

Short-arm human centrifugation with 0.4g at eye and 0.75g at heart level provides similar cerebrovascular and cardiovascular responses to standing

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Abstract

Background and purpose Orthostatic intolerance continues to be a problem with astronauts upon return to Earth as a result of cerebral and cardiovascular adaptations to weightlessness. We tested the hypothesis that artificial gravity from a short-arm human centrifuge (SAHC) could provide cerebral and cardiovascular stimuli similar to upright posture and thereby serve as a suitable countermeasure.

Methods We compared cardiovascular and cerebrovascular responses before, during, and after exposure to hyper-G with that of standing in healthy young participants. The head was positioned such that the middle cerebral artery (MCA) was 0.46 m from the center of rotation. Two levels of hyper-G that provided 1g and 2g at foot level were investigated. Continuous blood pressure, heart rate, calf blood volume, MCA mean blood flow velocity (MFV) and end-tidal CO₂ were measured.

Results Blood pressure at the level of the MCA (BP-MCA) and MFV was reduced during stand and at 2g. The relationship between MFV and BP-MCA at 2g was different from supine and similar to standing, while 1g

centrifugation was not different from supine. The cardiovascular system was also not different from supine at 1g but was similarly challenged in 2g compared to stand.

Conclusions Our data suggest that short-arm centrifugation 2g at the feet, with the head offset 0.5 m from the center, provides similar cardiovascular and cerebral responses to standing. This supports the hypothesis that passive 2g SAHC exposure at the feet could be used as a countermeasure for in-flight cardiovascular and cerebrovascular deconditioning.

Keywords Orthostasis · Baroreflex · Cerebral autoregulation · Space flight · Short-arm human centrifuge · Stand test

Introduction

Weightlessness instantaneously removes physical processes that depend on weight for their effect, and introduces physiological issues including impaired cardiovascular control functions and cerebral autoregulation (Blaber et al. 2013a, b; Goswami et al. 2013), as well as reduced musculoskeletal and sensorimotor system performance.

The cephalad-fluid shift and postural muscle unloading during microgravity are considered to be major contributing factors of the spaceflight deconditioning. Recently, ocular syndrome (a.k.a. visual impairment and intracranial pressure—VIIP) (Mader et al. 2011; Zhang and Hargens 2014) has been recognized as a possible serious hazard associated with long-term exposure to weightlessness; it led to loss of visual acuity in orbit as well as possible permanent visual impairment post-flight. Space-induced ocular syndrome is defined by visual and cerebral changes associated with exposure to microgravity. Astronauts have

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been shown to suffer from ocular changes such as visual edema, posterior globe flattening, choroidal folds, nerve fiber layer thickening, decreased near vision, and hyperopic shifts (Mader et al. 2011; Zhang and Hargens 2014). It has also been observed that some of these problems remain unresolved a year after space mission and that 60 % of long-duration mission astronauts experience degradation in distant and near visual acuity (Zhang and Hargens 2014; Fogarty et al. 2011). This leads to serious concerns for future long-duration missions unless a possible treatment/countermeasure is found.

Given the serious physiological consequences of weightlessness, the ability to mimic upright stimuli to the cardiovascular as well as cerebrovascular systems in space has important operational and clinical significance. It has been proposed that centrifuge-induced artificial gravity (AG), in particular short-arm human centrifuges (SAHC) due to current spacecraft designs, may provide an effective multi-system countermeasure against the spaceflight deconditioning. Ground-based studies have indicated that AG may improve orthostatic tolerance in ambulatory men (Iwase et al. 2002; Evans et al. 2004) and maintain orthostatic tolerance in bed rested men (Stenger et al. 2012). Despite the advantage of the compact nature of a SAHC, the orthostatic challenge on the human body is different from that of a normal gravitational field; G increases as a square of the distance from center. For example, on a SAHC in the average supine human, $1g$ acceleration at the heart would result in around $2.4g$ at the feet. It is therefore important to investigate the human physiological responses to this unusual G environment compared to standing in $1g$.

To date, there has not been a comprehensive study of the effects of SAHC on the cardiovascular and cerebrovascular systems and its relation to standing orthostatic reflexes. These data are critical since, unlike standing, the acceleration forces within a SAHC increase from head to toe as mentioned above. We have previously shown that the cerebral vasculature is affected by spaceflight (Blaber et al. 2011; Zuj et al. 2012). Effective application of AG should produce cardiovascular and cerebrovascular responses similar to standing. To this end, we compared SAHC with standing to determine if it could be used to simulate the physiological effects of upright posture while in space.

Methodology

Participants

All subjects were healthy, un-trained, non-obese, nonsmoking volunteers without history of vasovagal syncope. All subjects underwent a pre-screening assessment for medical and physical status. All subjects were free from any chronic

disease or any acute infectious disease or ENT, neurological (especially orthostatic hypotension and vestibular disorders), orthopedic, musculoskeletal and cardiovascular disorders. They had no alcohol or drug dependence and were not on medical treatment. Female subjects were excluded if they were pregnant, less than 3 months postpartum or were breastfeeding or less than 2 months post-breastfeeding prior to the commencement of the study. Once the subjects were cleared by the medical examiner, they underwent an orientation session in the short-arm human centrifuge with $1g$ at the foot for 10 min. If no cardiovascular or vestibular problems were noted the subjects were entered into the study.

Ten males and ten females participated in this study. Of these, five were excluded due to intermittent loss of the blood pressure signal, mostly during standing, blood pressure data from two subjects had excessive PhysioCal events where the finger cuff device performed automated calibration, two were excluded due to unacceptable cerebral blood flow velocity measurements, and one had no data recorded during post-centrifuge stand. Therefore, only ten subjects (six male—age: 29.1 ± 4.3 years, weight: 76.4 ± 4.6 kg; height: 1.77 ± 0.07 m and four female—age: 25.8 ± 3.1 years; weight: 59.2 ± 6.8 kg; height: 1.69 ± 0.04 m) had complete physiological data that could be used for comprehensive analysis.

Ethics approval was obtained from the University of Toulouse. Each participant gave written informed consent. The consent forms were then stored at MEDES, Toulouse where this study took place.

Test protocol

All tests were carried out in a semi-dark and quiet room ($22\text{--}23$ °C, $50\text{--}55$ % humidity). On the test day each subject had abstained from caffeine, alcohol, and any medications for the preceding 12 h; was at least 2 h postprandial; and had not exercised heavily in 24 h.

A safety harness was placed on the subject and they were strapped into the centrifuge with the subject's head near the center of the 2.8-m-radius centrifuge. The subject was instrumented for cardiovascular and cerebrovascular monitoring. All signals were verified for quality and integrity and safety procedures were reviewed with the subject before the upper part of their body was covered with a black lightproof hood to remove visual cues during rotation.

The subject remained in the supine position on the centrifuge for 20 min for baseline data. At the end of the 20 min, the subject was quickly raised to the upright position and assisted to a standing position at the side of the centrifuge. The transition from supine to stand was 1 min. The stand lasted for 5 min before the subject was returned

to the centrifuge in the supine position. After 15 min of supine rest, the centrifuge was activated and ramped up to a rate that applied the equivalent of 1g at the feet (0.22g at the middle cerebral artery, MCA, and 0.39g at the heart) for 5 min and increased to 2g at the feet (0.44g at the MCA, and 0.75g at the heart) for a remaining 5 min. Following the 10 min of centrifugation, rotation of the centrifuge slowed to a stop over 30 s. Once rotation had ceased the subject was again assisted to a 5-min stand after which they were returned to the centrifuge for 20 min supine recovery followed by a final 5-min stand test.

Data collection

Blood pressure (BP) was measured using the non-invasive photoplethysmography (Portapres: FMS, the Netherlands). To ensure that the appropriate measurements of heart level BP were obtained while supine, upright or during centrifugation, the finger with the continuous arterial pressure device was held at heart level by a system of Velcro straps and the BP measurements were corrected for mid-axillary level. Cardiac stroke volume (SV) was derived from the BP waveform measurements using the designated software of Portapres. Total peripheral resistance (TPR) was calculated using this derived SV along with mean arterial blood pressure (MAP) and heart rate (HR). For cerebral autoregulation analysis, BP at the level of the MCA (BP-MCA) was calculated as a linear hydrostatic gradient from heart level for standing and as a squared pressure differential based on the distance of the MCA and the finger cuff from the center of the centrifuge. Each participant was also instrumented for an electrocardiogram.

Selective deep calf Hb changes were measured via near infrared spectroscopy (NIRS) (Omegawave BOM-L1W); the electrodes for the NIRS device were placed on the thigh region. We have previously validated the NIRS method for the measurement of vascular responses in the calf during real orthostatic challenge (Hachiya et al. 2004, 2008) and simulated orthostatic challenge, using lower body negative pressure (Blaber et al. 2013a).

Mean blood flow velocity (MFV) of the MCA was measured through the right temporal window with 2-MHz pulsed Transcranial Doppler ultrasound (Multiflow: DWL, Germany). Signal processing and determination of MFV, cerebrovascular resistance index (CVRI), defined as BP-MCA/MFV, and dynamic cerebral autoregulation were performed as previously described in our paper on cerebral blood flow regulation in astronauts (Blaber et al. 2011). As carbon dioxide concentrations can alter cerebral autoregulation, carbon dioxide and respiratory frequency was assessed using a facemask and capnograph (CO₂ SMO 7100, Novametric).

Treatment of data

The analog signals from each of the devices were recorded simultaneously at 1 kHz per channel using a 16-channel digital tape recorder (National Instruments, Ireland). Beat-by-beat analysis of these data was performed offline. Autospectra of BP-MCA and CVRI were calculated using power spectral analysis to quantify low frequency (0.07–0.2 Hz) variability of the signals. The dynamic autoregulatory gain and phase were determined in the low frequency range using the transfer function method (BP-MCA → CVRI), which provided the responsiveness and phase lag of the cerebral autoregulation system. The application of power spectral analysis and transfer function method to the dynamic autoregulation analysis was previously described in detail (Blaber et al. 2011). Spontaneous baroreflex slope was determined by the beat sequence method, which has been shown to be highly correlated with the baroreflex response slope obtained during administration of drugs to raise or lower BP (Parlow et al. 1995). With statistical analysis of these data, the upper limit for acceptable type I errors was set at 5 %. Variations were indicated as mean ± SE. Changes in stand tests were tested using a two-factor (men/women, pre/post-centrifuge stand tests) ANOVA with repeated measures on one factor (pre/post stand tests). Tukey HSD post hoc test was used for multiple comparisons. Significance was accepted at $p < 0.05$.

Results

Due to the small sample size for gender comparison (six male and four female) and concern about statistical power, gender difference and related results are not presented.

Cerebrovascular

BP-MCA and MFV of the MCA were reduced during stand and at 2g. Although there was an apparent trend to reduced cerebrovascular resistance with stand, only at 2g was it significantly lower than supine baseline (Table 1; Fig. 1). End-tidal CO₂ was reduced in the pre-stand and post-recovery stand test (Table 1). Analysis of the relationship between MFV and BP-MCA revealed a different relationship with standing and 2g compared to supine and 1g (Fig. 2).

Autospectra of BP-MCA and CVRI revealed an increase in low frequency variability in stand and 2g for both the blood pressure and cerebrovascular resistance (Table 1). Transfer function gain (BP-MCA → CVRI) was significantly reduced during 2g with no change in phase angle ($-65^\circ \pm 6^\circ$) over all conditions.

Table 1 Cerebrovascular responses to short-arm centrifugation and stand

	BL	Stand 1	Pre-supine	1g	2g	Stand 2	Post-supine	Stand 3
BP-MCA (mmHg)	88.0 (4.4)	65.1 ^a (4.4)	93.8 (4.4)	80.8 (4.4)	64.7 ^a (4.4)	72.4 ^a (4.4)	83.5 (4.4)	61.6 ^a (4.4)
MFV (cm/s)	63.6 (3.1)	52.6 ^a (3.1)	66.0 (3.1)	63.0 (3.1)	56.3 ^a (3.1)	53.7 ^a (3.1)	64.2 (3.1)	53.6 ^a (3.1)
CVRI (mmHg s/cm)	1.40 (0.06)	1.30 (0.06)	1.42 (0.06)	1.29 (0.06)	1.15 ^a (0.06)	1.37 (0.06)	1.33 (0.06)	1.17 (0.06)
PETCO ₂ (mmHg)	35.9 (0.7)	31.9 ^a (0.7)	37.9 (0.7)	38.3 (0.7)	35.9 (0.7)	33.0 (0.7)	36.2 (0.7)	31.6 ^a (0.7)
LF-BP-MCA-power (mmHg) ²	2.93 (1.52)	10.61 ^a (1.52)	2.81 (1.52)	5.73 (1.52)	16.92 ^a (1.52)	9.96 ^a (1.52)	3.75 (1.52)	13.37 ^a (1.52)
LF-CVRI-power (10 ⁻³) (mmHg s/cm) ²	1.3 (0.4)	3.3 ^a (0.4)	1.2 (0.4)	1.5 (0.4)	3.6 ^a (0.4)	2.7 (0.4)	1.8 (0.4)	3.3 ^a (0.4)
LF gain (BP-MCA → CVRI) (10 ⁻³) (s/cm)	17.9 (1.4)	15.5 (1.4)	17.6 (1.4)	14.0 (1.4)	12.8 ^a (1.4)	15.1 (1.4)	18.2 (1.4)	14.1 (1.4)

Values presented: mean (SE)

BL: baseline; stand 1; pre-supine: supine before centrifugation; 1g, 2g: G at the feet, stand 2; post-supine: 20-min post-centrifugation recovery; stand 3

^a Different from baseline

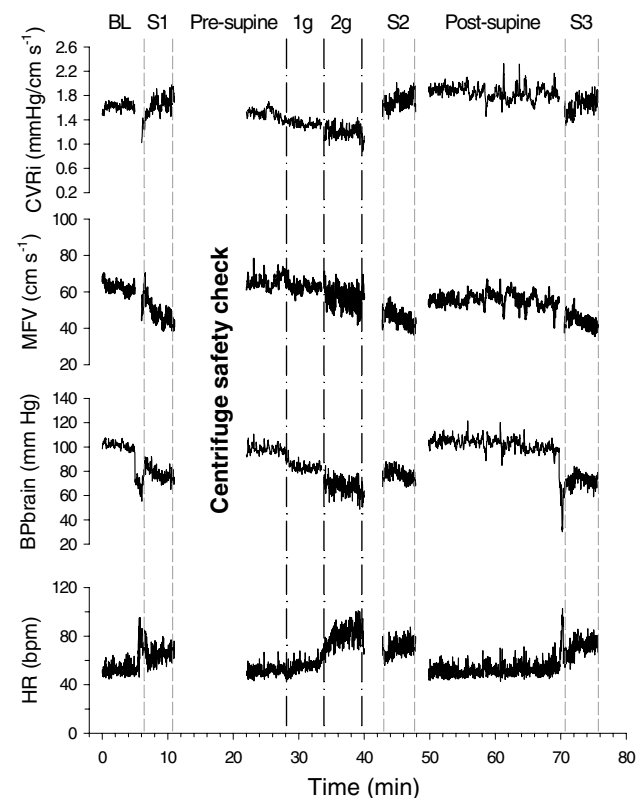


Fig. 1 Example of cerebrovascular responses of one subject during the centrifuge protocol [MFV: mean blood flow velocity in the middle cerebral artery as measured by transcranial Doppler ultrasound; BP-MCA: mean arterial blood pressure measured via the Portapres™ finger cuff and adjusted for the hydrostatic gradient between the heart and the MCA during stand and centrifugation; CVRI: cerebrovascular resistance index calculated as (BP-MCA/MFV); HR: heart rate]. Protocol: 20-min supine followed by (a) 5-min supine baseline data (BL); (b) assisted 5-min stand (S1); (c) 15-min supine rest; 5-min supine data (pre-supine) data; (d) 5 min 0.22g at MCA (1g); (e) 5 min 0.44g at MCA (2g); (f) 5-min stand test (S2); (g) 20-min supine (post-supine); and, (h) 5-min stand test (S3)

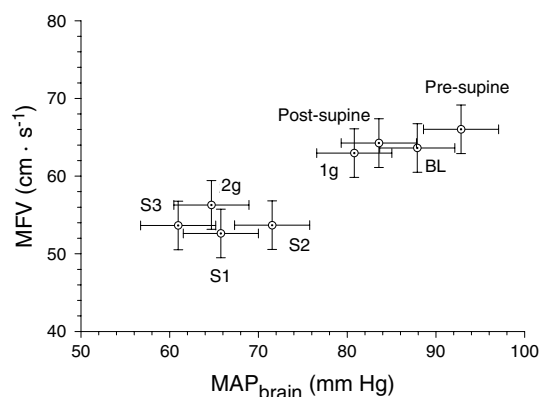


Fig. 2 Mean (\pm SE) for MFV and BP-MCA in the last minute of each condition. Protocol: 20-min supine followed by (a) 5-min supine baseline data (BL); (b) assisted 5-min stand (S1); (c) 15-min supine rest; 5-min supine data (pre-supine) data; (d) 5 min 0.22g at MCA (1g); (e) 5 min 0.44g at MCA (2g); (f) 5-min stand test (S2); (g) 20-min supine (post-supine); and, (h) 5-min stand test (S3)

Cardiovascular

The cardiovascular system was challenged during both centrifugation and stand. Heart rate increased with orthostatic challenge with the highest value at 2g (Table 2). Systolic blood pressure (SBP) was increased in the post-centrifuge stand, but not during centrifugation or the other two standing tests. Both SBP and diastolic blood pressure (DBP) in the post-centrifuge stand were elevated compared with 2g centrifugation. Mean arterial blood pressure (MAP) was maintained with the only significant increases in post-centrifuge stand and post-recovery stand compared with 2g (Table 2). SV was reduced from baseline at all levels of orthostatic challenge except for 1g (Table 2).

Table 2 Cardiovascular responses to short-arm centrifugation and stand

	BL	Stand 1	Pre-supine	1g	2g	Stand 2	Post-supine	Stand 3
HR (bpm)	63 ^b (3)	81 ^{a,b} (3)	64 ^b (3)	69 ^b (3)	92 ^a (3)	81 ^{a,b} (3)	58 ^b (3)	85 ^a (3)
SBP (mmHg)	133 (7)	139 (7)	143 (7)	139 (7)	131 (7)	150 ^{a,b} (7)	129 (7)	130 (7)
DBP (mmHg)	70 (4)	74 (4)	73 (4)	70 (4)	69 (4)	80 ^b (4)	66 (4)	70 (4)
MAP (mmHg)	89 (4)	84 (4)	90 (4)	89 (4)	82 (4)	97 ^b (4)	84 (4)	93 ^b (4)
Baroreflex slope (ms/mmHg)	16.0 (2.5)	8.5 (2.5)	17.1 (2.5)	13.3 (2.5)	5.2 ^a (2.5)	7.9 ^a (2.5)	22.2 (2.5)	6.8 ^a (2.5)
SV (mL)	109 ^b (6)	88 ^a (6)	110 ^b (6)	98 ^b (6)	82 ^a (6)	88 ^a (6)	110 ^b (6)	87 ^a (6)
TPR (mmHg min/L)	13.6 (0.9)	13.8 (0.9)	13.5 (0.9)	13.4 (0.9)	11.5 (0.9)	14.5 ^b (0.9)	13.5 (0.9)	12.4 (0.9)
DeOxy-Hb (AU)	0 (1.5)	13.57 ^{a,b} (1.5)	1.43 ^b (1.5)	10.95 ^{a,b} (1.5)	19.43 ^a (1.5)	14.26 ^{a,b} (1.5)	1.62 ^b (1.5)	13.38 ^{a,b} (1.5)
Oxy-Hb (AU)	0 (1.78)	2.97 (1.78)	0.88 (1.78)	2.77 (1.78)	1.63 (1.78)	3.04 (1.78)	1.24 (1.78)	4.56 (1.78)

Values presented: mean (SE)

BL: baseline; stand 1; pre-supine: supine before centrifugation; 1g, 2g: G at the feet, stand 2; post-supine: 20-min post-centrifugation recovery; stand 3

^a Different from baseline; ^b Different from 2g

The change in derived TPR indicated that TPR was increased upon standing immediately after centrifugation compared with 2g. The estimate of venous volume in the calf (DeOxy-Hb) showed an increase in the calf at all orthostatic conditions with the largest at 2g.

The spontaneous baroreflex slope was decreased almost threefold from baseline to 2g. Pre-centrifugation stand and 1g centrifugation were not significantly different from baseline, whereas both post-centrifugation stands were different from baseline while not different from 2g (Table 2).

Discussion

In this study, we examined the effect of SAHC artificial gravity on cerebral blood flow velocity regulation in the middle cerebral artery. In relation to the center of rotation, the MCA was at a radius of 0.46 m, the heart ~0.80 m, and the feet ~2.00 m depending on subject height. The subjects experienced two rotation rates inducing a G-loading at the level of the MCA of 0.22g and 0.44g and at foot level of 1g and 2g. We compared these results with supine and 5 min of standing. Our results showed that cerebral autoregulation was operating normally in both centrifuge conditions and that the 2g foot rotation (0.44g at the MCA) most closely resembled standing.

With the MCA located 0.46 cm from the center of rotation, a 1g loading at the feet produced an average 0.22g at the MCA. This level of G-force and the accompanying blood volume fluid shifts along the body axis were not sufficient to produce changes in MCA MFV, vascular resistance or associated autoregulatory spectral power (Table 1). However, with 2g at the feet and 0.44g at the MCA, the vascular responses within the MCA more closely resembled that of standing with similar decreases in BP-MCA

and MCA MFV and increases in both BP-MCA and CVRi autoregulatory spectral power (Table 1).

Given that, the data indicate an apparent difference in set point for cerebral responses between the supine/1g region and the stand/2g region (Fig. 2), it would seem at first glance that cerebral autoregulation at 2g is analogous to standing. While the 2g condition produced cerebrovascular responses similar to standing, there were some physiological differences between SAHC and standing. Although MFV and BP-MCA at 2g were not different from that of standing, at 2g, CVRi and cerebral autoregulation gain were significantly reduced from baseline unlike standing (Table 1; Fig. 2). This would suggest a difference in cerebrovascular control between stand and 2g.

The reduction in PETCO₂ from supine to standing has been well documented and is hypothesized to be related to postural changes in blood perfusion and air movement within the lungs. PETCO₂ was not significantly reduced at 2g, but was during the pre-centrifuge and post-recovery stand tests (Table 1). Given the potent effect of CO₂ on cerebral vasculature, the observed change in PETCO₂ of 4.0 mmHg with standing could account for the downward shift of the autoregulation curve during standing. Since PETCO₂ was not changed at 2g, it is possible that centrifugation at this level was not sufficient to change these ventilation perfusion dynamics.

With no noticeable change in PETCO₂ at 2g, the reduction in MFV during centrifugation could not be CO₂ related and may more likely be myogenic in nature; however, the drop in driving pressure to the MCA was the same as standing (Table 2; Fig. 1). Evidence of a strong myogenic response to the drop in BP-MCA is seen in the significant reduction of vascular resistance (CVRi) from baseline. It is most likely that the changes at 2g represent the effect of decreased blood pressure on cerebral vasculature, whereas

standing MFV was the result of the combined effect of CO_2 and decreased blood pressure. From this interpretation, we hypothesize that the effect of CO_2 on the overall autoregulatory response was minimal given that CVRi at 2g was not significantly different from standing although it was from baseline; 2g was at the end of a spectrum of autoregulation responses from baseline to stand/centrifugation. This is substantiated by 2g having the largest variations in BP-MCA and CVRi spectral power and the lowest vascular resistance and autoregulation gain; all of which were significantly different from baseline values but not from standing.

The cephalad-fluid shift observed in microgravity, along with the ICP/IOP mismatches during acclimation, is considered to be the main factors in cerebrovascular deconditioning and the development of space flight ocular syndrome (Mader et al. 2011; Zhang and Hargens 2014). Although we did not measure intracranial or ocular pressures, our data suggest that the SAHC could produce cerebrovascular responses similar to standing. If used during weightlessness, an SAHC with 2g at the feet should provide a sufficient decrease in cerebral perfusion pressure and increase in caudal blood flow gradient to reduce ICP and hence mitigate development of ocular syndrome as well as reducing the incidence of impairment of cerebral autoregulation observed in a segment of the astronaut population prone to post-flight orthostatic intolerance (Blaber et al. 2011; Zuj et al. 2012).

Cardiovascular effects

Our data also provide clear evidence that the use of an SAHC with 2g at the feet provides similar cardiovascular reflexes to standing, whereas 1g at the feet was not different from supine. Standing and 2g cardiac stroke volume and baroreflex were not different but were different from supine (Table 2). There were some subtle differences in cardiovascular responses at 2g compared to standing which may suggest that 2g at the feet may have been moderately more stressful: deOxy-HB at 2g was significantly higher than all standing conditions; heart rate was significantly elevated at 2g compared to pre- and post-centrifuge stand; TPR, SBP, and DBP at 2g were significantly lower than those of post-centrifuge stand (Table 2). The fact that MAP was not different between 2g and the pre-SAHC stand test (Table 2) and that none of the subjects were presyncopal suggests that this level of centrifugation could be passively tolerated.

These data suggest that centrifugation at 2g in space could be a suitable countermeasure for the effects of weightlessness on the cardiovascular system. Spaceflight-induced fluid redistribution causes reduced cardiac function and altered heart rate responses, a significant

reduction in total blood volume, and possible changes in leg, splanchnic or central vein compliance with blood pooling. Each of these may contribute to the exacerbation of the reduction in venous return that occurs on standing (Watenpaugh and Hargens 1996). There is also increasing evidence that many clinical effects of microgravity and most changes affecting the cardiovascular system after return from space are induced by alterations of the autonomic neural control (Mano 2005), which includes the baroreflex. While unchanged in-flight baroreflex was reported during long-term missions on the ISS (Hughson et al. 2012; Verheyden et al. 2010), reduction of baroreflex response has been observed after both short-term (Fritsch-Yelle et al. 1994; Gisolf et al. 2005; Verheyden et al. 2007) and long-term (Hughson et al. 2012) spaceflight and it has been suggested that the post-flight reductions in baroreflex response could be attributed to reductions in resting parasympathetic activity (Hughson et al. 2012; Verheyden et al. 2010).

While these data indicate that SAHC at 2g may provide sufficient stimulation to the cardiovascular system to simulate the effects of standing, there was also evidence to suggest that centrifugation altered the response to standing. Vascular resistance was significantly elevated immediately after centrifugation and although MAP, SBP, and DBP during 2g were similar to supine and pre-SAHC stand, post-SAHC stand values were elevated compared to SAHC at 2g. These data indicate that centrifugation induced elevated sympathetic activation that persisted. This may indicate that SAHC prior to stand may provide some protection against standing orthostatic hypotension as indicated in previous studies (Iwase et al. 2002; Evans et al. 2004; Stenger et al. 2012).

Study limitations

Although gender differences were observed (e.g., larger change of heart rate with orthostatic challenge and lower SV in female), the results were not presented in this study due to small sample size (six male and four female). Further investigations on gender comparison with larger number of subjects are warranted.

The test protocol performed the two SAHC interventions of different G-loading in sequence on the same day, which may induce accumulated effects of 1g centrifugation on the following 2g test. The results of this study indicated no significant cardiovascular or cerebrovascular differences between 1g centrifugation and supine and, therefore, it is unlikely that the 1g intervention had an appreciable effect on the response to 2g. However, protocols with randomized sequence of different G-loading tests between the subjects can be adopted in future studies to reveal interactions between different G-loading conditions.

Conclusions

Our data supports the hypothesis that SAHC could be used as a countermeasure for in-flight cardiovascular and cerebrovascular deconditioning and that passive exposure to 2g at the feet may be an effective level for protection.

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