Carotid distensibility, baroreflex sensitivity, and orthostatic stress

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Carotid distensibility, baroreflex sensitivity, and orthostatic stress. J Appl Physiol 99: 64–70, 2005. First published February 24, 2005; doi:10.1152/japplphysiol.01248.2004.—In this study, we tested the hypothesis that carotid arteries undergo rapid changes in distensibility on moving from the supine to head-up tilt (HUT) postures and, subsequently, that this change in carotid distensibility (cDa) might be associated with concurrent reductions in cardiovascularg baroreflex sensitivity (BRS). Thus the effect of posture on carotid vascular mechanics and cardiovascularg BRS with consideration for altered central hemodynamics (i.e., stroke volume; Doppler ultrasound) was examined.

Carotid pulse pressure (cPP; Millar transducer) and contralateral B-mode ultrasound images were assessed at the carotid artery during supine and 60° HUT postures. From these measures, cDa was calculated at 5-mmHg pressure increments experienced during the cardiac cycle (n = 6). cPP (n = 9) was not different in the two postures. A smaller stroke volume being ejected into a smaller carotid artery in HUT explained the maintenance of cPP in HUT. Also, compared with supine, cDa was reset to a lower level in HUT (main effect of posture; P < 0.05). Cardiovascularg BRS (sequence method) was diminished in HUT vs. supine (P < 0.05). A positive correlation was observed between the tilt-induced changes in maximal cDa (in early systole) and cardiovascularg BRS (r² = 0.75; P < 0.05), but there was little predictive relationship between changes in cPP, systolic vessel dimensions, or average cDa and the corresponding change in BRS. The present results indicate that HUT elicits rapid changes in carotid artery mechanics and further suggest that reductions in the maximal cDa measured in early systole contribute to reduced cardiovascularg BRS with HUT.

Cardiovascular Baroreceptors are mechanosensitive afferents that respond to increases in vascular transmural pressure. Therefore, the discharge of these neurons will be affected by the distensibility of the blood vessel and its absolute diameter during changes in background (5) or pulsatile (6, 8) blood pressure. During orthostatic stress, baroreceptors exert important control over reflex-mediated changes in vagal and sympathetic outflow to regulate heart rate and vascular resistance and thereby maintain blood pressure. The ability to rapidly adjust these adaptive responses is related to baroreflex sensitivity (BRS). Cardiac BRS, primarily reflecting vagal output, is reduced during orthostasis (10, 11, 27). In this study, we address the question of whether the change in BRS with head-up tilt (HUT) is mechanistically determined by rapid changes in the mechanical transduction from vascular stretch to baroreflex function. In this scenario, a fall in carotid diameter or stretch on going from supine to HUT would diminish baroreceptor afferent volleys such that reflex excitation of vagal outflow would be reduced.

The premise of diminished carotid sinus stretch in the above hypothesis may be based on two possible scenarios. First, a fall in stroke volume (SV) on assuming the upright posture could lead to diminished carotid pulse pressure (cPP) and, subsequently, vascular stretch. Alternatively, reductions in vascular distensibility may be involved such that the stiffer vessel cannot stretch as far for a given cPP. The cPP response during HUT is debated. In situ studies of isolated carotid sinus preparations indicate that radial artery pulse pressure (PP) clearly affects carotid baroreceptor afferent discharge patterns (6, 8). In humans, proposed reductions in central PP during HUT (42) are concomitant with the fall in cardiovascularg BRS. These associations point to a direct role of PP and SV rather than carotid mechanics (i.e., distensibility) per se. A major concern with current conclusions regarding the role of changing PP and cardiovascularg BRS in humans is that these variables are generally [but not always (1)] based on measures of blood pressure made in the periphery and not in the carotid artery. Thus the sensitivity of pulse-wave amplification in peripheral arteries to changes in SV and vasomotor tone may not represent PP changes that occur in the central arteries. Consequently, recent assessment indicated that cPP was unchanged between the supine and HUT postures despite reductions peripherally (45). These data challenge the concept that PP is the requisite stimulus that drives carotid baroreflex control of autonomic function during orthostasis.

The second scenario relates to a stiffening of the central vasculature. Monahan and colleagues (33, 34) have reported changes in BRS with age and endurance training that were associated with altered carotid artery compliance. Detailed examinations have supported the idea that at least part of the reduction in BRS with aging is due to stiffening of the baroreceptive regions in the carotid sinus (20), with the remaining alterations due to central processing changes. Whether these processes determine BRS changes during postural adjustments is not known.

The main purpose of the present study was to investigate the effect of posture on carotid artery distensibility (cDa) using measures of cPP rather than peripheral PP. To our knowledge,
such an approach has not been reported. We tested the hypothesis that the mechanical properties of carotid arteries undergo rapid adaptations to changes in posture. Furthermore, we tested the subhypothesis that the reduced carotid BRS in HUT, compared with supine, would be associated with a concurrent reduction in cDa. It was reasoned that such a change in cDa could account for findings of higher sympathetic and diminished parasympathetic reflex gain during HUT (35). The results indicate that acute reductions in carotid diameter and distensibility occur with HUT. In addition, although absolute values of maximal cDa (observed in early systole) predict the absolute level of BRS, the tilt-induced change in cDa did not correlate with concurrent reductions in BRS.

METHODS

Subjects. Nine healthy, nonsmoking, normotensive individuals volunteered for the present study (3 women, 6 men). The participants were 24 \(\pm 9\) yr of age, 175 \(\pm 10\) cm in height, and 70 \(\pm 19\) kg in weight (means \(\pm\) SD). Based on a health questionnaire, all subjects were free of cardiovascular disease and were not taking medications contraindicating participation in the study. All subjects provided informed, written consent for the experimental protocol as approved by The University of Western Ontario Health Sciences Research Ethics Board.

Experimental design. Subjects reported to the laboratory after fasting and abstaining from caffeine and alcohol ingestion for a minimum of 12 h before experimentation. On arrival, subjects were administered a standard breakfast of juice and a meal replacement bar to normalize hydration and satiety status. Before instrumentation, subjects voided their bladder.

After a period of at least 20 min in the supine position, weight-bearing HUT at 60° was performed for at least 5 min. No subjects experienced symptoms or showed signs of presyncope.

Measurements. Heart rate was calculated from R-R intervals of the ECG. Blood pressure waveforms were assessed and recorded continuously using two distinct methods. A fixed, wrist arterial tonometer was positioned over the right radial artery and calibrated to brachial artery pressure using an associated automated sphygmomanometer situated on the right arm (Pilot 9200, Colin Instruments, San Antonio, TX). Also, a hand-held tonometer (SPT-301, Millar Instruments, Houston, TX) was used to manually obtain pressure waveforms from the left common carotid artery. The absolute baseline represented by the Millar tonometer waveform is subject to hold-down pressure. Therefore, these waveforms were used for assessment of cPP only, which is unrelated to the absolute pressure. Analog signals for SV and blood pressure (from both the Colin and Millar devices) were sampled at 100 Hz, whereas ECG was sampled at 1,000 Hz, and stored with an online data acquisition and analysis package (PowerLab, ADInstruments).

The ascending aortic peak velocity envelope was recorded from the ascending aorta using a 2-MHz Doppler ultrasound probe positioned in the suprasternal notch (CFMT50, GE/Vingmed, Horten, Norway). The integral of this signal over a cardiac cycle was taken as the SV.

Aortic root diameter was obtained using two-dimensional B-mode ultrasound imaging along the parasternal long axis of the aorta using a 3.5-MHz sector transducer (System 5, GE/Vingmed). Subsequently, cardiac output was calculated. Digital video clips of the right common carotid artery (2-dimensional B-mode echo Doppler images; 10-MHz linear array transducer; System 5, GE/Vingmed) were recorded during three separate cardiac cycles and stored online for later analysis. The independent image video clips were separated by at least 30 s. These video clips, collected at \(\sim 15\) frames/s, were stored on optical disc for subsequent analysis. Only images where the intimal layer could be visualized were used for analysis because they provided the best contrast for measurement (see below).

Data analysis. Systolic blood pressure (SBP), diastolic blood pressure, PP, and mean arterial pressure were obtained from the wrist pressure waveforms. Mean values of each of these variables were calculated for the last minute of data collection during supine and HUT postures corresponding to the period of carotid artery image collection. Kornet et al. (25) reported that the rate of change in pulsatile pressure during systole may impact BRS. In the present analysis, the maximal slope of the tonometric waveform was obtained from the heart beat before, during, and immediately after the cardiac cycle during which the carotid artery image was recorded. These three values were averaged for each subject and posture.

Arterial diameter data and cDa were analyzed in two approaches. The discrete carotid artery diameters at diastole and systole were assessed in each image using manual digital calipers (EchoPAC software version 6.2, GE/Vingmed). In addition, frame-by-frame analysis of carotid artery diameter was performed in six of the nine participants whose images were of the best quality; this analysis was performed using a semi-automated edge-tracking approach (26, 48) and was used to establish the pressure dependency of diameter and cDa. Due to reductions in R-R interval with HUT, there were fewer frames of data during the upright vs. supine posture. To align measures of both cPP and diameter, the data were normalized to the duration of the cardiac cycle and fit with a best-fit regression. From the regression coefficients, new carotid artery diameter data were generated at 5-mmHg pressure increments across the cardiac cycle.

From the aligned frame-by-frame data, the slope of the diameter/cPP relationship was assessed at the 5-mmHg PP increments from the smallest to the largest pressures in the cardiac cycle. This provided the compliance of the carotid artery at specific pulse pressures. Subsequently, the compliance values were normalized to the minimum diameter in each incremental stage to standardize the values across vessels that changed in absolute diameter on going from supine to HUT positions and to derive vessel distensibility (cDa) using the standard equation (30, 39, 49):

\[
cDa = \frac{[sDIA - dDIA]/dDIA]}{Ps - Pd} \tag{1}
\]

where sDIA and dDIA are systolic and diastolic diameters and Ps and Pd are systolic and diastolic pressures, respectively. Indirect approaches to calibrate the hand-held tonometer (9) were not applied because these would have no impact on the results based on cPP alone.

Cardiovascular BRS was determined in both postures using the sequence method whereby the slope of the relationship of three or more beats of corresponding increases or decreases in R-R interval and SBP are averaged over a data series in both postures (18). This method provides directionally similar values as those achieved by spectral assessment of cardiovascular baroreflex gain (18).

Statistical method. Statistical analyses were performed using the Statistical Analysis System (SAS version 8.02, SAS Institute, Cary, NC). The effect of posture on selected variables was assessed using a repeated-measures one-way ANOVA. The level of probability for all statistical tests was set at \(P < 0.05\). Experimental data are reported as means \(\pm\) SE.

Evaluation of edge-tracking software for carotid diameter measures. Based on pixel dimensions and the image depth, the intrinsic error of image measurement based on manual caliper placement is \(\sim 0.1\) mm corresponding to approximately \(\pm 2\%\) of vessels that are 6 mm in diameter. In our hands, interobserver variations in carotid diameter measures using manual caliper placements are \(\sim 3\%\) (3 observers, average of 10 separate images over a cardiac cycle assigned in random order) with intraobserver variations of \(\sim 2\%\) (5 repeated measures of \(\sim 10\) images). To enhance the spatial resolution and diminish any observer bias in the analysis of multiple image frames from pictures of varying quality, an edge-tracking procedure was adapted for use in studying rapid changes in vascular diameter across the cardiac cycle (26). This semi-automated approach uses a discrete dynamic contour that interpolates between manually placed markers.
Table 1. Cardiovascular responses in supine to HUT

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>HUT</th>
</tr>
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<tbody>
<tr>
<td>HR, beats/min</td>
<td>63 ± 5</td>
<td>80 ± 6*</td>
</tr>
<tr>
<td>SV, ml</td>
<td>65 ± 3</td>
<td>33 ± 1*</td>
</tr>
<tr>
<td>Q, l/min</td>
<td>4.4 ± 0.3</td>
<td>2.6 ± 0.2*</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>84 ± 4</td>
<td>78 ± 9</td>
</tr>
<tr>
<td>SBP, mmHg</td>
<td>126 ± 3</td>
<td>109 ± 10</td>
</tr>
<tr>
<td>DBP, mmHg</td>
<td>65 ± 4</td>
<td>63 ± 8</td>
</tr>
<tr>
<td>PP, mmHg</td>
<td>62 ± 2</td>
<td>46 ± 5*</td>
</tr>
<tr>
<td>cPP, mmHg</td>
<td>39 ± 5</td>
<td>37 ± 5</td>
</tr>
<tr>
<td>Cardiovagal BRS, ms/mmHg</td>
<td>23 ± 5</td>
<td>9.4 ± 2.0*</td>
</tr>
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</table>

Data are means ± SE. HUT, head-up tilt; HR, heart rate; SV, stroke volume; Q, cardiac output; MAP, mean arterial pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, radial artery pulse pressure; cPP, carotid artery pulse pressure; BRS, cardiac baroreflex sensitivity. *Significantly different from baseline (P < 0.05; n = 9).

and deforms through a preselected number of iterations (usually 100), allowing the line to adapt to the distinct features in the image and define the wall boundaries. Thus multiple diameter measures are made over a 1- to 2-cm distance, providing a single average value while removing operator bias.

The reliability of this approach for diameter measures was determined with an 8-mm phantom where the coefficient of variation in three observers from eight images taken over depths of 3–8 cm was reduced from 1.2% in manual measures to 0.45% with the edge-tracking system. The reliability of this approach for diameter measures was determined with an 8-mm phantom where the coefficient of variation in three observers from eight images taken over depths of 3–8 cm was reduced from 1.2% in manual measures to 0.45% with the edge-tracking system. The reliability of this approach for diameter measures was determined with an 8-mm phantom where the coefficient of variation in three observers from eight images taken over depths of 3–8 cm was reduced from 1.2% in manual measures to 0.45% with the edge-tracking system. The reliability of this approach for diameter measures was determined with an 8-mm phantom where the coefficient of variation in three observers from eight images taken over depths of 3–8 cm was reduced from 1.2% in manual measures to 0.45% with the edge-tracking system.

RESULTS

Data for hemodynamic, BRS, cPP, and carotid diameters in systole and diastole were obtained from all participants in each of the supine and upright postures. The desired quality of carotid artery images for confident edge-tracking analysis was obtained in both postures in six of the nine subjects.

Compared with supine, heart rate increased (17 ± 3 beats/min; P < 0.01) and both SV and cardiac output decreased (P < 0.05) in HUT (Table 1). Mean arterial pressure recorded from the radial artery was unchanged from baseline during HUT (Table 1). Cardiovagal BRS was depressed in HUT compared with supine (P < 0.05). These results were similar whether determined by the manual caliper approach or by the edge-tracking system (Table 2). Based on the two-point measurements of systolic and diastolic diameters from all nine participants, cDa in supine (0.0031 ± 0.0005 U) was not different from HUT (0.0027 ± 0.0005 U).

However, vascular diameter is not linearly related to pressure. The nonlinear relationship between cPP and carotid diameter is shown in Fig. 2 for the six subjects from whom adequate image resolution was achieved for edge-tracking analysis. This pattern was retained in both postures but at a lower overall diameter in HUT compared with supine (main effect of posture, P < 0.05), agreeing with the manual caliper method of assessment in the nine participants.

The inverse linear relationship between cDa and incremental carotid pressure is presented in Fig. 3. The pressure-dependent distensibility of the carotid vessel was depressed in HUT compared with supine (Fig. 3; main effect of posture, P < 0.05). However, the overall response was largely due to a robust response in three of the six individuals assessed. Further examination indicated that changes in the maximal cDa (i.e., the distensibility in the early systolic phase when pressure is low but rising most rapidly) were more evident across subjects.
In contrast to the heterogenous nature of change in cDa in response to HUT, all subjects demonstrated some magnitude of reduction in cardiac BRS. Thus correlations between changes in cardiovagal BRS for given changes in cPP (Fig. 4A) or systolic diameter (Fig. 4B) were not impressive. There was little correlation between reductions in BRS and changes in average cDa (as measured over the entire cardiac cycle) ($r^2 = 0.29$; $P = $ not significant). Similarly, the average supine cDa over the cardiac cycle was weakly related to the change in cardiovagal BRS between supine and HUT postures ($r^2 = 0.39$; $P = $ not significant). In contrast, although based on a limited sample size ($n = 6$), there was a moderately strong positive relationship between the absolute levels of maximal cDa and the concurrent BRS ($r^2 = 0.73$; $P < 0.0005$) when data from the supine and HUT components were combined in the regression (Fig. 5A). Similarly, the change in BRS with HUT was more clearly predicted by the changes in maximal cDa ($r^2 = 0.75$; $P < 0.05$; Fig. 5B).

**DISCUSSION**

The new finding of the present study was that the upright posture acutely reduced carotid artery diameter and cDa without systematic effect on carotid PP. The reduction in cDa was evident when examined as a nonlinear effect of cPP but not when analyzed using differences between discreet measures of systolic and diastolic diameters. Thus the carotid arteries appear to undergo moderate changes in mechanical properties on moving from the supine to HUT postures. Although every subject demonstrated a reduction in cardiovagal BRS with HUT, the magnitude of response was varied, and this relative heterogeneity was most closely associated with concurrent reductions in the maximal cDa; that is, the cDa that occurred during the first 5-mmHg pressure increase during early systole. Thus an overall relationship was observed between the measures of maximal cDa and BRS in the two postures, but there was little predictive relationship between changes in carotid PP, systolic vessel dimensions, or average cDa and the corresponding change in BRS. The present results indicate that HUT elicits rapid changes in carotid artery mechanics and further suggest that reductions in the maximal cDa measured in early systole contribute to reduced cardiovagal BRS with HUT.
A major observation of the present study was the lack of change in cPP with HUT and the lack of relationship between radial PP and cPP on moving from the supine to tilted postures. These observations replicate those reported previously from our laboratory (45). Methods of use for the hand-held tonometer have been described (9), and the validity of this device in our hands to assess changes in PP (45), and in others (34), has been provided. Nonetheless, this approach may be criticized by the difficulty in calibrating the tonometer to actual pressures in the carotid artery. However, the need for absolute calibration is minimized in this study since it would not have affected the ability to detect a change in pressure across the cardiac cycle or the response between the two postures. It is also possible that application of the tonometer to the carotid artery impacted on BRS by reducing transmural pressure across the baroreceptive sites. To minimize this effect, the carotid artery was examined below the sinus. The reduction in cardiovagal BRS observed here is very consistent with that reported by other investigators using a variety of methods (18, 40), suggesting that any inadvertent effects of the tonometric method were small.

The lack of change in cPP during HUT appears to be explained by a smaller SV being injected into a slightly stiffer and smaller carotid vessel. The reduction in diameter likely was related to the hydrostatic pressure difference between supine and HUT. In addition, the sympathetic activation that occurs with HUT may have affected carotid artery diameter or distensibility, but this is difficult to predict. The effect of heightened sympathetic outflow on conduit vessel diameter and/or distensibility has been examined using a range of stimuli such as cigarette smoking (15, 17, 32), caffeine ingestion (31), cold pressor testing (3, 22), mental stress (3), and lower body negative pressure (44); to date, there is evidence for decreased (3, 15, 17, 31, 32), increased (22), and unchanged (44) distensibility values.

The finding of unaltered cPP during postural stress has important implications for studying central hemodynamics and for inferring a mechanistic basis of baroreflex function. Traditionally, peripheral blood pressure and pulse pressure measurements have been used in the investigation of central (i.e., aorta and carotid artery) vessel hemodynamics, despite cautions from other authors regarding the appropriateness of such methods (2). In particular, the direct application of peripheral PP measurements in baroreflex investigation may lead to erroneous conclusions about baroreceptor function if, in fact, cPP is not changing.

These data also challenge the direct role of cPP in baroreflex function. Certainly, changes in PP within the carotid sinus can evoke changes in carotid baroreceptor afferent firing (6–8). These earlier data, and the assumption that finger PP is representative of cPP, have led to the idea that reduced PP during postural stress is a primary stimulus for baroreflex cardiovascular control in humans. However, very few studies have looked at the comparative PP changes in the periphery and carotid arteries during orthostatic stresses. Moreover, baroreceptor afferent data obtained in isolated carotid sinus preparations point to the important role of sinus distension rather than PP per se and are consistent with the findings of Kornet et al. (25), who showed that more consistent results for BRS are obtained if baroreceptor stretch is measured directly rather than by relying on measures of PP. Thus the findings of the present experiments are in line with previous work indicating that PP is not the primary variable responsible for baroreceptor function during HUT.

An interesting observation of the present study, although based on a limited sample size, was that the reduction in cardiovagal BRS with HUT was more strongly associated with changes in the maximal cDa that occurred in early systole than with the average cDa that was measured over the entire cardiac cycle. Particularly, the largest reduction in BRS with HUT occurred in those individuals who demonstrated the largest reduction in maximal cDa. The magnitude of this relationship was similar to that based on population-based studies where a wide range of ages and BRS values were incorporated. These earlier studies reported that supine or baseline cDa predicted ~40–50% of the variability in cardiovagal BRS (28, 33, 34).

The results suggest that a portion of changes in baroreflex function with the upright posture is explained by adaptations in carotid mechanical properties. However, additional contributions from central processing of afferent and efferent signals must also be occurring (20).

Arterial baroreceptor activation is dependent on both the absolute diameter and distensibility of the vessel, whereas baroreflex function is affected by the afferent response as well as the integration and modulation of these signals centrally. The relationship between carotid artery factors and baroreflex function has been examined from many perspectives, and the inconclusive results underline the complexity of this feedback control system. For example, previous authors have cited the absolute diameter, relative change in diameter, and rate of change in vessel cross-sectional area as possible mechanical factors that affect BRS (4, 14, 25). The small reduction in the maximal slope of cPP in HUT compared with supine was not statistically significant but may have contributed to the reduced BRS in tilt as well.

The mechanistic basis of the stronger relationship between maximal cDa (or rate of distension) and BRS changes with HUT may include the various types of baroreceptor afferents from the carotid sinus that differentially affect blood pressure control (43). Compared with the type I afferents (largely myelinated A fibers), the type II afferents (largely unmyelinated C fibers) have a lower response threshold. In isolated aortic afferents, the C fibers impact on vagal outflow, whereas the A fibers have influence over both parasym pathetic and sympathetic nerve traffic (16, 23). If such a schema persists in humans, it might be speculated that the reduction in diameter and maximal distensibility of the carotid region in HUT alters the interactive effects of these two receptor types, contributing to the changes in dynamic baroreflex regulation. cDa values are highly variable in young healthy individuals (28), and this feature may impact on the ability to observe more consistent acute adaptations to postural perturbations.

**Study limitations.** During tilt experiments, where syncope is not the objective, care must be taken to prevent nonphysiologic reductions in blood pressure. Noninvasive methods that record spontaneous fluctuations in blood pressure and R-R interval circumvent these difficulties. However, the complexities of baroreflex function within a closed-loop model are not clearly understood. Thus the methodologies used to assess baroreflex function are not complete. The closed-loop nature of the sequence BRS method and its narrow range of response may explain its reduced sensitivity and correlation with open-loop pharmacological methods of assessing baroreflex function.
This limitation may also reduce the ability to detect strong relationships between carotid cDa and cardiovagal BRS within the current population compared with other approaches that perturb a larger baroreflex response. Nonetheless, although there is debate regarding the aspect of reflex function assessed, the sequence method has been acknowledged as an index of vagal tone. Furthermore, the sequence method has been shown repeatedly to detect the reduction in cardiovagal BRS that occurs with orthostatic stress, results that are supported by power spectral analysis (21, 36, 38), as well as neck suction/pressure (11, 36, 37) methods. Therefore, it is expected that the sequence method used here is adequate for exposing changes in cardiovagal BRS within the intervention of acute HUT. In line with this discussion, reductions in BRS were observed in every subject.

A potential limitation of the sequence method was that cardiovagal BRS, assessed in two postures, may be affected by the number of SBP segments observed under conditions where heart rate is different in the two conditions. Here, R-R interval and SBP data were analyzed over the final 4 min of each 5-min step. Indeed, the tachycardic response to tilt resulted in a smaller number of segments used to determine BRS in supine (23.5 ± 14.9) vs. HUT (35.8 ± 12.4; P < 0.05; n = 6). An additional influence might be the impact that postural adjustments have on respiratory-related BRS influences. However, there is strong evidence to suggest that the differences in sampling segments do not affect the outcome. In an earlier report (35), our laboratory presented the slope incidence data between supine and HUT postures, as used in the present manuscript. In that analysis, there was no difference in slope incidence calculation in the two postures. This result is very similar to the findings of Hughson and colleagues (18). As well, Tank et al. (46) did not find a difference in spontaneously determined cardiovagal BRS during supine rest or supine deep breathing. These data support the idea that spontaneous SBP ramps represent a persistent baroreflex stimulus (13). Therefore, the variations in number of cardiac and respiratory cycles between supine and HUT do not appear to have any important impact on the R-R/SBP slope determination.

A further concern with the present study was the inability to assess mechanical changes and pressure waveforms in both the carotid and aorta arteries. These vessels share similar structure and function (28), and it is likely that directionally similar reductions in diameter are present in both the carotid and aorta following the fall in hydrostatic pressure and SV with HUT. For example, Taylor and colleagues reported reduced aorta cross-sectional area during reductions of central blood volume (47). Although these effects might vary in magnitude in the two vessels, the results could summate or interact centrally to affect BRS.

In summary, the present data indicate that the carotid artery mechanics are not static but can adapt rapidly to changes in posture. Moreover, cPP was not affected by HUT. Neither changes in cPP nor in cDa could explain the consistent pattern of reduction in BRS. Therefore, although HUT alters mechanical properties of the carotid arteries, they do not appear to exert a singular impact on cardiovagal BRS change with posture.

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