Multimodal Signal Analysis of Simulated Microgravity on Cerebral-Systemic Hemodynamics and Cardiac Autonomic Regulation

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***Abstract*—Microgravity during spaceflight induces significant physiological changes due to headward fluid redistribution in the absence of gravitational forces, altering cerebral and cardiovascular hemodynamics and impacting astronaut health and performance. While prior studies have examined cardiovascular responses using ground-based microgravity analogs, the effects of simulated short-term microgravity remain relatively underexplored, particularly from an integrated cardiovascular-cerebral perspective. Therefore, this study investigates the effect of short-term 6° head-down tilt(HDT) on cerebral and systemic hemodynamics and cardiac autonomic function. This quasi pre–post experimental study included 19 healthy participants who underwent a 30-min HDT intervention, with pre- and post-measurements of cerebral hemodynamics using functional near-infrared spectroscopy, along with blood pressure and heart rate variability. Short-term HDT significantly increased prefrontal cerebral oxygenation, reflected by both HbO ($p = 0.0193$) and $Hbdiff$ ($p = 0.0288$). Systemic hemodynamics showed significant reductions in SBP ($p = 0.0001$) and MAP ($p = 0.0015$). Peripheral pulse dynamics revealed a significant decrease in $MeanHR$ ($p = 0.0098$) along with significant increases in $MeanIBI$ ($p = 0.0259$) and $SDNN$ ($p = 0.0236$). These findings indicate that simulated microgravity induces concurrent cerebral and cardiovascular adaptations, characterized by increased cerebral oxygenation and modulation of systemic hemodynamics and cardiac variability.**

***Index Terms*—Microgravity, Head-Down Tilt (HDT), Hemodynamics, Functional Near-Infrared Spectroscopy (fNIRS), Heart Rate Variability (HRV), Cardiac Autonomic Regulation, Oxyhemoglobin (HbO), Deoxyhemoglobin (HbR), Blood Pressure (BP).**

I. Introduction

Microgravity causes the loss of gravitational force, which leads to a headward redistribution of body fluids from the lower extremities to the upper body [1]. This results in alterations in systemic circulation and cerebral perfusion. These changes can influence cerebral oxygenation and cardiovascular regulation, making it essential to understand the physiological adaptations that occur during spaceflight to ensure astronaut health and mission safety [2].

Head-down tilt bed rest at 6° angle is used as a gold-standard ground-based analog to simulate the physiological effects of microgravity and investigate the associated cardiovascular and cerebrovascular responses [3]. Previous studies have reported altered cerebral oxyhemoglobin concentrations and cerebral blood flow during microgravity exposure [4]. In addition to cerebral circulation, microgravity can also affect systemic hemodynamics and cardiac autonomic function, potentially contributing to orthostatic intolerance [5] and cardiovascular instability upon return to normal gravity. Prior research has demonstrated that HDT induces diverse autonomic and hemodynamic responses depending on exposure duration [6], [7]. Long-duration HDT studies have reported modulation of baroreflex sensitivity, altered sympathetic–parasympathetic balance [5], and increased cerebral oxygenation due to enhanced cranial blood flow [8].

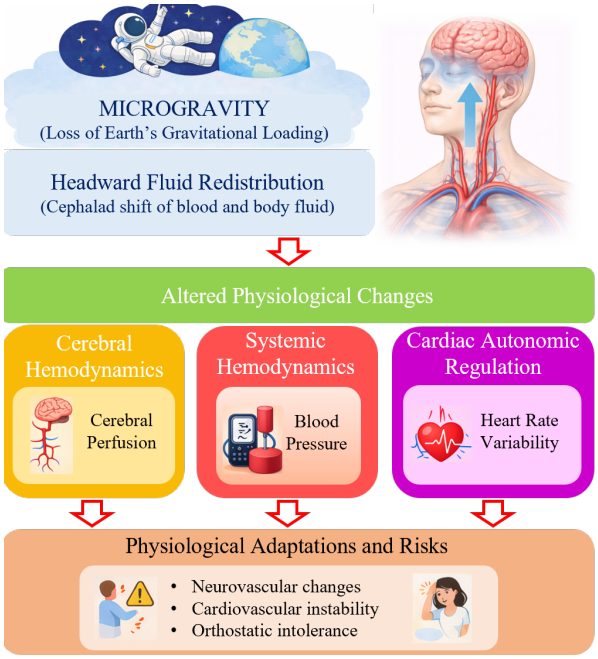

Fig. 1: Motivation for proposed study.

Previous studies have predominantly focused on long-duration microgravity exposure, whereas the physiological responses to simulated short-term microgravity remain insufficiently characterized. In particular, there is limited research examining the simultaneous behavior of cerebral hemodynamics, systemic blood pressure dynamics, and cardiac autonomic regulation during short-duration HDT exposure. A

comprehensive understanding of these integrated physiological responses is essential for identifying early biomarkers of microgravity-induced physiological stress with potential relevance for astronaut health monitoring and countermeasure development [2] as well as for future translational applications in clinical populations susceptible to orthostatic intolerance and autonomic dysfunction, including elderly individuals, immobilized patients, and those undergoing rehabilitation [9]. Such insights may also inform sports physiology in the context of training load management, recovery optimization, and autonomic reconditioning protocols [10].

To address the limited understanding of acute physiological adaptations to simulated microgravity, as shown in Fig. 1, this study investigates the effects of short-term 6° HDT on cerebral hemodynamics, blood pressure (BP), and cardiac autonomic function. The key contributions of this study are:

- Development of a multimodal physiological signal analysis framework integrating simultaneously acquired fNIRS signals, blood pressure, and peripheral pulsatile signals to capture cerebral, cardiovascular, and autonomic responses under simulated microgravity.
- Quantitative demonstration of coordinated neurovascular and hemodynamic adaptations to short-term 6° HDT through a paired pre–post experimental design.
- Evaluation of a non-invasive, signal-driven approach for assessment of acute microgravity-induced effects, with potential relevance to space health monitoring and neurovascular research.

II. METHODOLOGY

The proposed framework for this study is shown in Fig. 2.

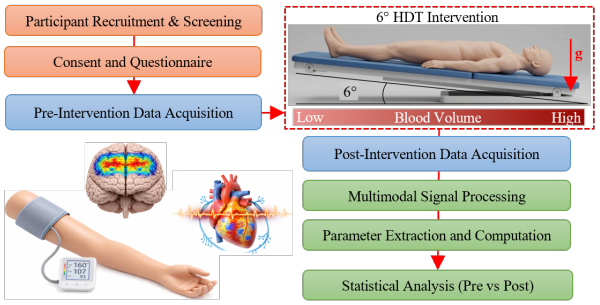

Fig. 2: Workflow of proposed physiological signal analysis framework under acute microgravity simulation.

A. Participants

A total of 25 healthy participants aged 23–34 years were initially recruited for the study through advertisements within the institute and by word of mouth. Participants were screened using the Physical Activity Readiness Questionnaire (PAR-Q+) to ensure eligibility. Subjects with smoking or alcohol use, regular medication intake, cardiovascular or neurological disorders, blood pressure abnormalities, diabetes, recent surgery, or any condition affecting cardiovascular or nervous system function were excluded. Out of 25 participants, 1 declined to participate, and 24 provided consent. Subsequently, 3 participants were excluded based on the inclusion criteria, resulting in 21 eligible individuals, of whom 2 were unavailable for the intervention. Finally, 19 eligible participants underwent the 6° HDT intervention and were included in the pre–post data analysis (Table I). The study was conducted with our custom-made lab setup in accordance with the Declaration of Helsinki and approved by our institute's ethical committee. Written informed consent was obtained from all participants prior to the commencement of the study.

TABLE I: Participants' demographic and anthropometric details.

Variables	Mean ± Standard Error of Mean
Participants	12 males, 7 females
Age (years)	26.95 ± 0.79
Weight (kg)	67.95 ± 3.26
Height (cm)	167.11 ± 2.40
Body-Mass Index (kg/m^2)	24.31 ± 1.02

B. Study Design

A single-group quasi-experimental pre–post study design was employed to investigate the acute physiological effects of simulated microgravity using 6° HDT. Upon arrival, participants' demographic information were taken. They were instructed to refrain from moderate-to-vigorous physical activity for 24 hours and avoid caffeine prior to testing. All sessions were conducted between 10:00 a.m. and 3:00 p.m. to minimize circadian influences. After adequate rest, baseline measurements of prefrontal fNIRS and fingertip pulse wave signals, along with systolic and diastolic blood pressure, were obtained for 5 minutes. Participants then underwent 30 minutes of 6° HDT, followed by post-HDT measurements using the same acquisition protocol for 5 minutes. Pre and post-HDT data were collected at rest in a seated upright position to minimize motion artifacts. The data acquisition hardware devices are summarized in Table II.

TABLE II: Physiological data acquisition hardware.

Physiological Data	Hardware
Blood Pressure Data	Digital Sphygmomanometer (Dr. Trust)
Peripheral Pulsatile Signal	Pulse Transducer TN1012/ST with PowerLab 15T-1340 (ADInstruments)
Prefrontal fNIRS Data	FNIR103S-1 54-channel 18-optode sensor pad with Model 2000S Imager Data Acquisition System (BIOPAC Systems, Inc.)

C. Microgravity Simulation Protocol

Participants were asked to lie on their back on a modified tilt bed, with the bed tilted downward at a 6° angle and head positioned lower than the feet, simulating headward fluid shifts similar to microgravity exposure, for a duration of 30 minutes, as shown in Fig. 3. They were instructed to remain awake, keep their legs straight, and avoid raising, contracting, or stretching them to minimize muscular activity.

D. Signal Processing Pipeline

The acquired multimodal physiological signals were processed using a structured signal processing pipeline before statistical feature extraction and analysis.

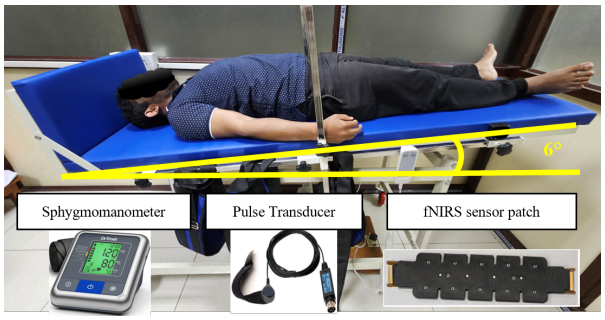

Fig. 3: Custom HDT laboratory experimental setup.

*1) **fNIRS Signal Processing**:* Recorded fNIRS signals were converted to *HbO* and *HbR* concentration changes using the modified Beer–Lambert law (MBLL), which were then interpolated to correct missing samples using linear interpolation. A 4th order Butterworth bandpass filter (0.005–0.5 Hz) was applied with zero-phase implementation to remove drift and high-frequency noise, including cardiac dynamics that fall above 0.5 Hz cutoff. Subsequently, Savitzky–Golay smoothing (3rd order, 51-sample window) was applied to preserve signal morphology, as shown in Fig. 4.

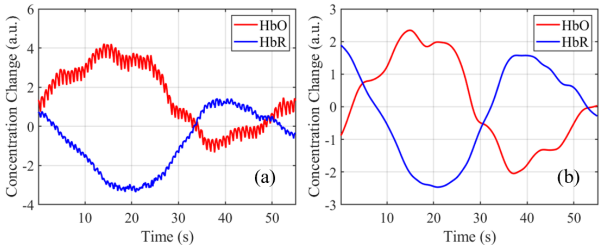

Fig. 4: Prefrontal fNIRS signals: (a) Raw signals and (b) Processed signals.

*2) **Peripheral Pulsatile Signal Processing**:* Peripheral pulse wave signals were interpolated to correct missing values, followed by FIR bandpass filtering (0.5–8 Hz) with zero-phase implementation to isolate the physiological waveform and remove noise. A 3rd order Savitzky–Golay smoothing (31-sample window) was applied to facilitate peak detection and derivation of inter-beat intervals (IBI).

E. Assessment of Cerebral Hemodynamics

Cerebral hemodynamics were assessed using a continuous-wave fNIRS system, with the sensor positioned over the prefrontal cortex [8] (centered at Fpz, 10–20 system). Measurements were acquired at 730 nm and 850 nm, corresponding to *HbO* and *HbR* absorption, with a sampling rate of 10 Hz. Relative concentration changes of chromophores *HbO* and *HbR* were derived from the recorded light intensity signals using MBLL. The relationship between changes in optical density (ΔOD) and chromophore concentration (ΔC) can be expressed as in Eq. (1)–(2):

$$\Delta OD = -\log\left(\frac{I}{I_0}\right), \tag{1}$$

$$\Delta C = \frac{\Delta OD}{\varepsilon \cdot L \cdot DPF} \tag{2}$$

where I and I_0 denote detected and incident light intensities, ε is the chromophore extinction coefficient specific to wavelength, L is the source–detector separation, and DPF accounts for photon scattering.

Further, to obtain an index of cerebral oxygenation, the *HbO–HbR* difference signal was computed as Eq. (3):

$$Hbdiff = HbO - HbR \tag{3}$$

which represents the balance between oxygen delivery and oxygen extraction in cortical tissue. An increase in $Hbdiff$ generally indicates improved cerebral oxygenation and regional cerebral blood flow [11]. *HbO* and $Hbdiff$ signals were averaged across all 48 retained long-separation channels spanning the prefrontal cortex region of interest (ROI), excluding 6 short-separation channels.

F. Assessment of Blood Pressure Dynamics

Systemic hemodynamics were evaluated by measuring arterial systolic (*SBP*) and diastolic blood pressure (*DBP*) in seated upright posture immediately before and after the HDT intervention. Pulse pressure (*PP*) and mean arterial pressure (*MAP*) were subsequently derived from *SBP* and *DBP* [12], using standardized equations Eq. (4)–(5):

$$PP = SBP - DBP, \tag{4}$$

$$MAP = \frac{SBP + 2 \times DBP}{3}. \tag{5}$$

G. Assessment of Heart Rate Variability

Pulse rate variability (PRV) derived from pulse-to-pulse intervals (PPI) was used as a surrogate for HRV in time-domain analysis due to its strong agreement with ECG-based HRV under resting conditions in healthy individuals [13]. Peripheral pulse wave signals were recorded at 1000 Hz sampling rate from the right index fingertip for 5 minutes at rest in the seated upright posture before and after the HDT intervention, minimising the potential influence of postural vascular changes on PRV-HRV agreement. The PPI series was used to compute mean heart rate (*MeanHR*), mean inter-beat interval (*MeanIBI*), standard deviation of inter-beat intervals (*SDNN*), and root mean square of successive interval differences (*RMSSD*).

H. Statistical Analysis

Normality of pre–post difference scores was assessed for each parameter using the Shapiro–Wilk test, supplemented by visual inspection of histograms. Parameters satisfying normality were compared using a paired t-test, while non-normal parameters were analysed using the Wilcoxon signed-rank test. Results are reported as mean (μ) $\pm$ standard error of the mean (SEM), supplemented by 95% confidence intervals (CI). Effect sizes for parametric comparisons were quantified using

TABLE III: Cerebral hemodynamic changes following 6° HDT.

Parameters	Pre ($\mu \pm$SEM)	Post ($\mu \pm$SEM)	p	d
HbO (a.u.)	0.0027 ± 0.0307	0.0851 ± 0.0287	0.0193	0.5896
$Hbdiff$ (a.u.)	0.0139 ± 0.0601	0.1682 ± 0.0567	0.0288	0.5451

Cohen's d, with thresholds defined as small (0.2), medium (0.5), and large (≥ 0.8), while rank-biserial correlation (r) was reported for non-parametric comparisons. The sign of effect sizes reflects the direction of change. To account for multiple comparisons, the Benjamini-Hochberg False Discovery Rate (FDR) correction was applied. All analyses were performed in MATLAB 2024a, with statistical significance set at $p < 0.05$.

III. Results and Discussion

Shapiro–Wilk test revealed that all parameters in Tables III, IV, V satisfied the normality assumption $p > 0.05$ except *DBP* ($p = 0.0271$), which violated normality. Wilcoxon signed-rank test was applied for *DBP*, while the paired t-test was applied for all remaining parameters.

HbO levels demonstrated a statistically significant increase following 6° HDT ($t(18) = 2.5701$, $p = 0.0193$), with a mean difference of 0.0823 ± 0.0320 a.u. (95% CI: $[0.0150, 0.1496]$) and a moderate effect size ($d = 0.5896$). Similarly, *Hbdiff* demonstrated a significant increase ($t(18) = 2.3762$, $p = 0.0288$), with a mean change of 0.1543 ± 0.0649 a.u. (95% CI: $[0.0179, 0.2907]$) and a moderate effect size ($d = 0.5451$), as evident in Fig. 5. Both *HbO* and *Hbdiff* survived Benjamini-Hochberg FDR correction, confirming the robustness of the observed cerebral oxygenation response. These findings, summarized in Table III, indicate a significant increase in prefrontal cerebral oxygenation under simulated microgravity conditions, which is visualized in Fig. 6 using oxygenation indices derived using BIOPAC fNIRSoft software from post-HDT fNIRS data of one subject. The concurrent elevation in both *Hbdiff* and *HbO* is consistent with headward fluid redistribution during HDT, which can enhance cerebral perfusion. The comparable moderate effect sizes across both parameters suggest a consistent neurovascular response, with *HbO* reflecting changes in oxygen delivery and *Hbdiff* capturing the net balance between oxygen supply and utilization.

As summarized in Table IV, *SBP* demonstrated a statistically significant reduction following 6° HDT ($t(18) = -5.267$, $p = 0.0001$), with a mean change of -4.4737 ± 0.8494 mmHg (95% CI: $[-6.2582, -2.6892]$) and a large effect size ($d = -1.208$). Similarly, *MAP* showed a significant reduction ($t(18) = -3.730$, $p = 0.0015$), with a mean change

TABLE IV: Systemic hemodynamic changes following 6° HDT.

Parameters	Pre ($\mu \pm$SEM)	Post ($\mu \pm$SEM)	p	d
SBP (mmHg)	113.42 ± 1.57	109.11 ± 1.57	0.0001	-1.208
DBP (mmHg)	78.32 ± 1.03	76.16 ± 1.12	0.0574	-0.496
MAP (mmHg)	90.14 ± 1.02	87.21 ± 1.10	0.0015	-0.856
PP (mmHg)	35.47 ± 1.42	33.16 ± 1.29	0.0567	-0.467

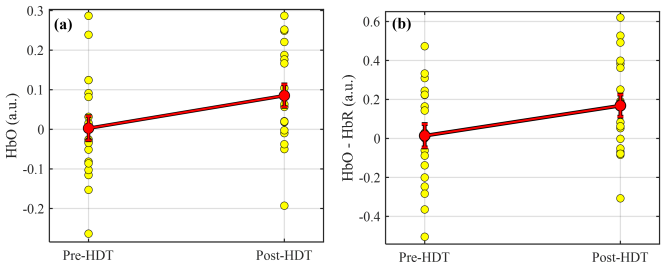

Fig. 5: Cerebral hemodynamic responses to HDT (Mean±SEM): (a) *HbO* and (b) *Hbdiff*.

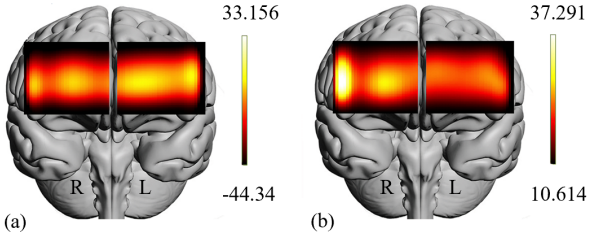

Fig. 6: Prefrontal cortex oxygenation heat-map: (a) Pre-HDT and (b) Post-HDT.

of -2.9298 ± 0.7855 mmHg (95% CI: $[-4.5801, -1.2796]$) and a large effect size ($d = -0.856$). *DBP* showed a statistically non-significant reduction ($W = 48$, $p = 0.0574$, $r = -0.495$) with a mean change of -2.1579 ± 0.9978 mmHg (95% CI:$[-4.2543, -0.0615]$). Similarly, *PP* showed a non-significant reduction ($t(18) = -2.037$, $p = 0.0567$, $d = -0.467$), with a mean change of -2.3158 ± 1.1370 mmHg (95% CI: $[-4.7045, 0.0729]$). Following FDR correction, *SBP* and *MAP* retained statistical significance, while *DBP* and *PP* did not survive correction, consistent with their borderline p-values. These findings indicate that short-term HDT induces significant reductions in *SBP* and *MAP*, while *DBP* and *PP* changes did not reach significance. The selective reduction in *SBP* and *MAP* with comparatively non-significant changes in *DBP* and *PP* illustrated in Fig. 7, suggests predominant modulation of mean arterial loading with relatively limited alteration in peripheral vascular resistance, likely attributable to cephalad fluid shifts and baroreflex-mediated cardiovascular adjustments.

Fig. 7: Blood pressure responses to HDT (Mean±SEM).

Peripheral pulsatile waveform analysis (Table V) revealed a significant reduction in *MeanHR* following 6° HDT

TABLE V: Cardiac autonomic changes following 6° HDT.

Parameters	Pre ($\mu \pm$SEM)	Post ($\mu \pm$SEM)	p	d
MeanIBI (s)	0.745 ± 0.018	0.762 ± 0.015	0.0259	0.5572
MeanHR (bpm)	81.397 ± 1.918	79.235 ± 1.540	0.0098	-0.6622
SDNN (s)	0.044 ± 0.004	0.054 ± 0.005	0.0236	0.5671
RMSSD (s)	0.037 ± 0.004	0.046 ± 0.005	0.0677	0.4459

($t(18) = -2.886$, $p = 0.0098$), with a mean difference of -2.1620 ± 0.7490 bpm (95% CI: $[-3.7356, -0.5883]$) and a moderate effect size ($d = -0.6622$), as shown in Fig. 8. *MeanIBI* showed a significant increase ($t(18) = 2.429$, $p = 0.0259$), with a mean change of 0.0177 ± 0.0073 s (95% CI: $[0.0024, 0.0330]$) and a moderate effect size ($d = 0.5572$). *SDNN* demonstrated a significant enhancement in overall variability ($t(18) = 2.472$, $p = 0.0236$), with a mean change of 0.0103 ± 0.0042 s (95% CI: $[0.0015, 0.0190]$) and a moderate effect size ($d = 0.5671$). *RMSSD* showed an increase post-HDT with a mean change of 0.0091 ± 0.0047 s (95% CI: $[-0.0007, 0.0190]$) but did not reach statistical significance ($t(18) = 1.944$, $p = 0.0677$, $d = 0.4459$). *MeanHR*, *MeanIBI*, and *SDNN* survived FDR correction, while *RMSSD* did not survive correction. These findings indicate significant HDT-induced alterations in cardiac timing dynamics, characterized by reduced *MeanHR* and increased *MeanIBI*. The increase in *SDNN* suggests enhanced overall variability, while the non-significant *RMSSD* change indicates limited short-term parasympathetic modulation. Collectively, these results suggest that simulated microgravity primarily affects global cardiac variability and timing, consistent with cephalad fluid redistribution augmenting venous return and triggering baroreceptor-mediated suppression of cardiac sympathetic drive.

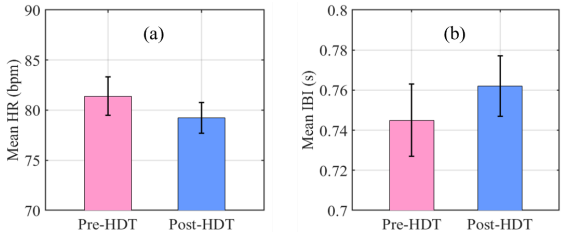

Fig. 8: Cardiac variability responses to HDT (Mean±SEM): (a) *MeanHR* and (b) *MeanIBI*.

IV. Conclusion and Future Scope

This study investigates acute physiological responses to short-term simulated microgravity using multimodal assessment of cerebral hemodynamics, systemic blood pressure, and peripheral pulsatile dynamics. Results show significant increases in both HbO and $Hbdiff$, indicating enhanced prefrontal cerebral oxygenation, alongside reductions in *SBP* and *MAP*, while *DBP* and *PP* showed non-significant changes. Peripheral pulsatile analysis revealed decreased *MeanHR* with a corresponding increase in *MeanIBI* and *SDNN*, and no significant change in *RMSSD*. All statistically significant findings were confirmed following FDR correction. The results indicate coordinated neurovascular and cardiovascular adaptations under simulated microgravity, characterized by enhanced prefrontal cerebral perfusion and modulation of cardiac timing and overall autonomic variability, consistent with hemodynamic adaptations to cephalad fluid redistribution, mediated by baroreflex-driven cardiovascular adjustments. The proposed framework highlights the potential of integrated biomedical signal analysis for astronaut monitoring and countermeasure development, and the findings may offer translational insights for clinical populations susceptible to orthostatic intolerance and autonomic dysfunction, as well as for sports physiology in the context of autonomic reconditioning and recovery optimization. The limitations of the present study include a relatively small sample size ($n = 19$) of only healthy young adults, short-duration HDT exposure, absence of a control condition, and prefrontal-only fNIRS coverage. Therefore, future work will focus on larger and more diverse cohorts with longer HDT exposures and inclusion of a control condition to better isolate HDT-specific physiological responses from posture and rest effects. Furthermore, incorporating cognitive task paradigms alongside resting assessments would help isolate neurovascular coupling from passive hemodynamic redistribution effects, providing deeper insight into HDT-induced cerebral oxygenation changes and their functional significance for astronaut cognitive performance.

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