Impact of Blood Pressure Perturbations on Arterial Stiffness

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Running head: Arterial stiffness, blood pressure, and vascular reactivity

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Abstract

Although the associations between chronic levels of arterial stiffness and blood pressure (BP) have been fairly well studied, it is not clear if and how much arterial stiffness is influenced by acute perturbations in BP. The primary aim of this study was to determine magnitudes of BP-dependence of various measures of arterial stiffness during acute BP perturbation maneuvers. Fifty apparently healthy subjects, including 25 young (20-40 years) and 25 older adults (60-80 years), were studied. A variety of BP perturbations, including head-up tilt, head-down tilt, mental stress, isometric handgrip exercise, and cold pressor test, were used in order to encompass BP changes induced by physical, mental, and/or physiological stimuli. When each index of arterial stiffness was plotted with mean BP, all arterial stiffness indices, including cardio-ankle vascular index or CAVI \( r=0.50 \), carotid-femoral pulse wave velocity or cfPWV \( r=0.51 \), brachial-ankle pulse wave velocity or baPWV \( r=0.61 \), arterial compliance \( r=-0.42 \), elastic modulus \( r=0.52 \), arterial distensibility \( r=-0.32 \), \( \beta \)-stiffness index \( r=0.19 \), and Young’s modulus \( r=0.35 \) were related to mean BP (all \( P<0.01 \)). Changes in CAVI, cfPWV, baPWV, and elastic modulus were significantly associated with changes in mean BP in the pooled conditions while changes in arterial compliance, arterial distensibility, \( \beta \)-stiffness index, and Young’s modulus were not. In conclusion, this study demonstrated that BP changes in response to various forms of pressor stimuli were associated with the corresponding changes in arterial stiffness indices and that the strengths of associations with BP varied widely depending on what arterial stiffness indices were examined.

Key words: pressor response, pulse wave velocity, arterial compliance
Introduction

Arterial stiffness, particularly aortic pulse wave velocity, plays an important role in the pathogenesis of cardiovascular disease (CVD) and is an important independent risk factor for CVD (13, 17). Since changes in arterial stiffness can be detected before the appearance of clinically apparent and overt vascular disease, arterial stiffness can be used as an early marker for subclinical vascular dysfunction (11). There are various techniques used to measure arterial stiffness (16), with each technique measuring a different aspect of arterial stiffness. Thus, these techniques may be affected by behavioral, physiological, and extraneous factors to a different degree. Among these factors, dependence of arterial stiffness on blood pressure (BP) has been a topic of much discussion and debate among investigators working in this field. Indeed a recent scientific statement from the American Heart Association task force recommended that blood pressure should be recorded at the time of an arterial stiffness measurement and taken into consideration (27).

Arterial stiffness has been implicated as the primary cause of age-related elevation in arterial BP and described as a physiological mechanism underlying hypotensive effects of lifestyle interventions and antihypertensive medications (8, 15). On the other hand, some have questioned the utility of arterial stiffness above and beyond the traditional brachial BP measurement as changes in arterial stiffness are often accompanied by the corresponding changes in BP (10). Most of the available literature focusing on BP dependency on arterial stiffness deal with basal levels of BP or chronic changes with pharmacological or non-pharmacological interventions (19, 20,
Currently, it is not clear what extent acute changes in BP are accompanied by corresponding changes in arterial stiffness.

With this information as background, the primary aim of the present study was to determine the effect of acute BP perturbation on various measures of arterial stiffness. There are a number of BP perturbation maneuvers that have been utilized in research settings (18, 25), and these maneuvers use different forms of stress (psychological, physical, and mechanical) to elicit pressor responses. In order to comprehensively address the stated aim, we used five different BP perturbations. Additionally, we hypothesized the magnitude of change in arterial stiffness during BP perturbations might be influenced by age (2), young (20-40 yrs) and older (60-80 yrs) adults were studied. The working hypotheses were that all measures of arterial stiffness would be dependent on blood pressure and that the magnitude of changes in arterial stiffness parameters induced by acute changes in BP would be variable among various measures of arterial stiffness and widely differed age groups.

**Methods**

**Subjects.** A total of 50 subjects were studied. Half of the subjects were young (20-40 yrs) and the other half older (60-80 yrs). Subjects were recruited from the city of Austin and the surrounding community using flyers and e-mails to various organizations and information sharing. Subjects with overt heart disease, diabetes, or other cardiovascular problems, as assessed by medical history questionnaire, were excluded from study participation. Additional exclusion criteria included pregnancy, chronic smoking, a recent illness or surgery, or any medical intervention in the 12 hours before any of the study sessions. Subjects were instructed to abstain from strenuous
exercise for 12 hours, caffeine consumption for 6 hours, and food for 4 hours prior to experimental trials. The Institutional Review Board at University of Texas at Austin approved this study. Written informed consent was obtained from all subjects.

**Experimental design.** The measurements were performed on two separate visits lasting ~2 hours under comfortable laboratory conditions. The two testing sessions were scheduled at the same time of the day to avoid any potential diurnal effects. Complexity of the experiments necessitated the two separate testing sessions in order to accommodate all the BP perturbation tests and the testing apparatus. In the first testing session, head up tilt, mental stress, and cold pressor test were performed. In the second testing session, head down tilt, isometric handgrip, and cold pressor test were conducted. Each test was separated by at least 15 minutes. Cold pressor test was performed on both sessions to accommodate all the arterial stiffness tests (i.e., to avoid the measurement of arterial pressure and waveform on the limb that the cold stress was applied). Selection of right and left sides between the days was randomized. Cold pressor test was always conducted as the final perturbation for each testing visit to eliminate the long-lasting residual effects that could influence other BP perturbation tests. Isometric handgrip test was performed on both sides of the body during the second testing visit, and the order of sides being tested was randomized. Importantly, there were no significant differences in pressor responses between the two testing sessions for either isometric handgrip or cold pressor test demonstrating the stability of the pressor responses.

**Head-up tilt and head-down tilt.** Head-up and head-down tilt was conducted as previously described (25). Subjects were in a supine position on a tilt table with two
Velcro straps (DFL, Velcro Inc., Manchester, NH) placed over the chest and thighs and tilted up to 30 degrees. For head-down tilt, the subjects laid in a supine position on a tilt table, tilted down to 10 degrees, and instructed to remain still. The straps prevented subjects from sliding down the table, and ensured subjects were not bearing weight during the testing period; this prevented muscular contraction of the lower body, which could affect hemodynamics and pressor responses (i.e., skeletal muscle pump).

Mental stress test. The Stroop Test (22) is a cognitive task inducing psychological stress that can be performed in a laboratory setting. A slide show contained randomly-colored words of color names and was programmed to proceed at a rate of one slide per second to produce a sufficient psychological stress. The entire slide show was composed of two types of slides: (a) congruent color-word slide: equivalent printed color and words of a color (i.e. the word “red” appeared in red font on the slides); (b) incongruent color-word slide: inequivalent printed color and words of a color (i.e. the word “red” appeared in green font on the slide). To increase the stressfulness of the test, subjects were instructed to comply with the speed of the slide show (one second per slide) and to respond according to the slide type presented. Subjects were provided with a trigger as a method of response in each hand and instructed to respond in their right hands whenever a congruent color-word slide was presented and with the left hand whenever an incongruent color-word slide was presented. The Stroop test proceeded for two minutes while measurements were obtained. The number of correct answers was not quantified, but the subject was not aware of this during the test.

Isometric handgrip exercise. Subjects performed two minutes isometric
handgrip exercise (HDM-915; Lode Instruments, Groningen, The Netherlands) at 40% of maximal voluntary contraction (MVC). During the test, the force exerted by subjects was shown on a visual display, and the subjects were instructed to hold 40% of their MVC throughout the testing period. Subjects were asked to breathe normally, avoid a Valsalva maneuver (3), and contract only the flexors of the hand in order to avoid recruiting additional musculature of the upper arm and shoulder.

Cold pressor test. Subjects submerged their foot in ice water (2-5 °C) for two minutes (4). The foot was selected, rather than the hand, in order to stimulate maximum hemodynamic and sympathetic responses (18). Subjects were instructed to breathe normally, avoid a Valsalva maneuver (3), and maintain relaxation.

Measurements. The following arterial stiffness indices were measured in the present study.

Cardio-ankle vascular index (CAVI). Unilateral brachial BP, ankle BP, and heart rate were measured simultaneously by using oscillometric pressure sensor cuffs, electrocardiograms, and phonocardiograms (VaSera VS-1000, Fukuda Denshi, Tokyo, Japan). CAVI was calculated by incorporating the stiffness parameter β, which is known to be a BP-independent measure of arterial stiffness, into an equation (23). The equation to calculate CAVI was:

\[ CAVI = a\left[\frac{2\rho}{\Delta\text{Pressure}}\right] \times \text{ln}\left(\frac{\text{systolic BP}}{\text{diastolic BP}}\right)\text{PWV}^2 + b \]

where \( a \) and \( b \) = scale conversion constants to match CAVI values with aortic PWV, \( \rho \) = blood density, and \( \text{PWV} \) = pulse wave velocity from aortic valve to ankle. CAVI is a relatively new measure, and its reliability has not been well established. Accordingly, the measurements of CAVI were taken three times each by
two different investigators at the beginning of each testing day to obtain inter- and intra-
observer reliability.

**Pulse wave velocity (PWV).** Both carotid-femoral pulse wave velocity (cfPWV) and brachial-ankle pulse wave velocity (baPWV) were simultaneously measured by the vascular testing device (VP-2000, Omron Healthcare, Kyoto, Japan). Arterial applanation tonometry sensors incorporating an array of 12 micropiezoresistive transducers were placed on the carotid and femoral arteries to acquire pulse pressure waves (1). In addition, oscillometric pressure sensor cuffs were placed unilaterally on arm and ankle, electrocardiogram sensors were attached to both wrists, and a phonocardiogram was placed above the sternum. baPWV was obtained from the oscillometric pressure sensor cuffs. cfPWV was obtained from distance divided by the time delay of the pulse wave. Distance traveled by pulse waves for cfPWV was determined by the measurement of body surface distance from the site of transducer placement on the carotid and femoral arteries with a segmometer specifically constructed for PWV studies. For baPWV, pulse wave travel distance was calculated automatically based on one’s height.

**Carotid artery compliance.** The simultaneous measurement of ultrasound imaging of the common carotid artery and applanation tonometrically-obtained arterial pressure waveforms from the contralateral carotid artery allows noninvasive determination of arterial compliance (26). Common carotid artery diameter was measured from the images derived from an ultrasound machine equipped with a high-resolution linear array transducer (Philips iE33 Ultrasound System, Bothel, WA). Carotid artery diameter was analyzed using image analysis software (Brachial Analyzer,
Medical Imaging Applications, Coralville, IA), and pressure waveforms of the carotid artery were analyzed using a waveform analysis software (WinDq 2000, Dataq Instruments, Akron, OH). Carotid blood pressure signal obtained by applanation tonometer was calibrated by equating the carotid mean and diastolic blood pressure to the brachial artery value because the baseline levels of carotid blood pressure are subjected to hold-down force. Arterial compliance, elastic modulus, arterial distensibility, β-stiffness index, and Young’s modulus were subsequently calculated using the following formulas.

- **Arterial compliance** = $\frac{\Delta \text{Diameter}}{\Delta \text{Pressure}}$
- **Arterial distensibility** = $\frac{\Delta \text{Diameter}}{(\Delta \text{Pressure} \times \text{Diameter at end-diastole})}$
- **Elastic modulus** = $\frac{(\Delta \text{Pressure} \times \text{Diameter at end-diastole})}{\Delta \text{Diameter}}$
- **Young's modulus** = $\frac{(\Delta \text{Pressure} \times \text{Diameter at end-diastole})}{\Delta \text{Diameter} \times \text{IMT}}$
- **β-stiffness index** = $\ln\left(\frac{\Delta \text{Pressure}}{\Delta \text{Diameter}}\right) = \ln((\Delta \text{Pressure}) / [(\Delta \text{Diameter}) / \text{Diameter at end-diastole}])$

**Statistical analyses.** The intraclass correlation coefficients as well as coefficients of variation were obtained to determine inter- and intra-observer reliability of the CAVI. Analyses of variance (ANOVA) were used to evaluate the group differences. If a significant F-value was detected, a post-hoc test using LSD was performed to identify the significant group differences. Correlation and regression analyses were performed to determine the relations between changes in arterial stiffness indices and mean BP induced by various vascular reactivity tests. In order to account for the influence of heart rate, partial correlational analyses were also conducted. All data were presented as means±SEM. Statistical significance was set *a priori* at p<0.05.
SPSS statistics software version 22 (IBM, Chicago, IL) was used for all statistical analyses.

Results

Table 1 presents selected subjects’ physical characteristics. A total of 50 subjects, including 25 young and 25 older adults, were studied. There were no significant differences in height, body mass, and body mass index between young and older individuals.

Table 2 illustrates the reliability of CAVI measurements. Coefficients of variation of intra-observer and inter-observer reliability were 3.4 and 2.4%. The intra- and inter-observer reliability test showed that intraclass correlation coefficients were 0.99 and 0.98.

Figure 1 shows changes in mean BP to various BP perturbation tests. As shown in Table 3, changes in CAVI, cfPWV, baPWV, and elastic modulus were significantly associated with changes in mean BP in the pooled conditions. Changes in arterial compliance, arterial distensibility, β-stiffness index, and Young’s modulus were not significantly related to changes in mean BP. When each index of arterial stiffness was plotted with mean blood pressure, all the arterial stiffness indices, including CAVI (r=0.50), cfPWV (r=0.51), baPWV (r=0.61), arterial compliance (r=-0.42), elastic modulus (r=0.52), arterial distensibility (r=-0.32), β-stiffness index (r=0.19), and Young’s modulus (r=0.35) were related to mean BP (all P<0.01) (Figure 2). Because heart rate is known to modulate parameters of arterial stiffness, we performed partial correlational analyses after partialing out the changes in heart rate. In general, many of the correlations between arterial stiffness measures and blood pressure remained
statistically significant (Table 4).

In order to determine if the associations between blood pressure and arterial stiffness indices are modulated by age and sex, we stratified the data by age and sex and presented in Table 5. In general, the strengths of associations were generally greater in males than in females. For CAVI, cfPWV, and baPWV, young subjects had stronger association with changes in mean BP than in older subjects.

Discussion

The most salient finding of the present study is that all of the arterial stiffness indices examined had some degree of BP dependency to various forms of pressor tasks. This was true for indices of arterial stiffness that are thought to be BP independent; β-stiffness index and CAVI.

One of the strengths of the present study is the use of various types of pressor tests employed. These maneuvers use different forms of stress to elicit BP responses. For instance, mental stress is psychological, cold pressor test is physical, and isometric handgrip is mechanical. A total of 5 different BP maneuvers were utilized in order to comprehensively evaluate the effects of acute changes in BP on arterial stiffness. Each test produced different magnitudes of pressor responses, with the most profound increases in BP observed during the cold pressor test. In conjunction with the BP changes, we also observed corresponding changes in most of the arterial stiffness indices during each maneuver.

When various levels of BP induced by pressor tests were plotted against arterial stiffness, all of the arterial stiffness indices examined in the present study demonstrated significant associations with BP. The strongest association was obtained with baPWV.
(r=0.61) and the lowest with β-stiffness index (r=0.19). When the data were analyzed by plotting changes in BP and the corresponding changes in arterial stiffness, CAVI, cfPWV, baPWV, and elastic modulus showed significant associations with changes in BP in the pooled population whereas no such relations were observed for arterial compliance, arterial distensibility, β-stiffness index, and Young’s modulus. These results suggest that some measures of arterial stiffness are more affected by acute changes in BP than others and that the magnitude of BP-dependency varies widely among various measures of arterial stiffness.

CAVI has been proposed as an arterial stiffness indicator that is independent of BP (20). The original study from Japan has reported no association between CAVI and BP (r=0.01-18) whereas baPWV was significantly associated with BP (r=0.34-0.46) (20). Since then a number of investigators have confirmed the observation that CAVI was less dependent on BP compared with other arterial stiffness indices (7, 9, 24). CAVI was originally developed by incorporating β-stiffness index, which is thought to be a BP independent measurement of arterial stiffness (24). Indeed in a recent study, the incorporation of β-stiffness index substantially reduced the BP dependency on PWV (29). In marked contrast to these previous studies, the present study demonstrated that CAVI was dependent on mean BP whether it is expressed as absolute levels of BP or changes in BP. It should be noted that the previous studies utilized chronic or basal BP whereas the present study used BP that was changed with acute BP perturbations.

As expected, lowest associations with BP were observed in β-stiffness index though its BP dependency was evident during several pressor tests. The original assumption involved in β-stiffness index is a simple exponential relation between the
intraluminal pressure and the distension of arteries (6). However, the exponential
relation was constructed in a limited BP range obtained in a healthy normal population,
and the validity has been questioned as to whether it is applicable to other populations
(12). Indeed highly significant associations between BP and β-stiffness index have
previously been reported in a variety of patient populations (12). Interestingly, blood
perturbations affected CAVI and β-stiffness index differently even though CAVI was
calculated by incorporating the stiffness parameter β into an equation. However, CAVI
differs from β-stiffness index in a number of ways. For example, beta-stiffness index is
a measure of local arterial stiffness determined from the pulsatile changes in arterial
diameter and blood pressure. On the other hand, CAVI is a measure of segmental
arterial stiffness that is derived from pulse wave velocity. While β-stiffness index is an
index of central arterial stiffness, CAVI reflects a combination of central and peripheral
artery stiffness.

Prevalence of hypertension increases markedly with advancing age in both men
and women (5). Men are generally at greater risk for developing hypertension than
age-matched women (5). Accordingly, we stratified the data for age (young and older)
and sex (men and women) and performed separate statistical analyses. In general,
the associations between changes in arterial stiffness and BP appear larger for men
than for women and for young than for older in many of the arterial stiffness indices.
These results are consistent with previous studies reporting that BP reactivity is
modulated by both age and sex (21, 28). Although the exact reasons are not clear, it
may be related to a “ceiling effect” due to higher arterial stiffness values at baseline.

The simplest premise of the present study is that any changes in arterial
stiffness would be attributed to changes in BP induced by pressor tests. But these changes in arterial stiffness and BP could be independent. For instance, sympathoexcitatory stimuli induced by BP reactivity tests can act on the smooth muscle cells surrounding the large elastic arteries and stiffen arteries (14). The same vasoconstrictor tone can stimulate smooth muscle cells on the peripheral muscular arteries and increase vascular resistance and increase mean BP via the Ohm’s law (14). Thus, it should be noted that changes in arterial stiffness may not be epiphenomenon of BP changes induced by various pressor tests.

There were several limitations that should be taken into consideration. First, because only apparently healthy subjects were included in the present study, the results may not be extrapolated to patient populations. Second, vascular reactivity tests were neither measured simultaneously nor randomized in this study. The present protocol provided substantial procedural challenges in placing all the measurement devices on a given subject. In spite of a number of pilot studies conducted, it was not possible to accommodate everything in one testing session. Additionally, some vascular reactivity tests such as cold pressor test induces a prolonged BP changes and had to be placed at the end of the testing session. However, in an attempt to establish the reliability of pressor responses, we performed isometric handgrip or cold pressor test twice in different testing sessions and found that there were no significant differences in pressor responses between the two testing sessions. Furthermore, although vascular reactivity tests were not all randomized in this study, each two sessions of isometric handgrip and cold pressor test was randomized that eliminates the order effects.

In summary, the results of the present study indicate that BP changes in
response to various forms of pressor stimuli were associated with the corresponding changes in arterial stiffness indices examined and that the strengths of associations varied widely depending on what arterial stiffness indices were examined.

Acknowledgements

This study was supported by Fukuda Denshi.
References


Figure Legends

Figure 1. Changes in mean blood pressure (BP) during vascular reactivity tests

Data are △mean ± SEM. HUT=head-up tilt, HDT=head-down tilt, MS=mental stress, IHG=isometric hand grip, CPT=cold pressor test. *P<0.05 vs. HUT, †P<0.05 vs. HDT, ‡P<0.05 vs. MS.

Figure 2. Relationship between arterial stiffness indices and mean blood pressure (BP).

CAVI=cardio-ankle vascular index, cfPWV=carotid-femoral pulse wave velocity, baPWV=brachial-ankle pulse wave velocity.
Table 1. Selected subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total (n = 50)</th>
<th>Young (n = 25)</th>
<th>Older (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>45.4 ± 2.7</td>
<td>27.1 ± 0.6</td>
<td>63.8 ± 0.9*</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>29 / 21</td>
<td>17 / 8</td>
<td>12 / 13</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>172 ± 1</td>
<td>174 ± 2</td>
<td>170 ± 2</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>75.9 ± 2.3</td>
<td>78.6 ± 3.4</td>
<td>73.3 ± 3</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>25.6 ± 0.7</td>
<td>25.7 ± 1.0</td>
<td>25.4 ± 1.0</td>
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<tr>
<td>Heart rate (bpm)</td>
<td>57±1</td>
<td>57±2</td>
<td>57±2</td>
</tr>
<tr>
<td>Systolic BP (mmHg)</td>
<td>124±2</td>
<td>117±2</td>
<td>129±3</td>
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<tr>
<td>Diastolic BP (mmHg)</td>
<td>75±1</td>
<td>71±1</td>
<td>78±2</td>
</tr>
<tr>
<td>Mean BP (mmHg)</td>
<td>91±1</td>
<td>86±1</td>
<td>95±2</td>
</tr>
</tbody>
</table>

Data are mean±SEM. BP=blood pressure. *P<0.01 vs. Young.
Table 2. Intra- and inter-observer reliability tests of cardio-ankle vascular index (CAVI)

<table>
<thead>
<tr>
<th></th>
<th>CV</th>
<th>ICC</th>
<th>95% Confidence Interval</th>
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</tr>
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<tbody>
<tr>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Minimum</td>
<td>Maximum</td>
</tr>
<tr>
<td>Intra-observer reliability</td>
<td>3.4 %</td>
<td>0.99*</td>
<td>0.98</td>
<td>0.99</td>
</tr>
<tr>
<td>Inter-observer reliability</td>
<td>2.4 %</td>
<td>0.98*</td>
<td>0.98</td>
<td>0.99</td>
</tr>
</tbody>
</table>

CV=coefficient of variation, ICC=intraclass correlation coefficient. *P<0.05
Table 3. Relations between changes in arterial stiffness and mean blood pressure during vascular reactivity tests

<table>
<thead>
<tr>
<th></th>
<th>∆ Mean Blood Pressure (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Head-Up Tilt</td>
</tr>
<tr>
<td>∆ CAVI</td>
<td>NS</td>
</tr>
<tr>
<td>∆ cfPWV</td>
<td>NS</td>
</tr>
<tr>
<td>∆ baPWV</td>
<td>.32*</td>
</tr>
<tr>
<td>∆ Arterial compliance</td>
<td>NS</td>
</tr>
<tr>
<td>∆ Elastic modulus</td>
<td>NS</td>
</tr>
<tr>
<td>∆ Arterial distensibility</td>
<td>NS</td>
</tr>
<tr>
<td>∆ β-stiffness index</td>
<td>.32*</td>
</tr>
<tr>
<td>∆ Young’s modulus</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data are Pearson correlation coefficients. *P<0.05. CAVI=cardio-ankle vascular index; cfPWV=carotid-femoral pulse wave velocity; baPWV=brachial-ankle pulse wave velocity
Table 4. Relations between changes in arterial stiffness and mean blood pressure during vascular reactivity tests when associations were adjusted for changes in heart rate using partial correlational analyses

<table>
<thead>
<tr>
<th>Head-Up Tilt</th>
<th>Head-Down Tilt</th>
<th>Mental Stress</th>
<th>Isometric Handgrip</th>
<th>Cold Pressor Test</th>
<th>POOLED</th>
</tr>
</thead>
<tbody>
<tr>
<td>∆ CAVI</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>.55*</td>
<td>NS</td>
</tr>
<tr>
<td>∆ cfPWV</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>.35*</td>
<td>.35*</td>
</tr>
<tr>
<td>∆ baPWV</td>
<td>NS</td>
<td>NS</td>
<td>.63*</td>
<td>.37*</td>
<td>.62*</td>
</tr>
<tr>
<td>∆ Arterial compliance</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>∆ Elastic modulus</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
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<td>∆ Arterial distensibility</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>∆ β-stiffness index</td>
<td>.35*</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
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<tr>
<td>∆ Young’s modulus</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
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</table>

Data are partial correlation coefficients. *P<0.05. CAVI=cardio-ankle vascular index; cfPWV=carotid-femoral pulse wave velocity; baPWV=brachial-ankle pulse wave velocity.
Table 5. Associations between changes in arterial stiffness indices and mean blood pressure (BP) stratified by age and sex

<table>
<thead>
<tr>
<th></th>
<th>∆ Mean BP (mmHg)</th>
<th>MALE</th>
<th>FEMALE</th>
<th>YOUNG (20-40 yrs)</th>
<th>OLDER (60-80 yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>∆ CAVI (AU)</td>
<td>.42*</td>
<td>.26*</td>
<td>.45*</td>
<td>.22*</td>
<td></td>
</tr>
<tr>
<td>∆ cfPWV (cm/sec)</td>
<td>.44*</td>
<td>.29*</td>
<td>.56*</td>
<td>.23*</td>
<td></td>
</tr>
<tr>
<td>∆ baPWV (cm/sec)</td>
<td>.44*</td>
<td>.26*</td>
<td>.45*</td>
<td>.31*</td>
<td></td>
</tr>
<tr>
<td>∆ Arterial compliance (cm/mmHg)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>∆ Elastic modulus (mmHg)</td>
<td>.30*</td>
<td>NS</td>
<td>NS</td>
<td>.27*</td>
<td></td>
</tr>
<tr>
<td>∆ Arterial distensibility (mmHg⁻¹)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>∆ β-stiffness index (AU)</td>
<td>NS</td>
<td>NS</td>
<td>-.28*</td>
<td>NS</td>
<td></td>
</tr>
<tr>
<td>∆ Young’s modulus (mmHg/cm)</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>.22*</td>
<td></td>
</tr>
</tbody>
</table>

Data are Pearson correlation coefficients. *P<0.05.

CAVI=cardio-ankle vascular index, cfPWV=carotid-femoral pulse wave velocity, baPWV=brachial-ankle pulse wave velocity
\( \Delta \text{Mean BP (mmHg)} \)

- HUT
- HDT
- MS
- IHG
- CPT

*† ‡