects were investigated during each chamber run. Baseline measurements of balance and Doppler were performed at ground level (270 ft above sea level) in the chamber prior to flight.

Altitude chamber flight profile 1 was a simulated 25,000-ft exposure commonly used in the RNoAF for aircrew training. All subjects were pre-oxygenated at ground level with 100% oxygen for 45 min using a demand mask and regulator. During balance testing, oxygen supply was switched from the demand system to a continuous flow system using a double nasal catheter, with a flow of 6 $L \cdot min^{-1}$ at the two highest altitude levels. The catheter was chosen to avoid interference with the balance testing from a heavy demand mask and hose.

Chamber flight profile 1 is illustrated in **Fig. 1**A. An initial fast decompression (ascent) to 8000 ft was followed by a 4000 $ft \cdot min^{-1}$ compression (descent) to

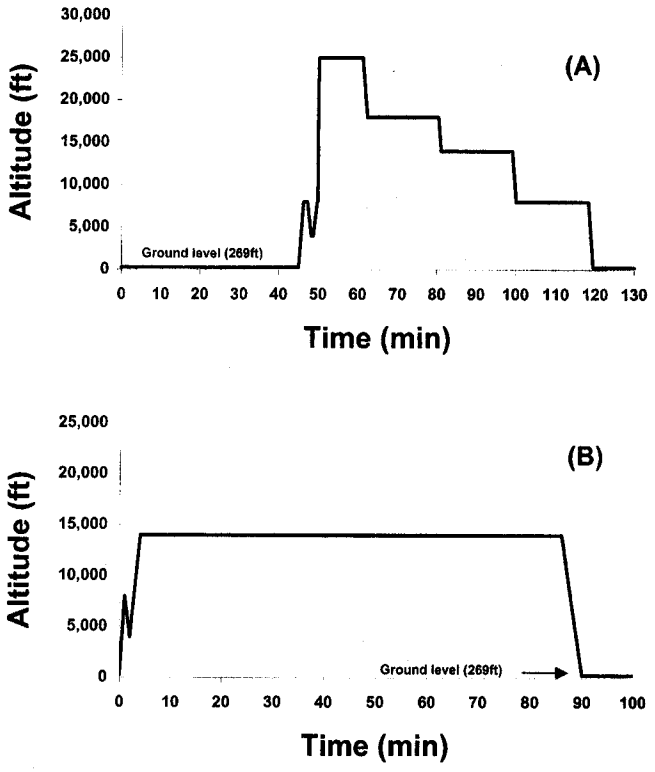

Fig. 1. Altitude chamber flight profile 1—up to 25,000 ft (A), and chamber flight profile 2 to 14,000 ft (B).

4000 ft to check for possible ear and sinus problems. The chamber was again decompressed to 8000 ft followed by a rapid decompression to 22,500 ft in 10 s, and a further ascent to 25,000 ft in 30 s. Exposure time at 25,000 ft was 11 min, as in our previous study. Due to time restraints, only half of the test subjects underwent balance testing at this altitude, while the other half were Doppler tested. The chamber altitude was then lowered to 18,000 ft, where balance testing was performed on all subjects. After 22 min, the chamber altitude was reduced to 14,000 ft for the next balance test. Oxygen flow through the nasal catheter was reduced to 4 $L \cdot min^{-1}$ during balance testing at 14,000 ft. After another period of 22 min at this altitude, testing was repeated at 8000 ft with an oxygen flow of 2 $L \cdot min^{-1}$. After 22 min at that altitude, descent to ground level was started. A final balance test was performed at ground level with the oxygen supply turned off. The total altitude exposure lasted for approximately 75 min.

Chamber flight profile 2 is illustrated in Fig. 1B. Here the subjects had an 80-min exposure to 14,000 ft without pre-oxygenation. Initial ascent was fast decompression to 8000 ft, followed by a 4000 $ft \cdot min^{-1}$ descent to 4000 ft. The chamber was then decompressed to 8000 ft, followed by a further ascent to 14,000 ft. No oxygen was given for the first 60 min at 14,000 ft. A continuous flow of 4 $L \cdot min^{-1}$ of oxygen was then provided through a double nasal catheter for the last 20 min at 14,000 ft. The total chamber flight lasted for approximately 90 min. Balance testing was performed before and after the chamber flight, and four times during the exposure. The

last balance test at 14,000 ft was performed after supplemental oxygen had been started.

During both chamber flight profiles heart rate and oxygen saturation were monitored by pulse oxymetry using a finger probe, except during periods of balance testing and Doppler monitoring.

Computerized stabilometry (posturography) used for measuring balance performance is non-invasive and causes no discomfort. It is widely used for clinical investigation of disturbances in the postural system. The procedure is described in further detail elsewhere (14,16). In our study, the subjects were instructed to stand quietly on a static balance platform with their feet 7 cm apart and the arms along their sides. Measurements were done for 1 min with the eyes open looking at a small eye-level target 2 m away, and for 1 min with the eyes closed. No training and only minimal instruction was needed. The tests were conducted under visually and acoustically standardized conditions in the altitude chamber.

A balance platform (Cosmogamma©, Via Zalloni, Bologna, Italy), measuring 40 × 40 cm, was used for data collection. The shift of the body's center of pressure (COP) at the soles of the feet during body sway was sensed by three mechanical-electrical transducers (strain gauges) in the platform. The signals were relayed by cable through a penetrator in the chamber wall to a computer (12 bit A/D resolution and 10 Hz sampling frequency) outside the altitude chamber. A monitor screen provided graphic and numerical presentations of different body sway characteristics.

The path length the COP described during each 1-min registration is determined by the gravitational force and the isometric muscle contractions, and thus related to the effort of the balance system in maintaining an upright posture. In addition, the mean speed of the corrective movements in the anteroposterior and lateral planes were chosen to evaluate the postural stability in the two planes.

The Romberg index (RI) is the ratio between measured parameters with closed and open eyes. It can be calculated for different parameters, such as the path length and the speed described by the COP. Usually, body sway will increase when closing the eyes, causing a detectable deterioration in performance. Accordingly, the RI will usually have a numerical value > 1.

Within subjects analysis of variance (ANOVA) with repeated measures was used to examine the various parameters describing the effect of the exposure on the postural system. When statistical significance was found ($p < 0.05$), Tukey HSD (Honest Significant Difference) post hoc comparison and Student's *t*-test were applied.

Possible subjective complaints of dizziness were noted by a trained physician in the chamber, and each subject was observed and asked for possible unsteadiness during testing.

Doppler monitoring was used to detect possible VGE. The testing was done before, at intervals of 15–20 min during, and shortly after the hypobaric exposure in both chamber flight profiles. During chamber flight profile 1, the Doppler monitoring was performed at 25,000, 18,000, 14,000, and 8000 ft. Due to time restraint, only half of the test subjects underwent Doppler testing at 25,000 ft, while the other half were tested on the balance platform at this altitude. During chamber flight profile 2, the monitoring was performed four times at 14,000 ft, the last measurement after supplemental oxygen had been started. A continuous Doppler instrument (Multi Dopplex II, Huntleigh Health Care, Cardiff, UK) was connected to a 2-MHz probe for precordial measurements. A 5-MHz probe was used for monitoring the subclavian veins. Monitoring was first performed with the subject standing quietly. In order to facilitate the release of stationary bubbles, the subject was asked to perform deep knee bends prior to precordial measurements and fist clenching during subclavian vein measurements. All measurements were tape recorded. Initial interpretation (presence or absence of venous microemboli) of the Doppler signals was performed online by a trained scientist. Doppler was scored according to the Kisman-Masurel protocol (7), with a score scale from 0 (no VGE) to IV (massive embolism). When VGE were detected, another trained observer, blinded to the initial results, also evaluated the score.

TABLE I. REPEATED MEASURES ANOVA WITHIN SUBJECTS EFFECT AT DIFFERENT ALTITUDES FOR CHAMBER FLIGHT PROFILE I TO 25,000 FT.

		F(4,44)	p
Path length	(EO)	0.876	0.486
Path length	(EC)	1.649	0.179
RI	(EC/EO)	0.886	0.480
Lateral speed	(EO)	1.403	0.249
Lateral speed	(EC)	1.162	0.341
RI	(EC/EO)	0.693	0.601
AntPost speed	(EO)	1.843	0.505
AntPost speed	(EC)	1.440	0.237
RI	(EC/EO)	0.498	0.737

EO: Eyes open; EC: Eyes closed; RI: Romberg index.

RESULTS

For chamber flight profile 1, the results from repeated measures ANOVA are given in **Table I**. No significant changes were observed for the path length or for the lateral or anteroposterior speed. **Fig. 2** shows the mean COP path length, lateral speed, and anteroposterior speed with corresponding Romberg Indexes at different altitudes for all 12 subjects.

There was no reduction in oxygen saturation during the test periods in this chamber flight profile. The lowest pulse oxymetry reading was S_{aO_2} 96%. Heart rate (HR) ranged from 60–120 bpm in different subjects.

All Doppler measurements were completed without technical problems. No VGE were observed by Doppler measurements during exposure to this chamber flight profile.

No subject reported symptoms of dizziness, and no

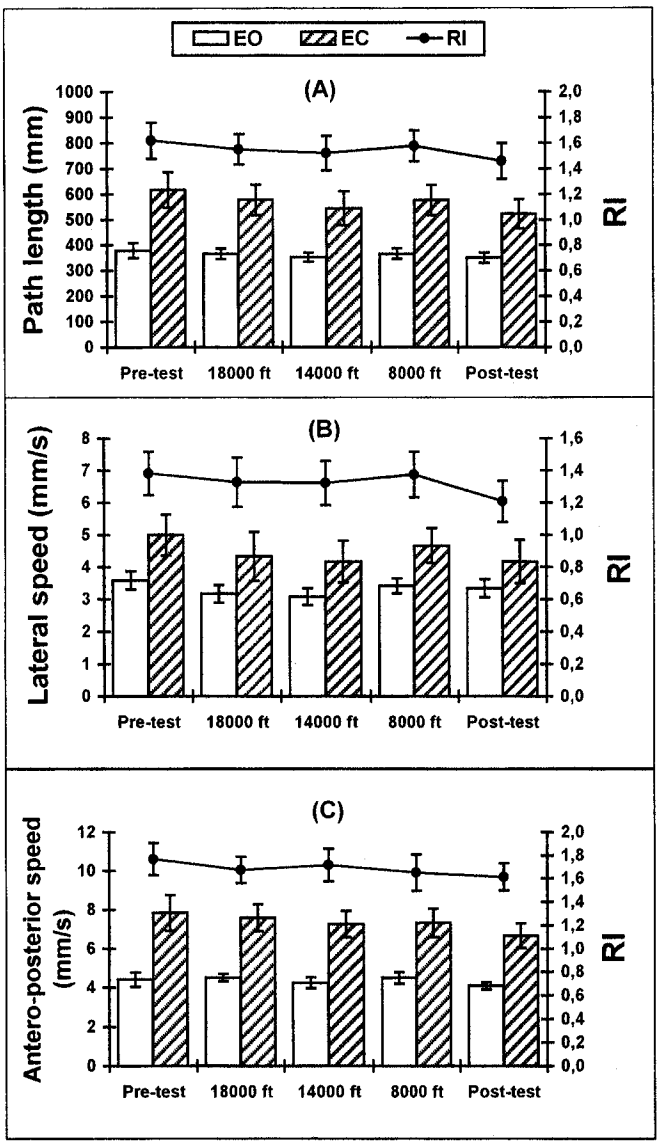

Fig. 2. Mean and standard error (SE) of path length (A), lateral speed (B), and anteroposterior speed (C); and the standard error (SE) for eyes open (EO) and eyes closed (EC) with corresponding Romberg Indexes (RI) at different altitudes for chamber flight profile 1 (n = 12).

unsteadiness was noted by the observer during this exposure.

For chamber flight profile 2, the results from repeated measures ANOVA are given in **Table II**. During the 14,000-ft exposure, change in path length with eyes open (EO) reached statistical significance ($p < 0.05$) according to repeated measures ANOVA. The Tukey HSD comparison revealed no significant differences between the registrations at the various altitudes. However, testing for difference in mean values between all registrations, applying Student's *t*-test, showed that path length for EO were significantly longer at test 3 ($p = 0.01$) and test 2 ($p = 0.04$) compared with test 1 at 14,000 ft. No significant changes were observed in the lateral or anteroposterior speed or for the path length with EO. **Fig. 3** shows the mean COP path length, lateral speed, and anteroposterior speed with corresponding Romberg Indexes for the 11 subjects completing chamber flight profile 2.

During this chamber flight profile, S_{aO_2} decreased in all subjects. Average of the lowest saturation readings in all subjects at 14,000 ft were 72.2% (range 63–80) with corresponding mean HR of 98.1 bpm (range 79–120). When 4 $L \cdot min^{-1}$ of oxygen was provided during the last 20 min at 14,000 ft, oxygen saturation normalized in all subjects, mean 96.4% (range 92–99), with a corresponding decrease in mean HR to 79 bpm (range 76–100). When given supplemental oxygen, most subjects realized that they had experienced mild hypoxic symptoms such as drowsiness, reduced light intensity, and narrowed peripheral vision, while breathing air at 14,000 ft.

VGE were observed in one subject only during this chamber flight profile. This subject, a 62-yr-old female, presented precordial VGE grade I-III and subclavian VGE grade I after 75 min at 14,000 ft. As reported earlier, she did not present VGE during exposure to 25,000 ft, and did not report any symptoms, nor did she have any objective signs of unsteadiness.

One other subject complained of dizziness, malaise, and showed signs of reduced consciousness during the initial part of chamber flight profile 2 during decompression to 14,000 ft. She immediately recovered when oxygen was given through an oronasal mask, but she was withdrawn from the experiment to avoid vasovagal or hypoxic syncope. She later completed chamber flight profile 1 without any problems. No other subject reported symptoms of dizziness, and no unsteadiness was noted by the observer.

DISCUSSION

Our previous investigation demonstrated that postural control became disturbed primarily in the anteroposterior plane with EO during acute hypobaric hypoxia at 18,000, 14,000, and 8000 ft, but normalized when ambient pressure returned to ground level (16). Whether these changes were caused by hypoxia alone or in combination with other effects of reduced barometric pressure, was uncertain.

In the present study, acute exposure to the same altitude chamber profile as in our previous study was

TABLE II. REPEATED MEASURES ANOVA WITHIN SUBJECTS EFFECT FOR CHAMBER FLIGHT PROFILE II TO 14,000 FT.

		F(5,50)	p
Path length	(EO)	2.767	0.028*
Path length	(EC)	0.957	0.453
RI	(EC/EO)	1.818	0.126
Lateral speed	(EO)	1.028	0.412
Lateral speed	(EC)	0.584	0.712
RI	(EC/EO)	1.419	0.234
AntPost speed	(EO)	1.420	0.233
AntPost speed	(EC)	2.192	0.070
RI	(EC/EO)	1.439	0.227

EO: Eyes open; EC: Eyes closed; RI: Romberg index; *$p < 0.05$.

tested in chamber flight profile 1, but this time our subjects were held normoxic during the test periods. Since there was no detectable deterioration of balance, it is suggested that acute hypoxia is the direct cause of the disturbed postural control observed earlier (16).

The results from repeated measures ANOVA for chamber flight profile 2 (Table II) showed a statistically significant change for the path length for EO only. However, the Tukey HSD comparison revealed no significant differences between the registrations at the various altitudes. Nevertheless, **Fig. 4** (y-axis from 320 mm and upwards) clearly indicates deterioration in the path length for EO, particularly during tests 2 and 3 when breathing air. This was confirmed when applying Student's *t*-test, which gave significant values for test 3 ($p = 0.01$) and test 2 ($p = 0.04$) compared with test 1 at

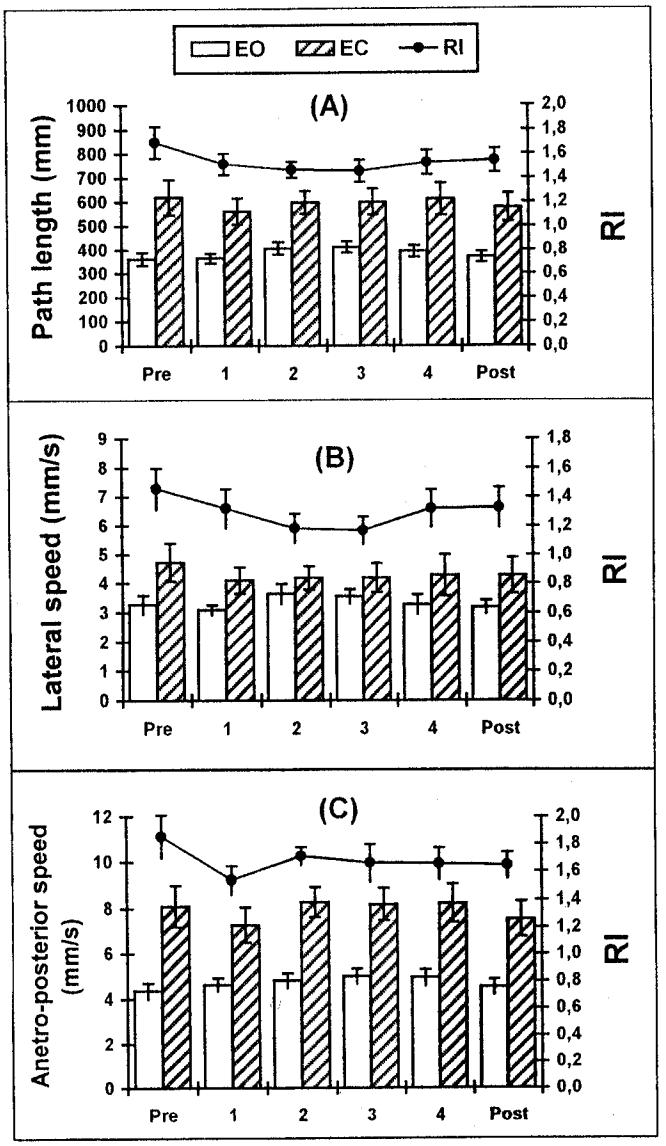

Fig. 3. Mean and standard error (SE) of path length (A), lateral speed (B), and anteroposterior speed (C); and the standard error (SE) for eyes open (EO) and eyes closed (EC) with corresponding Romberg Indexes (RI) for chamber flight profile 2 (n = 11). Pre- and post-exposure, test 1, 2 and 3 without supplemental oxygen, test 4 with supplemental oxygen.

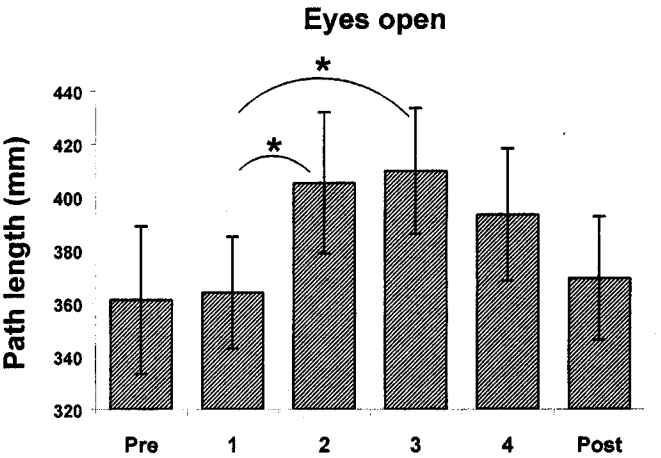

Fig. 4. Path length for eyes open (EO) for chamber flight profile 2—up to 14,000 ft (n = 11) Pre- and post-exposure, test 1, 2 and 3 without supplemental oxygen, test 4 with supplemental oxygen (* $p < 0.05$)

14,000 ft. After supplemental oxygen was started before test 4, an apparent normalization occurred. This is in agreement with our first study of acute hypobaric hypoxia at 18,000, 14,000, and 8000 ft (16). This suggests that the lack of oxygen is the direct cause of a disturbed postural control and is in agreement with other studies showing that vision is the first of the special senses to be altered by lack of oxygen (5), causing disturbance of balance.

Doppler monitoring was included to rule out VGE as a possible cause of disturbed postural control. A secondary objective was to investigate whether two commonly used altitude chamber training profiles would release VGE.

Precordial and peripheral vein continuous Doppler measurement is the traditional method for quantifying VGE in humans (13). More sophisticated techniques using two-dimensional cardiac ultrasound have been developed (4), but their use is restricted due to equipment cost and lack of tolerance to rapid pressure changes. Finer grading of VGE on the basis of their acoustic signature is associated with significant inter-observer differences (17). However, high inter-observer concordance is the rule when no VGE are present. The initial rating was performed by a scientist with 4 yr of Doppler scoring experience; to verify the conclusion of the one subject presenting VGE, her data (tape) were scored independently by another trained observer with the same result. We thus consider the Doppler data to be valid.

VGE were observed in one subject, during one exposure (14,000 ft) only. VGE after decompression to 15,000 ft have been observed previously (23,24), but to the best of our knowledge, no other published study has reported VGE in air breathing humans after decompression to less than 15,000 ft. Our data showed consistent, though short lasting, VGE in peripheral veins and mixed cardiac blood after 75 min at 14,000 ft, but the bubbles disappeared shortly after reaching ground level.

There was no apparent reason why this subject should demonstrate VGE. She had no recent hypo- or

hyperbaric exposures, used no medications, and was slim. However, at age 62 she was our oldest subject and she was also a smoker. Many reports from hyperbaric decompressions demonstrate a very high intra- and inter-individual variability in VGE production (13). Possible clinical effects of any VGE occurring during such modest hypobaric exposures are probably minor. Acute and long-term effects of VGE include decompression sickness (DCS) and decrement of pulmonary function (18,19). These effects are associated with bubble grades III or higher, or high accumulated bubble scores. It should be recognized that the power of this study is low with respect to VGE detection (the 95% confidence interval for the fraction of subjects experiencing VGE during the 14,000 ft altitude exposure is 0–0.3). To obtain more data, we recommend that Doppler monitoring be performed during hypobaric exposure to altitudes exceeding 14,000 ft.

The study was not designed to evaluate the occurrence of VGE in the simulated altitude exposures to 25,000 and 14,000 ft. Even if no subject with VGE had been observed in a group of 12, the 95% confidence interval would be 0–31% with respect to the estimated fraction of subjects actually presenting VGE in that population. Although the power of the test is low with respect to estimating the presence of VGE in the population at large, the practical and clinical consequences of any grade III and grade IV measurements may be important. Grade III and IV microemboli are associated with acute DCS (18), and although no firm accept or reject criteria has been established for Doppler score in decompression experiments (13), we would recommend a target of zero bubbles, and call for changes, compensatory measures, and alertness if grade III or IV observations were repeated.

The described Doppler technique will only detect circulating bubbles. There is currently no technique commercially available for in vivo measurements of in situ bubbles. Accordingly, possible in situ extravascular bubbles formed as a consequence of pressure reduction would pass undetected. The amount of VGE is considered an indirect measurement of the total free gas phase. However, unaffected postural control during hypobaric exposure in this study indicates that no bubbles were formed in or transported to the vestibular system.

CONCLUSIONS

Our study showed that acute hypobaric normoxia at 18,000, 14,000, and 8000 ft caused no disturbance of postural control. In accordance with our earlier hypoxia study, prolonged hypoxia at 14,000 ft did cause a deterioration of postural control (16).

No VGE were found in altitude chamber profile 1 to 25,000 ft, indicating that this chamber flight profile, with a 45-min pre-oxygenation period, is a safe procedure and prevents bubble formation.

This study unexpectedly demonstrated that VGE may form during hypobaric hypoxia as low as 14,000 ft without pre-oxygenation. This calls for some caution and Doppler monitoring in future hypobaric experiments or other activities (e.g., hypobaric exercise conditioning) to similar altitudes.

However, there was no correlation between observed VGE and detectable change in postural control. This suggests that lack of oxygen was the direct cause of disturbed postural control at the tested altitudes, and that reduced barometric pressure causing VGE probably was not causing instability.

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