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No influence of steady-state postural changes on cerebrovascular compliance in humans

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ABSTRACT

The aim of this study was to determine the effect of posture changes on vascular compliance in intracranial (brain) vs. extracranial vascular beds (forearm). Eighteen young adults (nine females) performed a supine-to-seated-to-standing protocol involving five minutes of rest in each position. Continuous blood pressure, middle cerebral artery (MCA) blood velocity, and brachial artery blood velocity were recorded at each posture. Three to five consecutive steady-state cardiac cycles at each posture were analyzed by a four-element lumped parameter modified Windkessel model to calculate vascular compliance. Mean arterial pressure (MAP) increased from supine to seated (76[9] vs 81[12] mmHg; P=0.006) and from supine to standing (76[9] vs 82[13] mmHg; P=0.034). Mean blood flow was greater in the MCA relative to the forearm (forearm: 40[5] ml•min⁻¹, MCA: 224[17] ml•min⁻¹; main effect P<0.001). Conversely, vascular resistance (forearm: 3.25[0.50] mmHg⁻¹•ml•min⁻¹, brain: 0.36[0.04] mmHg⁻¹•ml•min⁻¹; main effect P<0.001) and compliance (forearm: 0.010[0.001] ml•min⁻¹•mmHg⁻¹, brain: 0.005[0.001] ml•min⁻¹•mmHg⁻¹; main effect P=0.001) were greater in the forearm compared to the brain. Significant main effects of posture were observed with decreasing values in upright positions for mean blood flow (P=0.001) in both vascular beds, but not for resistance (P=0.163) or compliance (P=0.385). There were no significant interaction effects between vascular bed and posture for mean flow (P=0.057), resistance (P=0.258), or compliance (P=0.329). This study provides evidence that under steady state conditions, posture does not affect cerebrovascular compliance.

Key words: posture, vascular compliance, cerebral blood flow, middle cerebral artery, transcranial Doppler ultrasound
INTRODUCTION

The demands of the cerebrovascular bed are complex and unique, which requires the functional integration of multiple regulatory mechanisms to supply constant oxygen to neural tissues (Willie et al., 2014). In addition to dynamic cerebral autoregulation, which adjusts cerebrovascular tone based on transient changes to blood pressure, the intricate anatomy of the cerebrovascular bed – including the Circle of Willis – introduces complexities to the passive abilities of blood vessels to contribute to blood flow control (i.e., vascular mechanics) (Hoiland, Fisher and Ainslie, 2019).

Indeed, vascular compliance is an integral contributor to pulsatile blood flow control and is a key feature of vascular mechanics involved in absorbing and storing the energy generated by the heart in systole, to be used later in diastole. Specifically, vascular compliance is the ability of blood vessels to mechanically distend by increasing volume for a given change in pressure (London and Guerin, 1999). This feature also minimizes pulsatile energy transition into the microcirculation while facilitating continuous downstream blood flow during diastole. Previous work from our laboratory using a modified Windkessel model demonstrated that reductions in blood pressure during a rapid sit-to-stand maneuver transiently increased compliance in the cerebrovascular bed (Moir et al., 2020). Importantly, cerebrovascular compliance increased prior to the onset of autoregulatory compensation (i.e., decreased cerebrovascular resistance) and preserved systolic velocity in the middle cerebral artery (MCA) (Schondorf, Benoit and Wein, 1997; Moir et al., 2020). In each of these earlier studies, increased cerebrovascular compliance occurred in conjunction with decreases in blood pressure, highlighting the importance of compliance in rapid control of blood flow during orthostatic stress.
Similarly, Zamir et al. (2018) used a four-element Windkessel model to predict that, under conditions of supine rest, the pressurized environment within the skull limits cerebrovascular compliance compared to extravascular arterial beds (e.g., the forearm) due to cerebral tissues creating a high intracranial pressure (ICP). However, ICP changes with postural shifts (via caudal cerebral spinal fluid movement) may also modify transmural pressure, meaning that extravascular pressure will decrease compared to intra-arterial pressure during upright posture, thereby exposing inherent vascular elastic properties. In fact, ICP is lower in the seated position where the head and spinal column are vertical compared to the supine position (Lawley et al., 2017). Accordingly, the independent effects of hydrostatic gradient due to body posture and transmural pressure due to rapid reductions in arterial pressure, immediately following a postural change or within a steady state, remain to be explored.

In healthy individuals, a decrease in blood pressure in response to postural shifts is transient (Moir et al., 2020), although residual hydrostatic differences between the heart and brain will persist beyond the early and transient period of orthostasis (Lawley et al., 2017). Therefore, evaluating cerebrovascular compliance in different steady-state postures, after transient hemodynamic changes have recovered, will allow us to dissect mechanisms of intravascular (arterial pressure) vs. extravascular (ICP) influences on cerebrovascular compliance.

The purpose of this study was to examine the effects of steady-state posture changes on cerebrovascular compliance. We tested the hypothesis that hydrostatic blood pressure mechanisms are primary determinants of cerebrovascular compliance, where cerebrovascular compliance increases with progressive changes into the upright posture. As a control, we
included measures of forearm vascular compliance sustained at heart level in which changes in extravascular pressure are unlikely to occur.

MATERIALS AND METHODS

Participants

Eighteen young, healthy adults (mean [standard deviation]; age 24[3] years, 9 males, 9 females, height 170[13] cm, weight 66[14] kg, BMI 23[4] kg•m⁻²) volunteered to participate in this study. Exclusion criteria were diagnosis with any form of cardiorespiratory, neurological, or metabolic disease, obesity, hypertension, smoking, pregnancy, ineligibility for magnetic resonance imaging (MRI), and any medication prescribed for these conditions. The study was approved by the Health Sciences Research Ethics Board at Western University (#112633) and conformed to the standards of the Declaration of Helsinki, with the exception of registration in a database. Written informed consent was obtained from each participant prior to testing.

Experimental Protocol

The experimental protocol consisted of two visits; a laboratory visit and a MRI visit. Both visits were conducted within a maximum timeframe of two months and were performed at the same time of day for each participant. Females were tested during the same phase of their menstrual cycle for both visits. Participants were asked to fast for at least 4 hours and abstain from exercise, alcohol consumption, and caffeine for at least 12 hours prior to each visit.

Laboratory Experimental Protocol

Participants laid in the supine position for at least ten minutes to reach a stable baseline and three brachial blood pressure measurements were collected with automated auscultation. Participants were also equipped with a lead II electrocardiogram to monitor heart rate, and finger
photoplethysmography (Finometer Model 1, Finapres Medical Systems, Amsterdam, The Netherlands) was used to measure arterial blood pressure waveforms continuously, with correction for brachial artery pressures. End-tidal carbon dioxide partial pressure ($P_{ET\text{CO}_2}$) was collected at the mouth and sampled via gas analyzer (ML206, ADInstruments, Colorado Springs, CO, USA). Participants were further instrumented with a 2 MHz PW Doppler ultrasound probe (Neurovision TOC2M, Multigon Industries, Elmsford, NY, USA) to record the peak blood velocity of the left middle cerebral artery (MCAv). As an experimental control for a vascular bed exposed to postural changes without extravascular pressure caused by the skull, forearm blood was also measured simultaneously to transcranial Doppler ultrasound. Duplex ultrasound (10-MHz probe; Vivid iQ System, GE Healthcare Canada, Mississauga, ON, Canada) was used to record the mean intensity-weighted blood velocity of the right brachial artery via power spectrum analyzer (500M, Multigon Industries, Elmsford, NY, USA). To analyze the diameter of the brachial artery offline, two-dimensional images were saved, and manual application calipers were used to measure the diameter. All continuous data were captured using an analog-to-digital conversion and data acquisition system (LabChart 8, 32/16 PowerLab, ADInstruments, Colorado Springs, CO, USA). The collection of the data was performed at 1,000 Hz and stored for offline analysis.

Following a minimum of ten minutes of rest in the supine position, one minute of data were collected. Participants then moved into a seated position at the edge of the bed. A sling was applied to secure the left arm and finger cuff at heart level. The right arm of the participants remained on an adjustable table, in a fixed position at heart level throughout the protocol. The participants rested in the seated position for five minutes to achieve a steady state data collection in the final minute. Lastly, participants transitioned into a standing position without assistance or
balance support, and remained in this position for five minutes, with data collection occurring in the final minute.

**MRI Experimental Protocol**

Magnetic resonance images were collected on a Siemens Magnetom Prisma 3 T scanner with a 32-channel head coil at the Centre for Functional and Metabolic Mapping at Western University in London, Canada. Participants remained in a supine position for the duration of the scan. Following time of flight angiography to map vessel anatomy, high resolution 2D T2-weighted images (0.35 mm isotropic) were acquired in the sagittal orientation (TE = 96 ms, TR = 3000 ms, FOV = 160 x 160 mm, BW = 225 Hz/pixel, TA = 1:15 min) through the M1 segment of the MCA to determine cross-sectional area (CSA). The CSA of the MCA was measured from MRI DICOM files using OsiriX software (Pixmeo©, Geneva, Switzerland). Two observers (ICC=0.969, range 0.862-0.994, P<0.001, n=8) manually traced the border of the MCA using the closed polygon measurement tool.

**Data Analysis**

The mean arterial pressure (MAP) was corrected using the manual blood pressure measurements that were collected at baseline. This was completed by finding one minute of constant blood pressure at baseline obtained from finger photoplethysmography and then adding or subtracting from systolic and diastolic values to match the average blood pressure obtained from baseline automated auscultations. These corrections were maintained throughout the study. Two observers (ICC=0.985, range 0.927-0.997, P<0.001, n=8) manually measured the distance from intima to intima of the near and far walls of the brachial artery aligned in time with the R-wave of the ECG. The blood velocity of the brachial artery obtained from the Duplex ultrasound and the blood velocity of the MCA obtained from the transcranial Doppler ultrasonography were
used to calculate blood flow (Equation 1) in combination with the corresponding CSA measures
(brachial artery: acquired from duplex ultrasound; MCA: acquired from MRI scans). Vascular
resistance was then calculated from blood flow and mean blood pressure values (Equation 2).

Equation 1: Flow = blood velocity * CSA * 60
Equation 2: Resistance = Mean blood pressure / Mean blood flow

During the minute of data collection in each posture, three to five consecutive cardiac
cycles were selected for analysis of hemodynamic and vascular mechanics variables (e.g.,
compliance, see below). Care was taken to ensure selections for data analysis were acquired from
sections of data with stable heart rate, blood pressure, and blood velocity. The same 3-5 cardiac
cycles were selected for both the brachial artery and the MCA. Blood pressure and blood
velocity waveforms were temporally aligned, using a shift value between -0.97 and -1.07
seconds, for each individual cardiac cycle prior to use in the Windkessel model.

Windkessel Analysis

The nature of pulsatile blood flow is determined by the mechanical properties of the
downstream vascular bed including vascular resistance (R), compliance (C), viscoelasticity (K),
and inertial effects (L). These four elements can be computed using the relationship between
oscillatory components of pressure and flow from a lumped Windkessel model (see below)
(Zamir et al., 2007, 2018). Vascular resistance is initially determined by the quotient of mean
blood pressure and mean flow over a cardiac cycle (Equation 2). Initial values for C, K, and L
are then set in the Windkessel model ($\omega=$ oscillatory frequency; $i=\sqrt{-1}$) for vascular impedance
(represented by Z; Equation 3) and through a series of up to 1000 iterations, the model
systematically modifies each element (C, K, L) until the predicted blood flow waveform
(Equation 4) in each of its first ten harmonics reaches the lowest error between predicted and measured flow waveforms.

Equation 3: \[ Z = \frac{R(\omega KC + i(\omega^2 LC - 1))}{\omega C(K + R) + i(\omega^2 LC - 1)} \]

Equation 4: \[ Q = \frac{P}{Z} \]

Here, \( R \) and \( C \) values are reported because they are relevant to our research question.

One forearm resistance value (supine) and one brain compliance value (supine) were excluded based on identification as extreme outliers according to Grubbs’ test (\( P < 0.05 \)).

### Statistical Analysis

Using the Shapiro-Wilk test it was determined that the data were normally distributed. A one-way repeated measures ANOVA was used to evaluate the effect of posture (i.e., supine, seated and standing) on mean arterial pressure (MAP), heart rate, and \( P_{ETCO_2} \). A two-way repeated measures or mixed model ANOVA was used to evaluate the interaction effect of posture (i.e., supine, seated and standing) and vascular bed (i.e., arm/brachial artery and brain/MCA) on mean flow, vascular resistance, and vascular compliance. Bonferroni-corrected pairwise comparisons were made following a significant interaction. A three-way repeated measures ANOVA was used to evaluate the interaction effect of posture (i.e., supine, seated and standing), vascular bed (i.e., brachial artery and MCA), and sex on vascular compliance. There were no main effects or interaction effects present with sex, therefore the data was pooled and sex-based differences are not reported. The reported data is in mean (standard deviation). Statistical significance was determined by using \( P \leq 0.05 \). The GraphPad version 9 software (Prism, San Diego, CA, USA) was used for the statistical analysis and creation of figures.

### RESULTS
Absolute values for heart rate and MAP are presented in Table 1. There was a main effect of posture (P<0.001) where heart rate increased from supine in both the seated (P=0.028) and standing (P<0.001) postures. Similarly, MAP increased when progressing through the postural changes (P=0.005). Specifically, post hoc comparisons revealed a significant increase in MAP from supine to seated (P=0.006) with a mean difference of 5(6) mmHg, and a significant increase between supine and standing (P=0.034) with a mean difference of 5(8) mmHg. There was no significant difference in MAP, between the seated and standing postures (P=0.999). No main effect of posture on pulse pressure was observed (P=0.063). There was a main effect of posture on P_{ET}CO\textsubscript{2} (n=17; P<0.001) where values decreased from supine [44(5) mmHg] in both the seated [42(4) mmHg; P=0.023] and standing [40 (5) mmHg; P<0.001] positions.

There was a main effect of vascular bed (P<0.001) for blood flow where mean MCA flow values were greater than the mean flow values for the brachial artery (Figure 1). There was a main effect of posture (P=0.001) with decreasing mean flow values in both vascular beds from supine, to seated, and to standing. No significant interaction effect between posture and vascular bed was present on blood flow (P=0.057). A main effect of vascular bed (P<0.001) was observed for vascular resistance, with significantly greater resistance in the forearm compared to the brain (Figure 2); however, no effect of posture (P=0.163) or posture by vascular bed interaction (P=0.258) was observed for vascular resistance. A main effect of vascular bed was observed (P<0.001) for vascular compliance with greater values in the forearm compared to the brain in each posture (Figure 3). No main effect of posture (P=0.385) or posture by vascular bed interaction (P=0.329) was observed for vascular compliance. As a relative change from supine, vascular compliance values were higher in the brain by 227 (595)\% in the seated and 77 (171)\% in the standing postures, respectively, albeit not statistically significant compared to the forearm in the seated [-7 (24)\%; P=0.125] or standing
postures [-7 (26)%; P=0.067]. Compared to supine, forearm vascular compliance values were lower
by 7 (24)% in the seated and 7 (26)% in the standing postures. However, there were no main effects
of posture (P=0.151) or vascular bed (P=0.082), and no interaction between vascular bed and posture
(P=0.099) existed for relative changes in vascular compliance.

**DISCUSSION**

The present study investigated the influence of steady-state posture changes on
cerebrovascular compliance, with a comparison to the compliance of an extracranial vascular bed
(forearm) fixed at heart level. There was an expected main effect of vascular bed where
compliance values were larger in the forearm compared to the brain (Figure 3), but, in contrast to
the hypothesis, there were no main effects of posture and no interaction effect of posture and
vascular bed for compliance. Additional findings indicate that MAP increased when changing
into the upright postures, and mean blood velocity decreased slightly (Figure 1) due, likely, to
the concurrent modest reductions in P_{ET}CO_2. We interpret these data as evidence that under
steady state conditions, cerebrovascular compliance is resistant to changes in posture.

Based on Windkessel modelling, Zamir *et al.* (2018) reported forearm blood velocity
waveforms when compliance was both lowered and raised in the forearm, but the predicted
cerebral blood velocity waveform only changed when compliance was increased in the brain.
Thus, Zamir *et al.* (2018) proposed that cerebrovascular compliance is restrained by the rigid
confines of the skull that prevent volume expansion of the intracranial fluids. Indeed, our current
results confirm these modelled outcomes and demonstrate that cerebrovascular compliance is
markedly lower than forearm vascular compliance (Figure 3). The lower values of compliance
found in the cerebral vasculature might be explained by the influence of ICP, which is
determined by all compartments of the brain (e.g., brain tissue, cerebrospinal fluid, arterial blood
volume, and venous blood volume). Since compliance is the ability of vessels to adjust CSA for a given change in pressure, the pressure applied by the extravascular environment onto a vascular bed could restrict its compliance. However, it is experimentally challenging to manipulate intracranial volume due to the cranial anatomy, cerebral autoregulation, and the technical difficulty in obtaining direct measures of ICP in humans. Lawley et al. (2017) leveraged the existence of Ommaya reservoirs in a group of individuals and tested the effects of posture changes on ICP. In these direct measurements, ICP was approximately 10 mmHg lower in the upright seated posture compared to supine, which can be attributed to fluid shifts (i.e., cerebrospinal fluid shifts into the spinal cord) (Magnaes, 1976; Lawley et al., 2017; Petersen and Ogoh, 2019). Therefore, it is possible that the reduced ICP would permit an increase in cerebrovascular compliance. However, the current study that focused on steady state conditions does not support this contention.

The lack of change in cerebrovascular compliance in the current study was not expected and explanations for this outcome remain speculative. First, the lack of change may be related to the conditions under which data were collected. Moir et al. (2020) reported an increase in compliance immediately following a decrease in MAP during the transition into the upright posture. In contrast, the current data focussed on steady state conditions at each posture when MAP normally returns to supine levels, or, as observed here, increased above supine levels. Notably, as reported by Moir et al. (2020), cerebrovascular compliance changes with moving from the seated to upright postures are transient and quickly recover to the seated baseline level. This earlier study did not report steady state values beyond the immediate recovery of blood pressure. Combined, the observations suggest that increased compliance on moving into the upright posture is a transient response related solely to hypotension and not posture per se.
Further, at steady state the gravitational effect on the hydrostatic gradient between heart and brain in the upright versus supine postures is compensated partially by the concurrent rise in MAP of ~5-6 mmHg. We note too that the earlier measures of ICP changes of ~10 mmHg between supine and upright postures (Lawley et al., 2017) would offset the hydrostatic reductions in MAP (intraluminal pressure) such that transmural pressure may not be different in the steady state conditions of different postures. In fact, Lawley et al. (2017) reported that, when accounting for the heart-to-head hydrostatic fluid column and changes in ICP, cerebral perfusion pressure was not different between supine and upright postures.

Increased heart rate (Table 1) may also counteract increases in compliance related to reduced transmural pressure, but the effects of atrial pacing have opposite effects in large vs. small arteries (Huo, Chen and Kassab, 2018) and the impact of heart rate on cerebrovascular compliance has not been tested. Similarly, distending pressure (i.e., pulse pressure) as a result of changing stroke volume and MAP may exert differential influences on compliance (Mitchell, 1999). Indeed, we observed that MAP increased on going from the supine position to standing, but pulse pressure did not change between postures (Table 1) and stroke volume was not measured. Further, the current changes in posture induced reductions in $P_{ET\,CO_2}$ and this may have affected the current outcomes. Previously, hypercapnia decreased cerebrovascular compliance (Moir et al., 2021), presumably by distention of the elastic component of the vascular wall. However, the direct effects of ~4 mmHg reduction in $P_{ET\,CO_2}$ as observed in the current study, on vascular compliance are unknown. It is clear, however, that cerebrovascular and forearm vascular beds behave differently in response to changes in $P_{ET\,CO_2}$. Specifically, extreme hypocapnia (~20 mmHg $P_{ET\,CO_2}$) through hyperventilation causes cerebral vasoconstriction but, as reported earlier (Burnum, Hickam and McIntosh, 1954), forearm
vasodilation. However, we note that vascular resistance contributes to regulation of the steady
state component of flow whereas vascular compliance reflects the distensibility of the vascular
wall and regulation over the oscillatory component of flow. The current data suggest that mild
hypocapnia has little effect on either cerebral or forearm vascular compliance.

There are several limitations to the present study that must be acknowledged. For
instance, the Windkessel model assumes that blood flow and blood pressure waveforms are
obtained at the same location in the vascular tree. However, measures of ICP and intracranial
blood pressure are not possible in most conscious humans. Therefore, peripheral blood pressure
was used as a surrogate for cerebral perfusion pressure. However, changes in ICP with postural
adjustments are reported (Magnæs, 1976; Lawley et al., 2017) and we are confident in their
general decline on moving from supine to seated and upright postures. We accept the limitation
of uncertain intracranial blood pressure waveforms and values recognizing that it is systematic
across all levels of the study and the results align with those of the forearm. Future studies using
direct measures of intracranial MCA blood pressure waveforms are needed; however, it is
expected that any differences in the blood pressure waveform will be similar in shape between
peripheral and cerebrovascular regions despite variations in absolute values by ~30% (Blanco,
Müller and Spence, 2017). In addition, PETCO₂ decreased with changes in posture and these may
affect cerebrovascular compliance. Specifically, we reported a decrease in cerebral compliance
(Moir et al., 2021) following inspiration of 5% carbon dioxide for four minutes, likely due to
dilation of the cerebrovascular bed and consequent transition from elastic vascular elements to
the stiffer collagen element. Whether the opposite occurs with hypocapnia is not known.
Available evidence indicates that hypocapnia in the supine position has a smaller effect on MCA
diameter relative to hypercapnia (Coverdale et al., 2014) and, considering the evidence above
that cerebral dilation reduces compliance, any hypocapnia induced constriction might lead to an increase in compliance. This was not observed in the current study with changes in posture.

The use of ultrasound and MRI to obtain the CSA of the vessels allowed for the calculation of blood flow and not solely velocity. Two authors manually measured the diameters and CSAs, providing strong inter-rater reliability and standardized analysis. However, MRI-based detection of cross-sectional area of the MCA was limited to strictly the supine position. It has been previously demonstrated by Serrador et al. (2000) that with the use of lower body negative pressure to implement a similar stress to the orthostatic changes, the diameter of the MCA remained unchanged; however, the use of 1.5T MRI at the time may not have had sufficient resolution to fully detect changes in the diameter of the MCA. More recent evidence using CT imaging corroborated the notion that the CSA of the MCA does not change from the supine to seated posture, but the small sample size may have led to insufficient statistical power to detect changes in MCA diameter (Kosugi et al., 2020). No further anatomical evaluation of vessels within the intracranial vascular bed, other than the MCA, were collected from the MRI images in the present study. Therefore, the current data cannot address inter-individual differences in brain vascular anatomy or how these might influence compliance or resistance. Finally, the differences in vascular compliance between the forearm and brain require judicious consideration. For example, reporting relative changes between vascular beds is one approach to account for baseline differences, but our results demonstrate vascular compliance values that vary by an order of magnitude between the forearm and brain. In this case, mathematical differences are innate when calculating percent changes.

Conclusion
The cerebrovascular bed exhibits lower compliance compared to the forearm supporting the idea that different pressurized environments, such as intracranial vs extracranial spaces, may influence pulsatile vascular mechanics (i.e., compliance). There were no changes to compliance in either the forearm or cerebrovascular beds following the transition from supine to seated or standing steady-state postures. These findings indicate that the contribution of compliance to blood flow control may be greater in the transient stages of posture change, as observed by Moir et al. (2020), rather than steady-state conditions evaluated in the present study, and that posture per se does not alter compliance.
Acknowledgements

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Competing Interests

The authors declare there are no competing interests.

Author Contributions

A.M.K., M.E.M., G.B.C., A.W.D., S.A.K., B.K.A. performed experiments; A.M.K., M.E.M.,
results of experiments; A.M.K., G.B.C. prepared figures; A.M.K., G.B.C. drafted manuscript;
A.M.K., M.E.M., G.B.C., J.K.S. edited and revised manuscript; A.M.K., M.E.M., G.B.C.,
A.W.D., S.A.K., B.K.A., J.K.S. approved final version of manuscript.

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BrainsCAN at Western University with funding from the Canada First Research Excellence
Fund.

Data Availability

Data will be made available upon reasonable request.
References


Kosugi, K. *et al.* (2020) ‘Posture-induced changes in the vessels of the head and neck: evaluation using conventional supine CT and upright CT’, *Scientific Reports*, 10(1), p. 16623. Available at: https://doi.org/10.1038/s41598-020-73658-0.


Table 1. Hemodynamic and mechanical properties of the peripheral (forearm) and cerebral (brain) vascular beds by posture (n=18, 9 females).

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>Seated</th>
<th>Standing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart Rate (bpm)</td>
<td>58 (6)</td>
<td>66 (10)*</td>
<td>75 (10)*</td>
</tr>
<tr>
<td>Mean Arterial Pressure (mmHg)</td>
<td>76 (9)</td>
<td>81 (12)*</td>
<td>82 (13)*</td>
</tr>
<tr>
<td>Pulse Pressure (mmHg)</td>
<td>50 (11)</td>
<td>48 (10)</td>
<td>42 (10)</td>
</tr>
</tbody>
</table>

**Forearm**

<p>| | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Cross-sectional Area (cm²)</td>
<td>0.103 (0.030)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Mean Blood Velocity (cm/s)</td>
<td>6.9 (4.6)</td>
<td>5.7 (4.0)</td>
<td>6.1 (4.4)</td>
</tr>
<tr>
<td>Mean Blood Flow (ml·min⁻¹)</td>
<td>46 (37)</td>
<td>36 (28)</td>
<td>39 (30)</td>
</tr>
<tr>
<td>Compliance (Δml·min⁻¹·ΔmmHg⁻¹)</td>
<td>0.011 (0.005)</td>
<td>0.010 (0.005)</td>
<td>0.010 (0.005)</td>
</tr>
<tr>
<td>Resistance (mmHg·ml·min⁻¹)</td>
<td>2.79 (2.14)</td>
<td>3.72 (2.76)</td>
<td>3.60 (2.84)</td>
</tr>
</tbody>
</table>

**Brain**

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<thead>
<tr>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td>Cross-sectional Area (cm²)</td>
<td>0.062 (0.014)</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Mean Blood Velocity (cm/s)</td>
<td>67.2 (14.8)</td>
<td>60.0 (14.6)</td>
<td>58.5 (13.7)</td>
</tr>
<tr>
<td>Mean Blood Flow (ml·min⁻¹)</td>
<td>244 (59)</td>
<td>217 (52)</td>
<td>212 (51)</td>
</tr>
<tr>
<td>Compliance (Δml·min⁻¹·ΔmmHg⁻¹)</td>
<td>0.005 (0.009)</td>
<td>0.006 (0.006)</td>
<td>0.004 (0.002)</td>
</tr>
<tr>
<td>Resistance (mmHg·ml·min⁻¹)</td>
<td>0.33 (0.09)</td>
<td>0.40 (0.13)</td>
<td>0.41 (0.13)</td>
</tr>
</tbody>
</table>

Values are reported as mean (standard deviation). *Denotes a significant difference (P≤0.05) from supine according to Bonferroni-corrected post hoc tests.
Figure 1. Mean blood flow (ml•min⁻¹) values at each posture of supine, seated, and standing (n=18, 9 females). Bars represent the group mean while error bars represent standard deviation, and each symbol represents an individual.

Figure 2. Vascular resistance (mmHg•ml•min⁻¹) values at each posture of supine, seated, and standing (n=18, 9 females). Note the right y-axis for brain values due to scale differences. Bars represent the group mean while error bars represent standard deviation, and each symbol represents an individual. NB: n=17 for forearm standing condition.

Figure 3. Vascular compliance (Δml•ΔmmHg⁻¹) values at each posture of supine, seated, and standing (n=18, 9 females). Bars represent the group mean while error bars represent standard deviation, and each symbol represents an individual. NB: n=17 for supine brain condition.
Posture $P=0.001$
Vascular bed $P<0.001$
Interaction $P=0.057$

- ○ Forearm
- ▲ Brain

*Figure 1.*
Posture P=0.163
Vascular bed P<0.001
Interaction P=0.258

Figure 2.
Figure 3.

Posture $P=0.385$
Vascular bed $P<0.001$
Interaction $P=0.329$

○ Forearm
△ Brain