Effect of upper body position on arterial stiffness: Influence of hydrostatic pressure and autonomic function

Running Title: Body position and arterial stiffness

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Abstract:

Objective: To evaluate changes in arterial stiffness with positional change and whether the stiffness changes are due to hydrostatic pressure alone or if physiological changes in vasoconstriction of the conduit arteries play a role in the modulation of arterial stiffness.

Methods: Thirty participants' (male=15, 24±4 years) upper bodies were positioned at 0°, 45°, and 72° angles. Pulse wave velocity (PWV), cardio-ankle vascular index (CAVI), carotid beta-stiffness index, carotid blood pressure (cBP), and carotid diameters were measured at each position. A gravitational height correction was determined using the vertical fluid column distance (mmHg) between the heart and carotid artery. Carotid beta-stiffness was calibrated using three methods: 1) non-height corrected cBP of each position, 2) height corrected cBP of each position, 3) height corrected cBP of the supine position (theoretical model). LF_{SAP} was analyzed as a marker of sympathetic activity.

Results: PWV and CAVI increased with position ($p<0.05$). Carotid beta-stiffness did not increase if not corrected for hydrostatic pressure. Arterial stiffness indices based on Method 2 were not different from Method 3 ($p=0.65$). LF_{SAP} increased in more upright positions ($p<0.05$) but diastolic diameter relative to diastolic pressure did not ($p>0.05$).

Conclusion: Arterial stiffness increases with a more upright body position. Carotid beta-stiffness needs to be calibrated accounting for hydrostatic effects of gravity if measured in a seated position. It is unclear why PWV increased as this increase was independent of blood pressure. No difference between Method 2 and 3 presumably indicates that the beta-stiffness increases are only pressure dependent, despite the increase in vascular sympathetic modulation.

Key Words: (3-10 – use MESH terms) Arterial stiffness, Positional change, Autonomic function, Vascular function

Introduction

Elasticity of the arterial wall is decreased in aging and disease, and arterial stiffness is a valuable predictor of cardiovascular events, hypertension and all-cause mortality [1,2]. An elastic aorta buffers the distending pressure generated with ventricular contraction, slowing pulse wave velocity, thus preventing the transmission of detrimental pulsatile energy to the microvasculature of target end-organs [2]. The gold standard for measuring arterial stiffness is carotid-femoral pulse wave velocity (PWV) (e.g. central PWV), typically measured non-invasively by either tonometry or oscillometric techniques [3]. Carotid beta (β)-stiffness is an alternative measure of arterial stiffness and utilizes ultrasound imaging of the common carotid artery with echo-tracking, while accounting for arterial pressure [4]. Cardio-ankle vascular index (CAVI) is a tertiary measure of arterial stiffness, which is blood pressure independent similarly to β-stiffness [5].

PWV, β-stiffness and CAVI are generally performed in and have been validated for the supine position [6,7]. However, both BP and arterial stiffness are often measured in a semi-recumbent or seated position in the clinic or during clinical investigations [7]. Previous research has shown an increase in heart rate and blood pressure (BP) moving from supine to upright positions [8-12], but only a few studies have addressed the effects of a semi-recumbent or seated position on parameters of arterial stiffness [8,13]. Body position may be especially important for determination of carotid BP and consequently β-stiffness, since carotid BP is calibrated in the supine position and this calibration will not account for changes in hydrostatic pressure as result of a more upright body position. For measures requiring a carotid pressure calibration, such as β–stiffness, the brachial pressures should potentially be corrected for the hydrostatic pressure differences to arrive at a better estimate of the true carotid pressure to calculate β-stiffness [14]. Further investigation of the influence of gravity

on blood pressure and arterial stiffness changes in the semi-recumbent seated position compared to the traditional supine position is warranted.

The change from supine to a more upright body position also involves modulation of sympathetic activity (e.g. baroreflex). In an upright position, baroreceptors are unloaded resulting in a reflexive heart rate increase due to parasympathetic withdrawal, followed by sympathetic activation resulting in increased systemic vascular resistance to help maintain cardiac output and blood pressure [15]. The physiological response is dependent on the gravitational challenge, but it is unknown if a semi-recumbent seated position would yield similar findings as previously reported from lower body negative pressure (LBNP) [16,17] or head-up tilt [18-21]. Therefore, it is important to understand the potential impact of sympathetic activation and vasoconstriction when moving from a supine to semi-recumbent position, and the impact on common arterial stiffness measures.

The question then arises whether the change in arterial stiffness noted with positional change is reflective of the effects of gravity on hydrostatic pressure alone and can be fully accounted for with an applied height correction, or whether physiological changes in vasoconstriction of the conduit arteries play a role in the modulation of arterial stiffness. Our aim was to investigate first if the change in β-stiffness, after application of a height correction to the estimation of carotid pressures, is similar in direction and magnitude to PWV, and second, whether there is an additional effect of autonomic modulation (on both diameter and pressures in the carotid artery) in a seated position compared to supine, after correcting for changes in hydrostatic pressure. We hypothesized that β-stiffness with a height correction and PWV will change across positions in a similar direction and magnitude, and that the seated position will not create a large enough gravitational challenge to influence autonomic modulation. If both our hypotheses are demonstrated to be valid, the applied height correction

will enable an adequate comparison of β-stiffness index between the seated and supine position.

Methods

Participants

Using a cross-sectional design, 30 volunteers between the ages of 18 and 35 years (15 males) were recruited for participation. All participants were free of known cardiovascular or pulmonary disorders, non-smokers, and not on any medication known to affect the cardiovascular system. On experimental days, participants reported to the lab having refrained from caffeine, alcohol, and heavy exercise for at least 24 hours and fasted for a minimum of 4 hours. A health history and physical activity questionnaire, as well as height (cm) and weight (kg) measurements, were completed. Written informed consent was obtained from all participants, and the University of Illinois at Chicago Institutional Review Board approved the study protocol.

Experimental Procedure

Participants were positioned on an examination table with an adjustable back at 0° (supine), 45° (semi-recumbent), and 72° (more upright) (Figure 1). To minimize the effect of position order, the starting position and following order was randomized using computer software (Excel, Microsoft, Redmond, WA) prior to the start of the study. Participants were prepared for 3-lead electrocardiography (ECG), PWV, CAVI, and continuous measurement of arterial blood pressure and respiratory waveforms. After 5 minutes of quiet rest in a temperature controlled-room (20°-22° C), data were collected under each randomized condition.

Hemodynamic Measures

Beat-to-beat heart rate was measured with a 3-lead electrocardiogram and arterial blood pressure was measured continuously via photoplethysmography on the middle finger of the right hand (Finometer Pro, Finapres Medical Systems, Amsterdam, Netherlands). Data were recorded on a computer for offline analysis using a data acquisition system (MP150, BIOPAC Systems, Goleta, CA) at a sampling rate of 1 kHz. Aortic diameter (measured in the supine position with ultrasonography) was used to correct for calculated hemodynamic variables, as suggested by the manufacturer. All data were free of ectopy and artifacts as confirmed by visual inspection of all recordings by one investigator. The beat-to-beat blood pressure waveforms were processed with WinCPRS software (Absolute Aliens Oy, Turku, Finland) to generate the beat-to-beat systolic blood pressure time series and determine, with autoregressive modeling, the LF_{SAP} (power in 0.04–0.15Hz) of blood pressure variability. LF_{SAP} is considered a non-invasive representation of vascular sympathetic modulation [22].

Carotid Pressure and Height Correction

Applanation tonometry (SphygmoCor, AtCor Medical, Sydney, Australia) was used to measure pressure waveforms of the right common carotid artery, calibrated with the brachial diastolic and mean arterial pressures (see below) (VaSera VS 1500AU, Fukuda Denshi, Japan). The height sensor on the beat-to-beat blood pressure cuff was used to estimate the vertical fluid column, expressed in mmHg, between the heart level and the level of the carotid blood pressure measurement during the 45° and 72° positions (Figure 1). We used this pressure difference as a 'height correction' to account for gravitational effects in the semi-recumbent and seated position. The pressure in mmHg was subtracted from brachial pressures (heart level) to calibrate the carotid pressure waveform in the applanation tonometry device (i.e. if the brachial blood pressure was 120/80 mmHg, mean arterial pressure 93 mmHg, and the height in mmHg between the heart and the carotid artery was 10 mmHg, the pressure utilized for the applanation tonometry calibration was 70 mmHg, 83 mmHg).

To calculate carotid arterial stiffness in each position, we used three different methods of estimating carotid pressures:

Method 1: pressure waveforms of the right common carotid artery were calibrated with the brachial diastolic and mean arterial pressures **of that position without any height correction,**

Method 2: pressure waveforms of the right common carotid artery were calibrated with the **height-corrected** brachial diastolic and mean arterial pressures **of that position**,

Method 3: pressure waveforms of the right common carotid artery were calibrated with the **height-corrected** brachial diastolic and mean arterial pressures **of the supine position** [theoretical model].

Method 3 was implemented under the assumption that carotid pressures only change from the supine to seated positions due to the effects of gravity. This method allows us to address aim 2, as a comparison with Method 2 will determine if the effects of gravity were the only influence on changes in pressure and stiffness or if the sympathetic nervous system also played a role.

Pulse Wave Velocity

Pulse wave velocity (PWV) was assessed measuring carotid artery pulse waves via applanation tonometry, while simultaneously obtaining femoral pulse waves via an oscillometric technique with a partially inflated cuff over the femoral artery, midway between the thigh and knee (SphygmoCor XCEL, AtCor Medical, Sydney, Australia). This device has previously been validated to produce reliable measurements compared to tonometry-based devices [23,24]. PWV was calculated using the ratio of the corrected distance between the

pulse measuring sites to the time delay between the carotid and femoral pulse waves [24]. Distance was measured with a large caliper (1) from the carotid site of measurement to the suprasternal notch, (2) from the suprasternal notch to the proximal edge of the thigh cuff and (3) from the inguinal ligament to the proximal edge of the thigh cuff. Distances 1 and 3 were subtracted from distance 2 and used in the calculation of PWV [24]. All measurements were made in duplicate with a less than 0.5 m/s difference between measures, and the mean value was used for subsequent analysis. In addition, PWV was normalized to brachial mean arterial pressure of each position (PWV/MAP) and the heart rate of each position (PWV/HR). PWV was not obtained from one participant due to device malfunction.

Carotid Beta Stiffness

The right common carotid artery was imaged longitudinally approximately 1-2 centimeters proximal to the bifurcation via ultrasonography (Hitachi-Aloka Alpha 7, Tokyo, Japan). Automatic wall detection echo-tracking software was used to create pressure waveforms and vessel diameter values for calculation of relative diameter change, β-stiffness index, arterial compliance and arterial elastic modulus. Relative diameter change = $[(D_{systolic}-D_{diastolic})/D_{diastolic}]*100$; β-stiffness index = $[\ln(P_{systolic}/P_{diastolic})]/[(D_{systolic}-D_{diastolic})/D_{diastolic}]$ [4]; arterial compliance (AC) = $\Delta volume/(P_{systolic}-P_{diastolic})$; and arterial elastic modulus (Ep) = $(P_{systolic}-P_{diastolic})/[(D_{systolic}-D_{diastolic})/D_{diastolic}]$ where $P_{systolic}$ and $P_{diastolic}$ are systolic and diastolic blood pressure (mmHg), respectively, and $D_{systolic}$ and $D_{diastolic}$ are the arterial diameters [7]. β-stiffness index was calculated in each position using the carotid pressures estimated by the three different methods, as described above.

Cardio-Ankle Vascular Index

CAVI was assessed using a non-invasive device (VaSera VS 1500AU, Fukuda Denshi, Japan). Blood pressure cuffs placed on the left upper arm and ankle to generate

plethysmograms, ECG leads placed on the wrists, and a phonocardiogram placed right next to the left sternal border were used to assess CAVI by incorporating the time between heart sound II and the plethysmograms taken at the brachial and ankle arteries [5].

Statistical Analysis

Descriptive data are reported as mean ± SD. For the first objective we used a linear mixed-effects model with repeated measures over position (time) and an unstructured covariance matrix to determine differences (direction and magnitude) across the positions for all outcome variables. Analyses took into account covariates including age, sex, and BMI. Data were checked for normality and log-transformed where necessary. All data are reported as raw means ± standard deviation (SD). When significant positional effects were noted, post-hoc analyses were conducted with Bonferroni corrections for multiple comparisons.

For the second objective, differences between method 2 and method 3 for β-stiffness index were analyzed using a two-by-three repeated measures ANOVA to determine if the effects of gravity were the only influence on changes in arterial stiffness or if another factor (presumably the sympathetic nervous system) played a role. Statistical analyses were performed with SAS software (SAS Institute, Cary, NC), and all *P* values are 2-sided, with an a priori α-level of 0.05 determined to be significant.

Results

Subject characteristics are presented in Table 1. Table 2 presents the effects of positional change on hemodynamic and vascular variables in all participants. The hemodynamic and vascular response to positional change was not influenced by sex (Supplemental Table 1).

Differences Across Positions

Positional change resulted in significant increases in heart rate and brachial SBP, DBP, and MAP ($p<0.05$). Carotid SBP (cSBP) and DBP (cDBP) (Method 1) increased from 0° to 45° and 0° to 72° ($p<0.05$), with no significant difference between 45° and 72° ($p > 0.05$). Application of the height correction to the cSBP and cDBP (Method 2) resulted in reductions in cSBP and cDBP across all positions ($p < 0.05$). Similar reductions in cSBP and cDBP were seen with the use of Method 3 for calculation ($p < 0.05$).

Central arterial stiffness measured by PWV increased across all positions (Figure 3), even after correction for MAP and HR ($p<0.05$). This increase in arterial stiffness with positional change was also observed in measurements of CAVI ($p<0.05$). β-stiffness index, on the other hand, did not change across positions ($p>0.05$) when calibrated using Method 1 for carotid blood pressures. Calibrating the carotid blood pressures with the height correction (as shown by Method 2, Figure 2) did result in an increase of β-stiffness index with change in position (Figure 3).

Impact of Autonomic Modulation

Figure 4 displays β-stiffness index determined using each method of carotid blood pressure calibration. In an attempt to determine if the effects of gravity were the only influence on changes in pressure and stiffness or if the sympathetic nervous system played a role, the results of Method 2 were compared to Method 3. There was no difference between Method 2 and Method 3 ($p=0.65$), indicating a possible lack of influence by the sympathetic nervous system on the carotid artery.

These results are seen albeit noting an increase in markers of sympathetic activation and reduction in markers of parasympathetic activity and baroreceptor sensitivity (Table 3). LF_{SAP} and LF/HF ratio increased in a more upright position compared to supine ($p <0.05$).

AlphaLF and RRI BRS decreased across positions ($p < 0.05$), as well as RMSSD and HF_{RRI}. LF_{RRI}, a mix of sympathetic and parasympathetic modulation did not change with position ($p > 0.05$). These responses were not influenced by sex (Supplemental Table 2).

Additionally, diastolic diameter decreases across positions. But, division of the diastolic diameter by the adjusted diastolic pressure at each position showed this vasoconstriction to be pressure dependent, as there was no difference in the diameter relative to pressure across positions ($p > 0.05$), and therefore not a result of the sympathetic nervous system activation.

Discussion

The main finding of the present study is that arterial stiffness increases with positional change, as observed with PWV and CAVI, and that using a height correction to account for changes in hydrostatic pressure is necessary to estimate β-stiffness in more upright positions. Additionally, the seated position creates a large enough orthostatic challenge to influence sympathetic activation in the periphery, but this activation did not affect the conduit arteries, and therefore, the gravitational height correction appears to be an adequate calibration of β-stiffness index at the common carotid artery.

Arterial Stiffness

The calibration of β-stiffness index using the unadjusted blood pressures of each position (Method 1), led to no differences in stiffness with the positional change. These results are incongruent with the increase in arterial stiffness measured using the gold standard PWV, and CAVI. The application of the height correction to calibrate the β-stiffness index measurements (Method 2) to account for the hydrostatic effects of gravity produced agreement between all arterial stiffness parameters, with each displaying an increase in stiffness with positional change.

Interestingly, the effect of body position on CAVI and PWV appears to be blood pressure independent. CAVI is theoretically a blood pressure independent measure [5], but PWV is typically thought of as a blood pressure dependent measure [25,26]. However, when PWV was divided by mean arterial pressure to account for the influence of blood pressure, the same relationship between positions remained. A previous study suggested a trend ($p=0.0987$) for increasing PWV between a supine and seated position, postulating this increase may be due to an increase in DBP [10]. DBP is correlated with PWV in young, healthy individuals [27], but this also did not change our positional effect. We speculate the increase in PWV and CAVI with positional change may be due to a decrease in the compliance of the arterial system because of the increased pressure from the hydrostatic effects of gravity, but this requires further exploration.

This study is not the first to incorporate a correction for the hydrostatic effects of gravity on vascular measures, as this methodology has been employed to make comparisons between supine and seated measures [28]. Gornik et al [28] utilized similar methodology to correct an ankle pressure (measured ankle pressure – hydrostatic pressure) in order to measure ankle-brachial index in the seated position instead of the gold-standard supine position. The correction of the ankle pressure allowed for accurate comparison of seated ABI to the gold-standard. Without the correction for hydrostatic pressure the results would lead to inaccurate screening and diagnosis of peripheral artery disease [28]. In our study, not correcting β-stiffness for hydrostatic pressure could lead to misleading conclusions, as arterial stiffness would be underestimated in a seated position. Furthermore, the carotid blood pressure change would be misinterpreted without correcting for changes in hydrostatic pressure.

The notion that arterial stiffness increases when moving from a supine to a more seated position has clinical importance as most people do not spend a majority of their day in the supine position. Additionally, as seen in the variability of the data in the figures, not all people

respond to the same extent or in the same direction. Those who have a more dramatic increase in stiffness with the positional change may be at increased health risk as it has previously been shown that increased stiffness is associated with cardiovascular events and all-cause mortality [1,29]. Assessing arterial stiffness strictly in the supine position may not be providing clinicians with a realistic picture, and it may be more beneficial to assess arterial stiffness in the seated position, similar to current blood pressure practices. Furthermore, in clinical research, it is not uncommon to allow patients to be in some degree of semi-recumbent instead of supine position for comfort. Our data indicate that body position affects all three measures of arterial stiffness used in this study, thus careful attention to body position is imperative.

Autonomic Modulation

In order to determine if the sympathetic nervous system stimulation with positional change was also influencing the conduit arteries and arterial stiffness parameters in this study, Method 3 for β-stiffness index was implemented. This theoretical model is attempting to decipher if the arterial stiffness alterations are due to physiological changes, such as sympathetic nervous system activation; the influence of gravitational hydrostatic pressure alone; or potentially both. Presumably, if the change in arterial stiffness in an upright position were only due to the hydrostatic pressure, then the theoretical model (Method 3) would yield similar values for arterial stiffness as Method 2. If Method 3 would have resulted in a lower stiffness than obtained by Method 2, a physiological adjustment could have been suggested, such as sympathetic nervous system activation. Comparison of Method 2 and Method 3 of β-stiffness index calibration resulted in no difference between methods, presumably indicating that the only effect on β-stiffness index with change in position is the hydrostatic effect of gravity, and not sympathetic activation affecting the vasculature at the level of the carotid artery.

With the increase in LF_{SAP} and LF/HF ratio, the current study appears to have induced a large enough orthostatic challenge to produce a sympathetic response which has also been shown by various degrees of head up tilt or lower body negative pressure (LBNP). The vasoconstriction in the periphery is suggested to begin at -20 mmHg of LBNP, whereas a 90 degree head up tilt is comparable to a -45 to -50 mmHg LBNP in terms of blood translocation [15]. A 60 degree head up tilt has shown to lead to significant increases in muscle sympathetic nerve activity (MSNA) and baroreceptor sensitivity compared to supine [18,20,30]. Cooke et al. [18] found that it took a head up tilt of at least 40 degrees to significantly increase the absolute number of sympathetic bursts measured by MSNA, with normalization and total MSNA significantly increasing at 60 degrees and greater compared with supine. Although the tilt in our study does not involve the lower extremity, the increases in heart rate and brachial blood pressure with the change in position support the increase in sympathetic activity in the periphery. Additionally, the other markers of autonomic function provided with the blood pressure and heart rate variability analysis indicate a reduction in parasympathetic modulation and a decrease in baroreceptor sensitivity. With the change in position we would expect unloading of the baroreceptors due to blood translocation from the effects of gravity, and therefore the decrease in baroreceptor sensitivity. But, we do not anticipate large enough changes in central blood volume were induced to elicit dramatic hormonal responses, such as renin or atrial natriuretic peptide, due to the legs remaining in a neutral position and only the upper body moved into different positions. Further, a full head up tilt is not warranted in clinical research unless explaining this unique physiologic response. Therefore, this study examined a more translational approach using positions seen in a majority of research and clinical settings.

Additionally, the reduction in diastolic diameter of the carotid artery also indicates vasoconstriction, potentially due to the sympathetic activation. Alternatively, once the

diameter was adjusted for the height corrected diastolic pressure, there was no longer a change in diameter across positions. This indicates the vasoconstriction was purely pressure dependent, attributable to the effects of gravity and the distending pressure, as previously suggested [31].

The question arises as to why there is an increase in arterial stiffness during orthostatic stress. Although a definitive answer cannot be determined from our results, one could postulate this is a physiological response in an attempt to prevent orthostatic hypotension and maintain blood flow to the brain. Research evaluating cerebral blood flow during orthostatic stress using a HUT indicated differential regulation of blood flow between the internal carotid artery and vertebral artery [32]. Cerebral blood flow was better maintained during HUT to the vertebral artery than the internal carotid artery, which may be related to the metabolism of the area and importance of the parts of the brain supplied by branches from the vertebral artery: the medulla oblongata, which is responsible for cardiac, vasomotor, and respiratory control [32]. If hypoperfusion of the medulla oblongata was to occur, the aforementioned centers may be inhibited, which may lead to the hemodynamic changes that take place before syncope and orthostatic hypotension. Therefore, the diameter reduction and increased stiffness noted in our results may be an attempt to maintain blood flow to the metabolically active areas of the brain to avoid hypoperfusion and symptoms of syncope.

Limitations

The data presented contains large variability in some measurements. This should lead to cautious interpretation of the data as not all individuals may respond in the same direction, as seen in the individually plotted data in Figures 3 and 4. All participants in this study were young and healthy individuals. Varying ages and disease status may attenuate the responses seen to positional changes and therefore the results may not be generalizable to other

populations. Additionally, data were not collected to account for diet or varying fitness levels, although most participants were at least recreationally active. Not all participants were measured during the same time of day, but, because this study was designed to investigate the response to change in position and not the absolute values of data points, this should not have greatly influenced the results.

Additionally, the influence of female sex hormones on hemodynamic parameters was not controlled, but a previous study has shown no significant differences in arterial wall properties of the common carotid artery during the menstrual cycle [33]. Due to the potential influence of sex hormones, the data was analyzed separately in each sex. In the original analyses, no sex*position interactions were evident, indicating sex did not influence the response between positions. Furthermore, the only major difference in the sex analyses was a lack of positional effect on height corrected β-stiffness index (Method 2) in females. However, the data appear to increase as seen in the overall dataset, and we feel the study is not powered with enough subjects to detect the difference in females, as this was not a primary aim of the study. Last, our measure of autonomic function was indirect and therefore a direct measure would provide more reliable data. Larger studies comparing arterial stiffness parameters and autonomic function during supine, head up tilt and seated positions are warranted to further elucidate stiffness responses.

Conclusion

In conclusion, arterial stiffness increases with a change in position. For the carotid artery, this increase appears to be dependent on hydrostatic pressure and not the involvement of the sympathetic nervous system. Therefore, when assessing arterial stiffness using β-stiffness index in a seated position, the measurement should be calibrated to a carotid blood pressure that takes into account the hydrostatic effects of gravity on the fluid column to allow

comparison between a supine and seated measurement. However, the increase in arterial stiffness with a more upright position measured by PWV or CAVI appear to be blood pressure independent.

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Table 1. Participant characteristics

	All	Males	Females
n	30	15	15
Age, yr	24 ± 4	24 ± 3	23 ± 4
Height, cm	170.4 ± 9.6	175.6 ± 8.4	165.2 ± 7.6*
Weight, kg	74.6 ± 15.2	82.3 ± 15.6	66.9 ± 9.8*
BMI, kg/m^2	25.6 ± 3.9	26.6 ± 4.2	24.5 ± 3.2

Data is reported as mean ± SD; *significantly different from males ($p<0.05$)

Table 2. Effects of positional change on hemodynamic and vascular variables.

	0°	45°	72°
Heart rate, bpm	58 (9)[a]	61 (11)	62 (11)
Brachial SBP, mmHg	122 (10)[b]	124 (11)	125 (10)
Brachial DBP, mmHg	74 (7)[a]	78 (7)	78 (6)
Brachial MAP, mmHg	91 (8)[a]	95 (9)	96 (6)
CAVI	5.6 (0.9)[a]	6.8 (0.8)[b]	7.4 (0.8)
PWV^{+}, m/s	4.7 (1.0)[a]	6.1 (0.7)[b]	6.4 (0.8)
PWV/MAP^{+}, $m/s*mmHg^{-1}$	0.052 (0.011)[a]	0.064 (0.006)[b]	0.067 (0.007)
PWV/HR^{+}, $m/s*bpm^{-1}$	0.08 (0.02)[a]	0.20 (0.02)	0.11 (0.02)
Height Correction, mmHg	0 (0)[a]	-11 (3)[b]	-16 (3)
Carotid SBP, mmHg (Method 1)	116 (13)[a]	122 (15)	122 (12)
Carotid DBP, mmHg (Method 1)	74 (8)[a]	78 (7)	78 (6)
Carotid SBP height corrected, mmHg (Method 2)	116 (13)[a]	110 (15)[b]	105 (12)
Carotid DBP height corrected, mmHg (Method 2)	74 (8)[a]	66 (7)[b]	61 (6)
Carotid SBP theoretical. mmHg (Method 3)	116 (13)[a]	105 (13)[b]	100 (13)
Carotid DBP theoretical, mmHg (Method 3)	74 (8)[a]	63 (8)[b]	58 (8)
β-stiffness (Method 1)	4.1 (1.3)	4.0 (1.5)	4.0 (1.2)
β-stiffness height corrected (Method 2)	4.1 (1.3)[b]	4.6 (1.7)	4.8 (1.5)
β-stiffness theoretical (Method 3)	4.1 (1.3)[a]	4.7 (1.6)[b]	5.0 (1.7)

Diastolic diameter, mm	6.1 (0.8)[a]	5.7 (0.7)[b]	5.6 (0.7)
Diastolic diameter/Carotid DBP height corrected, mm/mmHg	0.08 (0.02)	0.09 (0.01)	0.09 (0.01)
Relative diameter, %	11.7 (3.3)	11.7 (3.1)	12.2 (3.7)
LF_{SAP}, a.u.	6.4 (4.7)[a]	11.6 (6.6)	13.1 (10.1)

[+]n = 29

[a]: significantly different from all other positions, $p<0.05$

[b]: significantly different from 72°, $p<0.05$

Data presented as mean (SD). SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial pressure; CAVI: cardio-vascular ankle index; PWV: pulse wave velocity; a.u. = arbitrary unit

Table 3. Effects of positional change on autonomic function.

	0°	45°	72°
AlphaLF, ms/mmHg^2	16.6 (8.8)[a]	12.9 (5.2)	12.1 (6.2)
RRI BRS	20.2 (8.7)[a]	14.4 (5.1)	12.4 (5.5)
RMSSD, ms	70 (36)[a]	58 (35)	49 (33)
LF_{RRI}, ms^2	2081 (2930)	2081 (2207)	2053 (2946)
HF_{RRI}, ms^2	1285 (1516)[b]	983 (1257)	723 (1163)
LF/HF ratio	2.2 (1.8)[a]	3.5 (3.4)	5.6 (8.6)
LF_{SAP}, mmHg^2	6.4 (4.7)[a]	11.6 (6.6)	13.1 (10.1)

[a]: significantly different from all other positions, $p<0.05$

[b]: significantly different from 72°, $p<0.05$

Data presented as mean (SD). RRI: R-R interval; BRS: baroreceptor sensitivity; RMSSD: root mean square of successive differences; LF: low frequency; HF: high frequency; SAP: systolic arterial pressure

Figure 1. Height correction in 45° (1) and 72° (2) body positions.

Figure 2: (A) Carotid systolic pressures in three different positions estimated by the height corrected brachial pressures of that position [Method 2] (B) Carotid diastolic pressures in three different positions estimated by the height corrected brachial pressures of that position [Method 2]; *: significantly different from all other positions, $p < 0.05$, †: significantly different from 72°

Figure 3: (A) PWV in three different positions. (B) β-stiffness index in three different positions calculated with the height corrected carotid pressures of that position [Method 2]; *: significantly different from all other positions, $p < 0.05$, †: significantly different from 72°

Figure 4: β-stiffness index in three different positions calculated with: (1) the carotid pressures of that position [Method 1], (2) the height corrected carotid pressures of that position [Method 2], (3) the height corrected carotid pressures of the supine position [Method 3]