Optical Hemodynamic Imaging of Jugular Venous Dynamics During Altered Central Venous Pressure

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Abstract—Objective: An optical imaging system is proposed for quantitatively assessing jugular venous response to altered central venous pressure. Methods: The proposed system assesses sub-surface optical absorption changes from jugular venous waveforms with a spatial calibration procedure to normalize incident tissue illumination. Widefield frames of the right lateral neck were captured and calibrated using a novel flexible surface calibration method. Jugular venous optical attenuation (JVA) signals were computed using an optical model for quantifying hemodynamic changes, and a jugular venous pulsatility map was computed using a time-synchronized arterial waveform. JVA was assessed in three cardiovascular protocols that altered central venous pressure by inducing acute central hypovolemia (lower body negative pressure), venous congestion (head-down tilt), and impaired cardiac filling (Valsalva maneuver). Results: JVA waveforms exhibited biphasic properties and sub-wave timing consistent with jugular venous pulse dynamics with time-synchronized electrocardiogram waves. JVA correlated strongly (median, interquartile range) with invasive central venous pressure during a graded central hypovolemia (r=0.85, [0.72, 0.95]), graded venous congestion (r=0.94, [0.84, 0.99]), and impaired cardiac filling (r=0.94, [0.85, 0.99]). Reduced JVA during graded acute hypovolemia was strongly correlated with reductions in stroke volume (SV) (r=0.85, [0.76, 0.92]) from baseline (SV: 79 ± 15 mL, JVA: 0.56 ± 0.10 a.u.) to −40 mmHg suction (SV: 59 ± 18 mL, JVA: 0.47 ± 0.05 a.u.; p<0.01). Conclusion: The proposed non-contact optical imaging system demonstrated jugular venous dynamics consistent with invasive central venous monitoring during three protocols that altered central venous pressure. Significance: This system provides non-invasive monitoring of pressure-induced jugular venous dynamics in clinically relevant conditions where catheterization is traditionally required, enabling monitoring in non-surgical environments.

Index Terms—Optical imaging, near-infrared, Beer-Lambert, photoplethysmography, central venous pressure, jugular vein.

I. INTRODUCTION

The cardiovascular system is a closed-loop system in which cardiac output and venous return remain in balance under state-steady conditions. Adequate venous return is essential to maintain cardiac output and regulate arterial blood pressure. Venous return is largely determined by the pressure gradient between the systemic veins and the right atrium [1]. Due to the high compliance of veins, venous blood volumes provide pressure-related fluid biomarkers of cardiac, systemic, and cerebrovascular regulation [2, 3].

Central venous pressure (CVP) is a major determinant of cardiac filling pressures and preload. Elevated jugular venous pressure (JVP) is a primary indicator of congestion in heart failure [4]. Heart failure and other diseases that restrict venous drainage, such as jugular vein thrombosis, are primary causes of intracranial hypertension and papilledema [5, 6]. Head-down tilt (HDT), a model for spaceflight microgravity and venous stagnation [7], has demonstrated chronically elevated intracranial pressure above seated levels on Earth over 24 h [8], and recent investigations found venous thrombosis and stagnation/retrograde flow in astronauts during spaceflight [9]. In contrast, hypovolemic states, such as acute blood loss, induce arterial pressure changes secondary to reduced cardiac filling with lower CVP [10]. Monitoring changes in CVP may provide important biomarkers relevant to cardiac and cerebral function; however, this measurement is not routinely performed due to invasive surgical right heart catheterization required to assess these pressure changes.

Due to its proximity to the heart, the jugular vein hemodynamics closely reflect those of CVP in many conditions. Elevated JVP is a biomarker of congestive heart failure, and provides insight into patient fluid status and response to diuretics [11, 12]. Clinically, jugular venous assessment is a visual task prone to inter-rater reliability errors [13]. Ultrasound has been used to quantify jugular distension for evaluating heart failure and right atrial pressure [14, 15], but is not suitable for longitudinal continuous monitoring. Mobile health devices capable of tracking JVP may enable breakthrough heart failure management beyond patient-reported functional and physiological data [16].

There has been recent interest in non-invasive and non-contact optical jugular venous monitoring. Skin surface displacement assessment from video [17], [18], [19] can enhance jugular venous pulse identification, but does not contain information related to jugular distension. Other methods have been derived from non-contact photoplethysmographic imaging systems that traditionally monitor arterial hemodynamics [20]. Photon migration properties of near-infrared light enable deeper tissue penetration than superficial visible spectrum light [21]. Thus, optical systems have been investigated to assess the JVP waveform using variations in optical
tube assembly and mounted in the same plane as the camera, beneath the camera lens. Tissue reflection was cross-polarized to reduce surface-level specular reflection, and near-infrared bandpass filtered prior to being imaged by the camera (GS3-U3-41C6NIR-C, FLIR).

B. Optical Model

For bulk homogeneous tissue, light attenuation (or optical density) can be calculated by the modified Beer-Lambert law [24]:

\[ A = -\log\left(\frac{I}{I_0}\right) = \sum \mu_{a,i}L + G \]  

where \( A \) is attenuation, \( I \) and \( I_0 \) are the detected and incident illumination, \( \mu_{a,i} = \log(10)\varepsilon_i c_i \) is the absorption coefficient of chromophore \( i \) based on its molar extinction coefficient \( \varepsilon_i \) and concentration \( c_i \), \( L \) is the photon mean path length through the tissue, and \( G \) is an intensity loss term due to scatter. With pulsatile flow, we are interested in modeling the temporal dynamics of light attenuation due to changes in blood volume (vessel diameter). A standard approach in continuous wave near-infrared spectroscopy is to use the attenuation differential with multiple wavelengths to estimate changes in hemoglobin concentrations [25]. However, when considering pulsatile vessels in unchanging tissue, it is primarily the photon path length that changes with vessel expansion/contraction [26]. Thus, expressing Eq. (1) in terms of time in this pulsatile model yields:

\[ A(t) = -\log\left(\frac{I(t)}{I_0}\right) = \mu_{a,t}L_t + \mu_{a,v}L_v(t) + G \]

where \( \mu_{a,t} \) and \( \mu_{a,v} \) are the absorption coefficients for the surrounding tissue and vessel, respectively, similarly for \( L_t \) and \( L_v \). We can further expand the vessel absorption:

\[ \mu_{a,v} = \alpha \mu_{a,HbO_2} + (1 - \alpha) \mu_{a,Hb} \]

where \( \alpha \) is the blood oxygen saturation in the vessel. Expressing attenuation in terms of temporal differences yields:

\[ \Delta A = -\log\left(\frac{I(t_2)}{I(t_1)}\right) = \mu_{a,v}(L_v(t_2) - L_v(t_1)) \]

Thus, assuming constant oxygen saturation in the vessel (constant \( \alpha \)), and that changes in the bulk tissue properties are small compared to changes in vessel absorption, changes in measured attenuation are linearly proportional to changes in mean photon path length through the vessel resulting from changes in vessel diameter.

C. Imaging Calibration

The formulation of Eq. (4) only holds if \( I_0 \) is constant across the differential measurements. Changes in tissue-imaging orientation cause differences in illumination profiles, making cross-condition comparison unreliable, since \( I_0 \) becomes a function of camera-tissue orientation. To compensate for this...
The calibration target was secured to the tissue using surgical tape to account for anthropometric differences across participants. Three sizes of the calibration target were developed extending from the clavicle to the mandible with good lateral coverage. The participant’s neck with no gap between the tissue and target, was calibrated using a custom flexible diffuse calibration target that was wrapped around the participant’s neck such that it followed the contour of the tissue. The calibration target was constructed from a 1/16 in white nitrile rubber sheet. Its strong resilience at thin profiles allowed it to conform to the tissue profile. Two thousand grit sandpaper was used to produce a highly diffuse uniform reflectance profile, approximating a Lambertian surface. The predicted calibration reflectance profile \( R_d(x_i) \) was therefore modeled as a uniform distribution outside the imaging area of interest. A calibration image was obtained before every acquisition by averaging 120 frames of the calibration target wrapped around the participant’s neck.

![Fig. 2: Overview of the jugular optical imaging system. Frames were captured of the right lateral area of the neck. Reflectance was calibrated using a custom flexible diffuse calibration target. Attenuation frames were computed from motion corrected and denoised reflectance frames, and used with the finger arterial waveform to generate a venous pulsatility map for jugular pulse waveform extraction.](image)

Fig. 2 depicts the processing pipeline for extracting jugular waveforms from a set of optical images. Reference images were acquired using a custom flexible calibration target that was wrapped around the participant’s neck such that it followed the contour of the tissue. The calibration target was constructed from a 1/16 in white nitrile rubber sheet. Its strong scattering properties resulted in minimal transmittance and maximal reflectance, ensuring consistent spatial and temporal properties that are unaffected by underlying tissue. The material’s resilience at thin profiles allowed it to conform to the tissue profile. Two thousand grit sandpaper was used to produce a highly diffuse uniform reflectance profile, approximating a Lambertian surface. The predicted calibration reflectance profile \( \hat{R_d}(x_i) \) was therefore modeled as a uniform distribution at unity. Calibration geometry was designed to conform to the participant’s neck with no gap between the tissue and target, extending from the clavicle to the mandible with good lateral coverage. Three sizes of the calibration target were developed to account for anthropometric differences across participants. The calibration target was secured to the tissue using surgical tape, which was applied to the back of the calibration target outside the imaging area of interest. A calibration image was obtained before every acquisition by averaging 120 frames of the calibration target wrapped around the participant’s neck.

D. Venous Pulsatility Map

Prior to attenuation calculation, each diffuse reflectance signal \( R_d(x_i) \) was down-sampled using a 4x4 mean filter to increase signal-to-noise ratio, and then denoised using a hemodynamic Kalman filter formulation [28]. This temporal domain filter has been shown to preserve waveform shape and feature timings by incorporating system specific noise models into a linear state estimation system conditioned on previous states. Briefly, the system state at each time point \( k \) was defined according to the signal measurement and rate of change, and the denoised signal was estimated using a smooth continuous vessel wall motion prior:

\[
\mathbf{s}_k = \begin{bmatrix} s_k \\ \dot{s}_k \end{bmatrix} = \begin{bmatrix} 1 & \Delta t \\ 0 & 1 \end{bmatrix} \mathbf{s}_{k-1}
\]

where \( s_k \) and \( \dot{s}_k \) are the signal amplitude and rate of change of the amplitude. The measurement noise model was characterized by the pixel standard deviation across the sensor in a dark field, and the process noise model was computed using a logarithmic grid search for optimal hemodynamic fit. The denoised diffuse reflectance signal was extracted at each time point using an observation matrix on the estimated state:

\[
\hat{R}_d(x_i, t_k) = H \mathbf{s}_k
\]

where \( H = [1 \ 0]^T \). Finally, the calibrated attenuation for cross-condition comparison was calculated for each location in each frame:

\[
A(x_i, t_k) = -\log(\hat{R}_d(x_i, t_k))
\]
and arterial circulations that are out of phase [23]. Thus, to localize the jugular venous pulse, a spatial correlation filter was performed against a time-synchronized finger arterial waveform [29]. Specifically, correlation between attenuation signals and the arterial waveform was computed for each tissue location $x_i$, producing a venous pulsatility map:

$$V(x_i) = r(A(x_i), z) - \mathbb{1}_{R^-}(r(A(x_i), z))$$  \hspace{1cm} (10)

where $r(A(x_i), z)$ is the linear correlation between attenuation signals and the arterial waveform, and $\mathbb{1}_{R^-}$ is the indicator function of negative real numbers. Thus, signals that are negatively correlated with the arterial waveform (i.e., out of phase) were identified, along with their associated correlation strength.

III. DATA COLLECTION AND EXPERIMENTAL DESIGN

A. Participants

Twenty young healthy adults (10 female, 24 ± 4 years, 169 ± 11 cm, 66 ± 13 kg) with no history of vascular, inflammatory, or thrombotic disease were enrolled. Participants abstained from caffeine, nicotine, alcohol, and heavy exercise for 24 h prior to testing and were fasted for 2 h prior to testing. Participants were asked to consume 2 L of water 2 h prior to testing to ensure a hydrated state. One participant’s data were removed due to an insufficient area for imaging, and another participant’s LBNP data were removed due to acquisition issues. The study was approved by a University of Waterloo Research Ethics Committee (ORE #40394) and all protocols conformed to the Declaration of Helsinki. All participants provided written informed consent prior to testing.

B. Experimental Protocol

Participants were instrumented with an electrocardiogram (iE33 xMatrix, Philips Healthcare, Andover, USA), continuous arterial blood pressure plethysmograph with estimated stroke volume (SV) via Modelflow (NOVA, Finapres Medical Systems, Enschede, NL), a nasal cannula connected to a capnograph for end-tidal carbon dioxide ($P_{\text{ET}} CO_2$; CD-3A CO2 Analyzer, AMETEK Inc., Pittsburgh, PA, USA), and a respiratory belt (Model 0528, Respiratory Effort PVDF Sensor, Braebon, NY, USA). Continuous CVP was measured using a catheter placed in a vein in the right antecubital fossa [30]. The vein was punctured using a single-use 20G needle and attached to a pressure monitoring transducer through a saline-filled polyurethane catheter. The pressure transducer was positioned at the level of the right midclavicular line using a laser level to estimate the level of the right heart and manually calibrated at 0, 5, 10, and 15 cmH2O. Participants were tilted to the right to ensure a continuous column of fluid between the right atrium and an antecubital vein. LBNP and Valsalva pressures were monitored using a digital manometer. The imaging head was positioned such that it provided orthogonal illumination to the right lateral area of the neck. The system was moved with the participant between conditions to maintain a similar field of view and angle of illumination.

Data were recorded at 1000 Hz (PowerLab, LabChart, version 7.3.7; ADInstruments, Colorado Springs, CO). Hemodynamic frames were captured in uncompressed video format at 60 fps with 16 ms exposure time, and a finger photoplethysmography probe was transmitted concurrently with video frames at 100 Hz and down-sampled to 60 Hz to match the video frame rate. A hardware trigger was transmitted to PowerLab to synchronize the cardiovascular and imaging acquisitions.

Three maneuvers were performed that systematically changed CVP dynamics: LBNP (reduced venous return from central hypovolemia), HDT (venous congestion through cephalad fluid shift [32], [33]), and Valsalva maneuver (impaired cardiac filling from increased intrathoracic pressure). All participants underwent three HDT conditions (0, −3, and −6 °) and four LBNP conditions (0, −20, −30, and −40 mmHg), with randomized ordering of the two protocols. Approximately 5 min was spent in each condition. A calibration and 30 s video were acquired during the last minute of each condition. LBNP was terminated if systolic blood pressure fell below 70 mmHg at any point during the collection. HDT and LBNP protocols were separated by at least 5-minutes of supine rest. Participants then voluntarily completed a 15 s Valsalva maneuver to a pressure of 40 mmHg (or the participant’s maximum attainable pressure) while breathing into a mouthpiece, with one practice maneuver prior to collection. Data were recorded during the Valsalva, as well as 15 s before (baseline) and after (recovery). To compensate for participant movement during the Valsalva, frames were registered by tracking a salient feature on the neck using a linear dual correlation filter [34].

C. Data Analysis

Data were analyzed in LabChart (ADInstruments Inc., Colorado, USA) using beat-by-beat averages calculated over the same 30 s period in which imaging occurred. Baseline and recovery measures during the Valsalva protocol were extracted as 10 s averages starting at the beginning of the protocol and 5 s after the Valsalva maneuver was stopped, respectively. The peak measure during the Valsalva protocol corresponded with the CVP apex. The jugular vein was identified by a contiguous negative correlation segment on the right lateral area of the neck in the baseline frames. For each participant, a jugular venous waveform was extracted for each 30 s condition acquisition in the same area in the center of the vessel track, of approximately 1 cm long, identified by a strongly negatively correlated cohesive area close to the clavicle.

Main effects of protocol condition (HDT angle, LBNP pressure level, Valsalva stage) were determined by one-way repeated measures ANOVA followed by planned contrasts between condition to baseline using paired sample t-test when significant main effects were observed. Significant differences were reported at $p < 0.05$ with Bonferroni multiple comparison correction. Pearson correlation coefficients were computed to investigate linear relationships between normalized changes in JVA, CVP and SV. Data are presented as mean ± SD.
TABLE I: Cardiovascular Response to Three Types of Cardiovascular Stress

| Condition | HR (bpm) | SV (mL) | MAP (mmHg) | CVP (cmH2O) | JVA (a.u.) |
|-----------|----------|---------|------------|-------------|-----------|
| Lower body negative pressure | | | | | |
| 0 | 60 ± 7 | 79 ± 15 | 80 ± 9 | 11.2 ± 3.5 | 0.56 ± 0.10 |
| −20 | 64 ± 9* | 70 ± 17* | 81 ± 11 | 7.0 ± 3.9* | 0.51 ± 0.08* |
| −30 | 68 ± 10* | 64 ± 18* | 82 ± 9 | 6.5 ± 3.9* | 0.50 ± 0.07* |
| −40 | 75 ± 13* | 59 ± 18* | 85 ± 9 | 6.3 ± 5.0* | 0.47 ± 0.05* |
| Head-down tilt (tilt angle) | | | | | |
| 0 | 62 ± 8 | 81 ± 16 | 80 ± 9 | 11.6 ± 4.5 | 0.35 ± 0.09 |
| −3° | 61 ± 9 | 82 ± 15 | 81 ± 9 | 13.8 ± 4.6* | 0.58 ± 0.10* |
| −6° | 62 ± 9 | 80 ± 15 | 80 ± 11 | 16.4 ± 4.6* | 0.61 ± 0.10* |
| Valsalva | | | | | |
| baseline | 67 ± 11 | 84 ± 20 | 91 ± 8 | 11.7 ± 4.3 | 0.71 ± 0.23 |
| peak | 85 ± 16* | 43 ± 19* | 96 ± 13 | 29.0 ± 11.6* | 0.78 ± 0.22* |
| recovery | 64 ± 7 | 83 ± 19 | 99 ± 11* | 13.1 ± 3.3* | 0.74 ± 0.23* |

Values (mean ± SD) were averaged across the 30 s video acquisition in lower body negative pressure and head-down tilt. Valsalva values show 10 s average 5 s before (baseline) and after (recovery) the maneuver, and during maximal CVP (peak). HR: heart rate; SV: stroke volume; MAP: mean arterial pressure; CVP: central venous pressure; JVA: jugular venous optical attenuation (calibrated). *p<0.05 compared to baseline (paired sample t-test).

Fig. 3: Individual and averaged jugular venous waveforms for each participant while supine, normalized to the cardiac cycle. The start of each signal was time aligned to the ECG R wave.

IV. RESULTS

A. Jugular Venous Attenuation Signal Extraction

Fig. 3 shows per-participant average and individual JVA waveforms during supine baseline. Each signal’s start time was set to the ECG R wave, and expressed as a fraction of the cardiac cycle. The biphasic nature and primary subwaves (a, c, x, v, y) of the JVP were visually apparent across participant waveforms. Recognizing the start time as the peak of the R wave (ventricular depolarization), the following pulse wave characteristics were observed: a small rise in pressure during early systole (c wave); a downstroke following the R wave during ventricular contraction from right atrial relaxation and tricuspid valve closure (x wave); an inflection point approximately half way through the cardiac cycle consistent with right atrial filling (v wave) and tricuspid valve opening (y wave); and an upstroke preceding the R wave during atrial contraction (a wave).

B. Acute Central Hypovolemia

Acute central hypovolemia through graded LBNP resulted in progressively reduced venous return and, accordingly, SV (Fig. 4). Significant main effects were observed in heart rate, SV, CVP, and JVA (Table I). Heart rate was elevated and SV was depressed during all LBNP levels compared to baseline. These changes in SV are indicative of decreased cardiac filling pressures due to the translocation of blood to the lower extremities. This was corroborated by CVP, which decreased significantly from baseline (11.2 ± 3.5 cmH2O), with the largest decreases occurring on initial −20 mmHg LBNP (7.0 ± 3.9 cmH2O) followed by smaller absolute decreases in −30 mmHg (6.5 ± 3.9 cmH2O) and −40 mmHg (6.3 ± 5.0 cmH2O) LBNP. A similar pattern of JVA changes was observed, with significant decreases in all grades compared to baseline (0.56±0.10 a.u.), the largest occurring upon LBNP onset (0.51±0.08 a.u.) followed by smaller absolute decreases in −30 mmHg (0.50±0.07 a.u.) and −40 mmHg (0.47±0.05 a.u.). JVA exhibited strong positive linear correlation (median, interquartile range) to CVP (r=0.85, [0.72, 0.95]) and SV (r=0.85, [0.76, 0.92]) during LBNP.

C. Venous Congestion

HDT induced a cephalad fluid shift through hydrostatic pressure gradient toward the head, resulting in increased venous pressure (Fig. 5). There were no significant main effects of central cardiovascular variables (heart rate, SV, MAP) with increased HDT compared to supine baseline (see Table I). Significant main effects were observed in CVP and JVA. CVP increased significantly from baseline (11.6 ± 4.5 cmH2O) during −3° (13.8 ± 4.6 cmH2O) and −6° (16.4 ± 4.6 cmH2O) HDT, with a concomitant significant increase in JVA from supine (0.55 ± 0.09 a.u.) to −3° (0.58 ± 0.10 a.u.) and −6° (0.61 ± 0.10 a.u.) HDT, signifying increased optical absorption with pressure-induced venous dilation. JVA exhibited strong positive linear correlation (median, interquartile range) to CVP during HDT (r=0.94, [0.84, 0.99]).
Fig. 4: Change in jugular venous attenuation (JVA) compared to central venous pressure (CVP) during (a) head-down tilt, (b) Valsalva, as well as (c) CVP and (d) stroke volume (SV) during lower-body negative pressure. Data are expressed as changes relative to the participant’s baseline supine measures. Error bars indicate 95% confidence intervals. See Table I for statistical comparisons.

**D. Impaired Cardiac Filling**

Venous return was experimentally impeded through a Valsalva maneuver. Fig. 5 shows a representative example of the effect of Valsalva on JVA and CVP signals. Following normal baseline respiration, Valsalva strain resulted in an increase in intrathoracic pressure causing an increase in external pressure on the heart and thoracic blood vessels. Compression of the superior vena cava impedes venous return and results in distended IJV and increases in superior vena cava and central venous pressures [35]. This duality was demonstrated by significantly lower SV at peak (43±19 mL) compared to baseline (84±20 mL), from reduced venous return, and significantly higher CVP at peak (29.0±11.6 cmH$_2$O) compared to baseline (11.7±4.3 cmH$_2$O; see Table I and Fig. 4). A similar significant increase in JVA was observed from baseline (0.71±0.23 a.u.) to peak (0.78±0.22 a.u.), consistent with jugular venous distension. Both CVP and JVA increased progressively with continued fluid congestion until strain release, and exhibited marginally higher levels than baseline during recovery (CVP: 13.1 vs. 11.7 cmH$_2$O; JVA: 0.74 vs. 0.71 a.u.). JVA exhibited strong positive linear correlation (median, interquartile range) to CVP during the baseline-peak-recovery phases ($r=0.94$, [0.85, 0.99]).

**V. DISCUSSION**

In this work, we proposed a non-contact, sub-surface optical imaging system to monitor changes in jugular venous dynamics. We have shown that JVA closely tracks induced increases and decreases in CVP during hemodynamic stresses induced by LBNP, HDT and Valsalva. Three physiological protocols were designed to test multiple mechanisms of CVP modulation: (1) reducing venous return through LBNP; and (2) increasing venous pressure through venous congestion in HDT; and (3) restricting venous return by increasing intrathoracic pressure with a Valsalva maneuver. Taken together, the span of effects of these three protocols have clinical and physiological

![Fig. 5](image-url)
cardiovascular responses to spaceflight, with 6° being widely
has been extensively used as an Earth-based analog to study
pressure and jugular vein thrombosis in space [8], [9]. HDT
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changes may enable enhanced monitoring in trauma.

Non-contact optical imaging methods for extracting hemo-
dynamic signals, or summary measures such as heart rate, are
relatively new technologies that have been gaining increased
interest in the literature [20]. Instrumentation varies between
studies, but many comprise some combination of a camera
with or without a controlled light source. Relying on the same
optical theory as finger photoplethysmography, these systems
produce unitless measurements, and thus are not suitable for
longitudinal comparison in their basic form. Diffuse optical
imaging and spectroscopy methods are able to quantify ab-
sorption per unit length of tissue through calibration pro-
cedures [27], [38], but are typically restricted in geometry
and assumptions about tissue homogeneity. We combined the
two approaches to yield a calibrated non-contact optical
imaging method for comparing changes in optical attenuation
from venous dilation by effectively determining the per-pixel
incident illumination by normalizing inhomogeneities in the
optical system and resulting from the surface profile.

LBNP causes a translocation of central blood volume to
the lower body vascular compartments without altering grav-
itational or muscular loading effects, and is an experimen-
tal model for cardiovascular response to orthostatic stress
and acute hemorrhage [39], [10]. Our experimental design
simulated the equivalent of approximately 500–1000 mL of
blood loss across the chosen pressure levels [40]. This central
hypovolemia reduced venous return leading to activation of
cardiopulmonary baroreceptors and reduction in stroke volume
that reduces arterial pulse pressure causing elevated heart
rate, sympathoexcitation and release of arginine vasopressin
to reflexively maintain arterial and cerebral perfusion pres-
sures [41]. Our data tracked the effects of greater LBNP with
reduced CVP and commensurate JVA. In some participants,
CVP was close to zero during the last stage of LBNP. MAP
remained unchanged after 4 min of exposure to each level,
indicating an effective cardiovascular response of vasocon-
striction and increased heart rate. Monitoring arterial pressure
may lead to missed early biomarkers of shock [42], whereas
venous pressures may be more indicative of early signs of
hypovolemia [10]. Thus, optical monitoring of jugular venous
changes may enable enhanced monitoring in trauma.

Venous congestion and stasis during spaceflight results from
the removal of the gravitational force which modulates venous
return. In comparison to on Earth, a cephalad fluid shift moves
venous blood away from the lower limbs toward the heart
and head, and has been associated with increased intracranial
pressure and jugular vein thrombosis in space [8], [9]. HDT
has been extensively used as an Earth-based analog to study
cardiovascular responses to spaceflight, with 6° being widely
used as a model of 0 G [43]. The resulting jugular distension
can be visually seen on astronauts aboard the International
Space Station, and is consistent with increases in JVA ob-
served in this study. Exposure to 24 h simulated spaceflight
microgravity has shown that intracranial pressure levels are
chronically elevated above those observed during 90° seated
position on Earth [8], which has implications in ocular re-
modeling in space [7]. A mismatch between intracranial and
intraocular pressure is one of the primary hypotheses behind
visual acuity impairments in space [44]. Non-intrusive contin-
uous measurements of jugular venous volume and waveform
over an entire mission may enable identification of abnormal
responses to spaceflight, or evaluation of the effectiveness of
countermeasures designed to limit the severity of spaceflight
associated neuro-ocular syndrome.

Restricted venous return is present is right heart failure,
where inefficient cardiac function leads to fluid overload. In
congestive heart failure, reduced cardiac function results in
central fluid accumulation with elevated CVP. Jugular venous
distension is associated with prognosis of hospitalization and
disease progression in heart failure [45]. Thus, a large em-
phasis is placed on managing, and therefore monitoring, fluid
levels [46]. Since jugular venous distension correlates well to
right atrial pressure and left ventricular filling pressures [47],
[15], we used a Valsalva maneuver to systematically increase
intrathoracic and cardiac pressures. Changes in jugular ve-
nous diameter through ultrasound assessment are a positive
predictor of heart failure status due largely to elevated right
atrial pressure [15], [14]. In our healthy participant sample,
we observed significant changes in JVA during the Valsalva
strain compared to baseline, indicating a measured difference
in jugular distension, and suggesting that it may be a clinically
relevant non-contact alternative for heart failure monitoring.

The primary limitation of this work is the assumption that
all other physiological and environmental systems remain
constant across conditions, and that the change in optical
attenuation was solely due to changes in blood volume. This
was achieved by performing the three protocols in a single
data collection session, where environmental and physiological
conditions could be controlled. However, during longitudinal
monitoring, additional factors that could alter tissue optical
properties must be explicitly modeled, such as blood oxygen
saturation, hematocrit, and skin blood volume. We also ac-
knowledge that CVP was used to validate observed changes
in JVA, which is a measurement of blood volume in the vein,
and could be influenced by venous compliance. Exceptionally
high pressures may result in a reduced volume response. Thus,
the generalizability of these results to changes in already high
venous pressures as well as changes in venous tone requires
further research with affected populations.

VI. Conclusion

In this paper, we proposed a non-contact optical imag-
ing system for assessing changes in jugular venous optical
attenuation resulting from altered central venous pressure.
Comparison of optical attenuation of the jugular vein in dif-
ferent imaging configurations was performed using a proposed
surface profile calibration and signal denoising pipeline. Using a time-synchronized arterial waveform, the jugular venous waveform was extracted and compared in three physiological protocols: central hypovolemia, venous congestion, and impaired cardiac filling. Results showed that changes in jugular venous optical attenuation strongly correlated with changes in central venous pressure, as well as reduced stroke volume during central hypovolemia. These results suggest that non-contact optical imaging can be used for assessing clinically relevant hemodynamic conditions without the need for invasive catheterization.

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