VENOUS GAS EMBOLI AND AMBULATION AT 4.3 PSIA (PRELIMINARY)

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INTRODUCTION: Ambulation imparts compressive and decompressive forces into the lower body[1], potentially creating quasi-stable micronuclei that influence the outcome of hypobaric depressurizations[2-4]. *Hypotheses*: ambulation before the conclusion of a denitrogenation (prebreathe) protocol at 14.7 psia is not sufficient to increase the incidence of venous gas emboli (VGE) at 4.3 psia but is sufficient if performed after tissues become supersaturated with nitrogen at 4.3 psia. **METHODS:** VGE results from 45 subjects that performed exercise prebreathe without ambulation before or during a 4-hr exposure to 4.3 psia (Phase II control) are compared to 21 subjects that performed the same prebreathe but ambulated before and during the hypobaric exposure (Phase II-1) and to 30 subjects that only ambulated before the hypobaric exposure (Phase II-3). VGE in the pulmonary artery were detected at about 16 min intervals using precordial Doppler ultrasound (2.5 mHz) and assigned a Spencer 0 – IV grade. The highest grade assigned during the exposure is one metric, and Grade III or IV were combined as "high VGE grade". We used Fisher's exact directional χ^2 for VGE count data. We evaluated survival curves for onset of high VGE grade using non-parametric Kaplan-Meier. Finally, we used logistic regression (LR) to describe the binary response of high VGE grade during an exposure across the three protocols with the three categorical ambulatory states (k) transformed into k-1 dummy variables, plus the following explanatory variables: gender (74 men and 22 women), age (19-57 years), height (155-191 cm), weight (44-115 kg), body mass index (17.5-35.5, as weight in kg / height as m^2), and aerobic fitness through peak oxygen (O_2) consumption as $\dot{V}O_2$ peak (26.1-64.9 ml $O_{2(STPD)} \times kg^{-1} \times min^{-1}$). **RESULTS:** The incidence of any VGE grade was greater in Phase II-1 (62%) compared to Phase II (31%, P = 0.01) and Phase II-3 (30%, P = 0.01). The incidence of high VGE grade for Phase II-1 (57%) was also greater than Phase II (17%, P = 0.001) and Phase II-3 (16%, P = 0.002). Mean onset times for high VGE grade were similar for control (105 $\pm$ 72 min SD), II-1 (104 $\pm$ 55), and II-3 (102 $\pm$ 24) conditions. Differences among the three Kaplan-Meier survival curves were significant from log-rank tests ($P \leq$ 0.001), but not just between II and II-3 ($P >$ 0.85). Only age and ambulation status from LR were needed to describe the probability of high VGE grade. **DISCUSSION:** VGE are increased by mild ambulation conducted under a supersaturated state (Phase II-1 vs II-3); however, no increase in VGE was observed with mild ambulation during the undersaturated state alone (Phase II control vs II-3).

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